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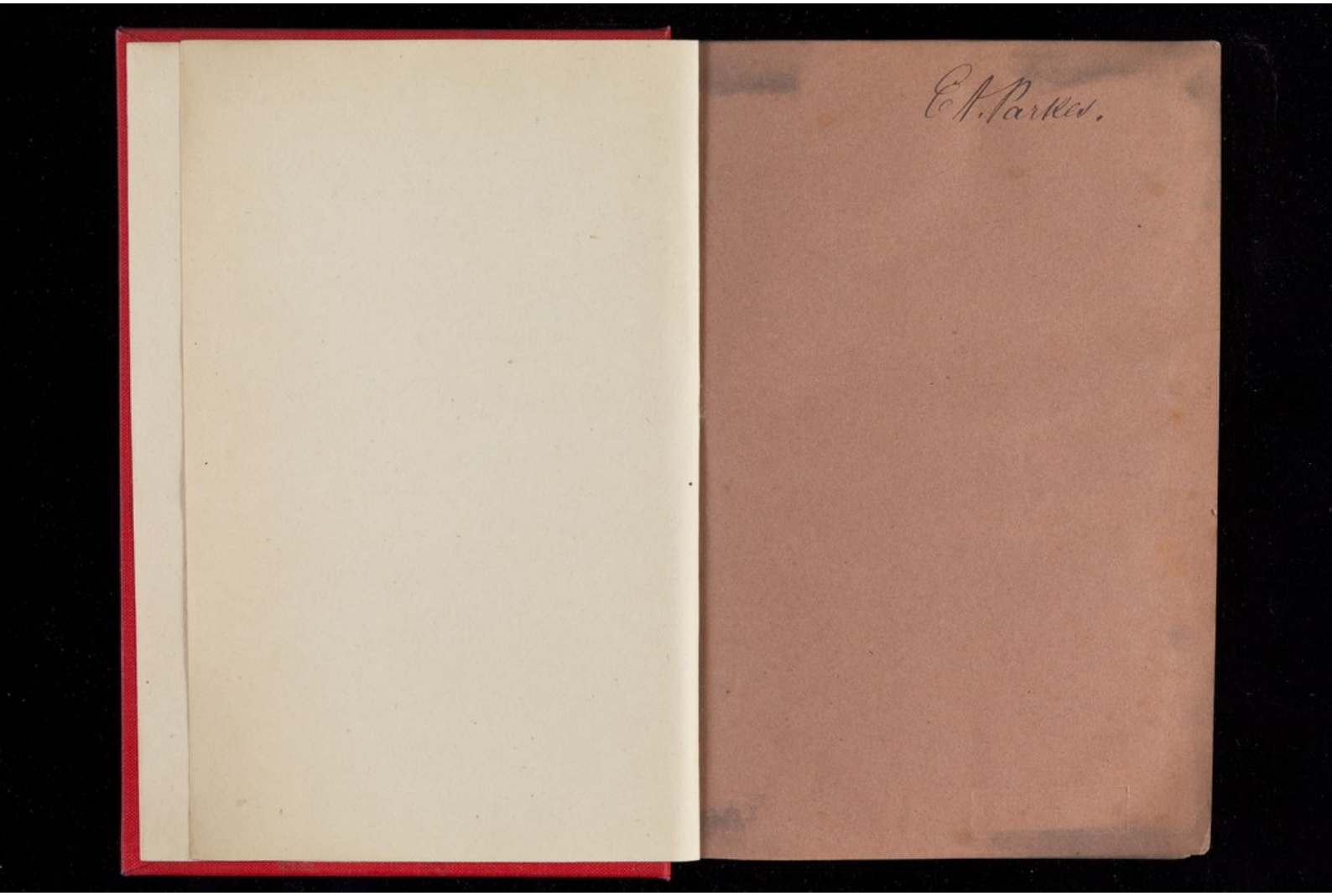
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E. A. Parker.

Name	Subject
Chevers	Pulmonary artery.
Thomson	Piriform Concretions.
Jenner	Specific Cause of Typhus Typhoid Relapsing fever (Med. Chir. Trans.) (with a plate of the eruptions)
Blair	Local origin of the Yellow Fever in British Guiana.
Mc William	Yellow Fever in Brazil - 1849.
	Sanitary Committee of New York - 1849
Taylor	Cholera in Huddersfield
Swain	Stethometer
Gaujee	Vomiting in horse
Maclagan	Colchicum autumnale,
Schultze	De arteriarum nonn: structura
Benecke	Oxaluria (see vol. for 1848-50 & the vol. marked "Pamphlets without date")
Hall	Stethometer
Gaujee	Enchondroma.
Blacklock	Intermittent Fever
Crisp	Blood vessels. (appendix)
Little	Oration on Transfusion
	Colloid
Ballard	Cancer of the Peritoneum (Med. Chir. Trans.)

Bequeathed
by Dr. E. A. Parkes.

COLLECTION OF FACTS

ILLUSTRATIVE OF THE

MORBID CONDITIONS

OF THE

PULMONARY ARTERY.

ROYAL ARMY MEDICAL
COLLEGE LIBRARY.

BY

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(*Published in the* London Medical Gazette, *between* July 1846 *and* September 1851.)

Bequeathed
by Dr. E. A. Parkes.

LONDON:

PRINTED BY WILSON AND OGILVY,

57, Skinner Street, Snowhill.

1851.

*** Several typographical errors will be met with, especially in the latter chapters of this memoir. The manner in which the book is paged has prevented the author from making out a list of corrigenda. It is trusted, however, that the reader will make due allowance for these unavoidable mistakes, some of which, unfortunately, render portions of the text extremely obscure.

ON THE

MORBID CONDITIONS

OF THE

PULMONARY ARTERY,

&c. &c.

MANY remarkable and important cases of various forms of disease affecting the structures of the pulmonary artery may be found scattered through the pages of our medical literature; and, recently, there have appeared several valuable memoirs illustrative of particular organic lesions which the tissues of that vessel are liable to undergo. Among these the essays by Mr. Paget in the Medico-Chirurgical Transactions, and the papers of Dr. Craigie and Dr. Ormerod in the Edinburgh Medical and Surgical Journal, deserve especial mention. Still I am not acquainted with any work in which the student can meet with a general and comprehensive history of the principal diseases that are known to affect this artery, or to which the morbid anatomist who happens to observe remarkable deviations from the natural condition of the vessel can at once refer with the probability of being able to compare his cases with similar recorded instances.

The pulmonary artery appears to be constructed in a manner which renders it far less liable than are the systemic arteries to undergo extensive and conspicuous organic changes: and the manner in which post-mortem examinations of the thoracic viscera are usually conducted—that of removing the heart and lungs separately from the body, and of making vertical incisions through the pulmonary structures—appears to have always led to a want of caution in examining the entire tract of a vessel thus mutilated and divided. From this it has resulted that the diseased conditions of this artery have remained, upon the whole, matters of neglected inquiry: morbid anatomists have very rarely been at the trouble of systematically examining its whole extent in cases of pulmonary and cardiac disease, and hence indolence, rather than demonstration, has led to the establishment of a general but most erroneous belief that the lesions to which this vascular tract is prone are less frequent and less important than those which are continually observed in the structures of the systemic arteries.

A practical investigation of the diseases of the vascular system, pursued in a large public institution during a period of more than ten years, has enabled me to collect a considerable number of original and, I venture to think, important memoranda of the abnormal conditions to which this artery is liable. Some remarkable instances of rare lesions of this vessel have also occurred in the practice of my friends, who have obliged me with a sight of the diseased structures, and have furnished me with narratives of the cases. In the following pages I have endeavoured to combine my own facts with those which have occurred in the experience of others, in such a manner as to present, in a condensed form, the outline of a history of the principal diseases of the pulmonary artery.

Interesting as I venture to believe the observations thus collected will prove to the scientific investigator, I should still not have felt willing to undertake the task of eliciting and recording them, did I not hope to deduce from their correlation results of greater importance than those which usually accrue from the discussion of any merely anatomical question; and if it did not appear that such an inquiry would, in no small degree, tend to advance that great purpose which must ever be the final object of all pathological research—the treatment and mitigation of disease. In the following pages I shall lose no opportunity of applying the facts adduced to the illustration of certain definite and leading principles which I believe it to be most necessary to establish for the proper therapeutic management of diseases of the heart and lungs.

Before entering upon the main subjects of the present inquiry, it will be necessary to devote a few observations to some leading points in the

Normal Anatomy of the Pulmonary Artery.

This is a vessel *sui generis*, partaking almost equally of the structural characteristics of the arteries and the veins; resembling the former in the character and arrangement of its principal tissues, and the latter in its pliability, thinness, and dilatability.

As the distance which the blood has to be propelled through the pulmonary vessels is short compared to that which it traverses in permeating the extended tract of the systemic arteries, and as the entrance of the venous blood to the lungs is greatly aided by the respiratory movements, the distending power which the right ventricle ordinarily exercises upon the pulmonary artery is considerably less than that of the left ventricle upon the aorta. Still, the muscular force of the right ventricle, in a state of health, is by no means inconsiderable, and the shortness of the tract of the pulmonary artery, together with the frequent causes of transient obstruction to which it is almost necessarily liable, render it requisite that it should be constructed in a manner which will at once enable it to escape rupture, to undergo considerable distension, and to return to its natural condition immediately after the cause of over distension shall have ceased to operate.

It is evident that a vessel possessing the comparatively rigid and inelastic structure of the aorta would, if occupying the position of the pulmonary artery, very shortly become inefficient under the frequent causes of temporary and permanent obstruction to which that vessel is exposed—its strong and closely knit fibres would soon become stretched and inelastic, its inner tissues would become thickened and loaded with fibrinous and bony deposits (the necessary concomitants of great dilatation in the systemic arteries), and, in a few years, aneurism, or laceration of its coats, would almost certainly result.

Hence we find that the pulmonary artery is constructed in a manner which renders it capable of long resisting the injurious effects of over-excited cardiac action and pulmonary obstruction. The recurved form of its branches in great measure prevents the impulse of the ventricle from being too powerfully exerted upon the minute vessels of its subdivisions; and the thinness, pliability, and tenacity of its structures, fit it to receive powerful shocks with impunity, and to suffer extraordinary distension without at once becoming permanently dilated.

I have been at some pains to determine the relative dilatability of the aorta and the pulmonary artery, and have found, by accurate measurements, that the ascending portion of the latter is very far more dilatable laterally (or when its tissues are stretched in the direction of the fibres of its middle coat), than is the latter at the commencement of its arch; while, although the structures of the pulmonary artery are somewhat more extensible in a vertical direction than are those of the main systemic trunk, the difference in this respect between the two vessels is far less considerable. The aorta, on the contrary, although less yielding in every way than the pulmonary artery, is more extensible in the vertical than in the horizontal direction.* This might be anticipated from our knowledge of the anatomical relations and the functions of the parts. The entrance of the blood to the pulmonary artery being, in great measure, due to the action of the lungs, and the right ventricle possessing, under ordinary circumstances, a very moderate degree of muscular power, the vessel does not require to be possessed of remarkable extensibility in the line of its axis, but the frequent delays to which the circulation through the lungs is liable render it absolutely necessary that it should be capable of great lateral distensibility, so as to perform, during brief periods, the office of a temporary reservoir for the blood, which can neither return to the ventricle, nor gain a ready transit through the lungs. The aorta, on the contrary, is, under ordinary circumstances, far less liable to causes of temporary obstruction. During periods of great muscular effort, &c. the passage of blood through its branches is somewhat delayed, but this retardation occurs to a less degree, and operates within a more extended system of vessels, than is the case in the pulmonary artery; hence remarkable dilatability is not requisite in the aorta;—on the other hand, however, the impulse of the powerful left ventricle acts with great extending force upon the whole aortic tract, and therefore it becomes necessary that the tissues of this vessel should possess great elastic extensibility in the vertical direction.

In a previous essay† I have described the beautiful arrangement of a structure situated between the epithelial lining and the elastic coat of the arteries, and which appears to be in great measure designed to assist them in resisting undue vertical extension. This structure appears to be far less fully developed in the ascending pulmonary artery than in the aorta, and the latter vessel seems to owe its vertical elasticity principally to the tissues which are disposed upon the exterior of its middle coat. As I shall endeavour to shew, it is probably to the rudimentary development of the sub-epithelial fibrous tissue in the ascending pulmonary artery, that the comparative rarity of semi-cartilaginous and earthy interstitial deposits in this portion of the arterial tract is due.

The question of the *comparative size of the pulmonary and aortic orifices* has occupied the attention of many anatomists, the results of whose researches have been so greatly at variance as to leave the matter altogether in an undecided position; one or two authorities declaring that the pulmonary ostium is decidedly narrower than the aortic; while the majority concur in the opinion that the former is naturally the wider of the two.

To ascertain the true natural relative size of these two vessels is certainly a matter of no slight difficulty. It will be found comparatively easy to discover the dimensions of the aorta, which may be readily measured without producing any extension of its tissues, but it will be seen that to take an accurate measurement of the width of the pulmonary artery, without stretching its thin and yielding coats, is a piece of manipulation which requires considerable tact and caution. It is almost impossible to measure this vessel either by compasses, a rule, or a cylinder, without either allowing it to remain in a partially collapsed state, or producing an amount of dilatation which, of course, does away with the accuracy of the experiment. In measuring the respective width of these vessels in recent subjects a fallacy is also liable to arise, from the fact that, under ordinary circumstances, the right cavities and vessels of the heart are, at the moment of death, in a state of extreme distension, while those on the left side are comparatively empty,—under which conditions the pulmonary artery is, in a great measure, prevented from undergoing that degree of tonic contraction which is known to occur after death in the whole of the systemic arteries. Lastly, it appears to be certain that, in by far the larger number of individuals, the temporary obstructions to which the pulmonary circulation is always liable, produce a certain degree of permanent dilatation of the artery, which slight dilatation, notwithstanding the exceeding frequency of its occurrence, the early age at which it is often established, and the unquestionably healthy condition of adjacent parts with which it is generally accompanied, must, in reality, be considered as a decidedly abnormal and morbid state.

I have taken exact measurements* of four pulmonary arteries selected with the greatest caution from young well-formed adults (three males and one female), whose deaths had been produced by causes not directly influencing the cardiac and pulmonary functions: and in whose lungs and circulatory apparatus no disease could be discovered. In these specimens I found the average measurements of the pulmonary orifice to be as follows:—Circumference—immediately above the valves, 32½ English lines; directly below the valves, 38½ English lines.

In eight cases, the average measurement of the upper (and narrowest) portion of the aortic orifice was also within a very small fraction of 32½ lines. The average width of the lower part of the aortic orifice is 36½ lines—nearly two lines less than that of the corresponding portion of the pulmonary artery; but it is evident that in measurements of this kind, the width of the *narrowest* part of the outlet is the point of most importance, as it alone represents the dimensions of the orifice, by which the blood has exit from the ventricle. The greater width of the lower part of the pulmonary ostium must not be regarded as the effect of dilatation—it is due to the peculiar form of the right ventricle, which is naturally rather broader at its upper part than the left. From the caution which I employed in choosing these specimens with reference to the stature, age, and cause of death of the individuals—rejecting many scores of specimens to which more or less objection might have been made on account of the height of the subjects, the causes of dissolution, or the engorged or otherwise faulty state of the lungs—I have no hesitation in stating my conviction that the size of the two arterial outlets of the heart is naturally the same, and that every departure from this correspondence must be regarded as a diseased condition.

* In one of my experiments, I took a square portion of the aorta of a young adult from the sinus of Valsalva, and another portion, of precisely equal dimensions, from the ascending pulmonary artery (their structures were perfectly free from any morbid change), and I found their relative dilatability and extensibility to be as follows:—Taking the transverse and longitudinal measurements of both portions as 27, the *lateral dilatability* of the *aortic portion* was represented by 41½; that of the *pulmonary* by 47. The *longitudinal extensibility* of the *aortic* fragment was equal to 43; that of the *pulmonary*, to 45.

† Account of the Sub-serous Membrane of the Aorta, Guy's Hospital Reports, No. 10.

* The mode in which I have always been in the habit of measuring the orifices of the large arteries, and which I believe to be the least susceptible of error, is to lay the vessel open by a vertical incision, without removing it from the heart, or subjecting it to any rough manipulation, and to place across the part of which it is required to obtain a measurement, a piece of common twine which has been previously sufficiently wetted to enable it to adhere to the whole inner circumference of the artery: when the string has been accurately applied in a direct line across the vessel, its ends are cut off on either side precisely at the spot where they touch the divided edge of the middle coat of the artery, and the measurement of the portion of string thus separated is immediately taken. This, of course, precisely represents the width of the vessel to which it has been applied.

I have made another set of observations of this kind in subjects who have died from chronic disease entirely apart from the heart and lungs, in whom the bulk of the circulating fluids had been long diminishing, and who had been compelled to remain in a state of complete inaction for long periods previously to their dissolution. In subjects of this kind (providing there has been no considerable previous lesion of the heart and its appendages) the measurements of the aortic and pulmonary outlets are found to be either precisely identical or varying in an exceedingly slight degree. Still it is undeniable that, in the larger proportion of adult hearts, the measurement of the pulmonary artery above its valves more or less exceeds that of the aorta in the corresponding situation. But, although this disproportion is so frequent, it is certainly unnatural. The more healthy the entire pulmonary and cardiac apparatus is, the more nearly will the upper ostia of the two great arteries be found to correspond in width; and I have long felt no hesitation in deciding, wherever (independently of morbid thickening, or any other visible structural change) the pulmonary artery, immediately above its valves, is found wider than the aorta in the same situation,—that the former vessel is unduly dilated,—and, on the contrary, when the pulmonary is the narrower of the two (its tissues retaining their elasticity), that the mouth of the aorta has become morbidly stretched. *The relative dilatability* of the upper and lower portions of the pulmonary and aortic ostia is also a matter of some anatomical interest. I have found the former artery, measured above the valves, to be dilatable to 43½ lines, —directly below to 46, while the greatest dilatability of the upper aortic ostium is 43, that of the lower 39½ lines. Hence, the upper ostia of the two vessels are nearly equally dilatable; but although, as in the aorta, the lower portion of the pulmonary orifice is the wider, the pulmonary artery differs from the aorta in presenting naturally a greater disparity in its width below and above the valves, and also in having its lower ostium more extensible and more yielding than the upper. This is evidently attributable to the fact that while the lower margins of the aortic valves are attached to a strong and almost unyielding fibrous structure, an exceedingly thin fibrous expansion exists in the right ventricle beneath the pulmonary sigmoids. Under circumstances of long-continued distension, there is, however, occasionally observed considerable thickening and rigidity of the sub-endocardial fibrous tissue at this spot.

These apparently trivial minutiæ are, in reality, of considerable importance, and for this reason—that the size of the heart and its vessels of course varies exceedingly in different subjects, according to the stature, age, sex, and thoracic development of the individuals, and the quantities of their circulating fluids;—hence, although correct averages may be drawn from very accurate measurements of perfectly well-formed hearts, it will rarely happen that the size of any heart will be found to correspond precisely with the standard. And, indeed, it is clear that neither departure from, nor accordance with any fixed standard can alone be regarded as a certain indication either of the healthy or diseased condition of a part. Thus a vessel, which is considerably below or above the average size, may, in reality, be in a perfectly healthy condition; while another the width of which precisely corresponds with the standard, may have undergone a very considerable amount either of dilatation or contraction. Hence the necessity of observing and comparing the *relative* width and dilatability of the two great arteries, as well as of the upper and lower portions of their orifices. It is scarcely of so much importance, even, to remark whether the bulk of the whole cardiac apparatus be large or small, as to observe how far the relative width and dilatability of its several portions have been preserved. A heart in which this relation persists, can scarcely be in an inefficient state; while, in a diseased organ, the relative proportions of the orifices, and their due elasticity, are rarely if ever maintained.

The researches of Hasse with regard to the relative size of the aorta and pulmonary artery, have led that observer to conclude with Bizot, that the latter is naturally wider than the former; still one or two of his observations are strongly confirmatory of my own views. It has been stated*, that the relative disproportion between the two arteries is discoverable as early as the seventh year, and it is certainly true that this morbid disproportion may be established at almost any age. M. Hasse, however, observes that, "in children the orifices of the two arteries continue equally capacious until the sixth or even the tenth year,"—a very strong evidence that any consequent disparity must necessarily be the result of unnatural dilatation.

M. Hasse observes, that, as life advances, the progressive enlargement of the two arterial orifices of the heart differs; both increase equally until the meridian of life, but the aortic orifice enlarges more rapidly in advanced life than that of the pulmonary artery, so that, in old persons, the latter is even narrower than the aorta. This variation M. Hasse has met with earlier in males than in females, (in the former from the 50th year upwards, in the latter not till after the 60th). In a still later period of life it no longer applies*. I have found that, in persons above the age of 60 years, there is always considerable dilatation of both arteries, but that their relative proportions vary in great measure according to the nature of the causes which have occasioned death. Thus, in an extremely enfeebled woman, who died of bronchitis at the age of 65, the circumference of the pulmonary outlet exceeded that of the aortic by three lines. On the contrary, in two men who died of malignant disease at the respective ages of 74 and 77, the circumference of the aortic ostium exceeded that of the pulmonary by one and a half, and two lines. But, in a man who died of gangrene in his 89th year, although (as in the other cases) the tissues of the pulmonary artery and ascending aorta appeared almost entirely free from any other change than loss of elasticity, the lower portion of the orifice of the former measured 45 lines, while that of the latter was only 39 lines in circumference.

I noted it as a remarkable fact, that, in this aged person, the narrowest portions (which were here the lower ostia) of the pulmonary artery and aorta had respectively become dilated very nearly to the utmost extent to which I have shewn that these parts of the vessel are naturally capable of being stretched. It will be found in the pulmonary artery, as in the aorta, that in an exact ratio to the degree in which any part of the vessel has become permanently dilated, is the loss of elasticity in that portion of the tube: thus we have seen that the natural measurement of the lower ostium of the pulmonary artery is 38½ lines, and that it admits of being stretched about eight lines: hence, in adult males, where this portion of the vessel is 41 lines in circumference, it may be premised that it will probably not admit of being dilated more than four or five lines; and, in cases where it is 44 or 45 lines in circumference, it will be found to have nearly or entirely lost its elastic properties.†

In cases where the upper or lower portions only of the two great arteries are much dilated, life may be considerably prolonged; but it is probable that, whenever both the upper and lower portions of their cardiac orifices become permanently dilated to the greatest extent which the natural yielding of their structures is capable of admitting—even independently of any very apparent change in their tissues beyond mere loss of elasticity—life begins to be held on an exceedingly frail tenure. It is true that, in a *few* cases, the widest part of the pulmonary orifice has been found to exceed its natural measurement by an inch or more, but the subjects of this lesion have invariably died from failure of the heart's action. And it can scarcely be doubted that a considerably less degree of dilatation than this is sufficient to occasion sudden death in consequence of any cause which tends to produce over-distension of the ventricles.

This question appears to be by no means undeserving of further investigation, especially as illustrative of the immediate cause of death in extreme old age, and in cases of long-standing disease of the heart and lungs.

The following are the principal subjects which will be considered in the succeeding observations:—Congenital Irregularities in the Mode of Origin and Distribution of the Pulmonary Artery; Unnatural Dilatation and Contraction of its Canal; Inflammation; Pulmonary Apoplexy; The causes which determine the deposition of Clots in the Pulmonary Artery; The Diseases of its Valves; Ulceration and Laceration of its cardiac and pulmonary divisions. The Pathological Condition of the Ventricles, especially of the Right, in cases of Disease of the Pulmonary Artery and Lungs; Symptoms and Diagnosis of the Diseases of this vessel; a Sketch of the General Principles of Treatment which it is necessary to pursue in these and other forms of Organic Obstructive Disease in the Cardiac and Pulmonary Apparatus.

CONGENITAL MALFORMATIONS OF THE PULMONARY ARTERY, AND IRREGULARITIES IN ITS FORM AND MODE OF ORIGIN.

As it almost amounts to an invariable rule that each of the severer kinds of congenital malformation of the heart is characterised by more or less departure from the natural condition of the pulmonary artery, a complete description of all the cases where this vessel is found in an abnormal condition must involve an allusion to nearly every one of the congenital malformations to which the cardiac apparatus is liable. I shall therefore endeavour to render this portion of the history as brief as possible consistently with my plan of furnishing reference to all the

* Anatomical Description of the Diseases of the Circulation and Respiration. By C. E. Hasse. Translated by W. E. Swaine, M.D. Published by the Sydenham Society, 1846.

* Cyclopæd. of Anat. and Physiol.; Art. Heart.

† When any vessel is found dilated, if its standard capacity be known, the loss of elasticity in its tissues may generally be taken as an indication of the degree in which its calibre has become increased. But this test does not, of course, hold good after the canal has become permanently widened beyond the extent to which it is *naturally* dilatable. Whatever slight amount of elasticity varicose and aneurismal vessels may possess, is due to the increased development of their elastic tissues. It is scarcely necessary to add, that a test of this kind only avails to show that every vessel has a naturally fixed amount of elasticity, as well as an average capacity.

most remarkable examples of the several kinds of malformation in which the pulmonary artery is especially implicated; merely stating the leading particulars of each instance, and especially selecting those details which afford the amplest data in illustration of the pathology and symptomatology of this interesting class of diseases.

Malformations by defect of the Pulmonary Artery.

Absence and congenital obliteration.

In the vertebrate animals, an aorta is an inseparable and essential constituent of the circulating apparatus*. The absence of a true pulmonary artery, originating by a separate outlet from the heart, is, however, a condition which has been not unfrequently observed. The ascending aorta has been found absent only in acardiac fœtuses; where there is a heart there is always a pervious ascending aorta, however narrowed, malformed, or displaced that vessel may be: but imperforation, or entire absence of the ascending pulmonary artery, may coexist with a rather high degree of cardiac development.

A single ventricle giving off a single artery (aorta), which distributes one branch to the lungs.

The condition of the heart in its earliest stage of development here becomes permanent: the organ remains in the form of two simple cavities, undivided by septa, and the pulmonary and systemic vessels are distributed from a common trunk. The most singular instance of this species of malformation is one related by Mr. Wilson†. The child lived 7 days, and had merely slight lividity of the lips on the day of its birth and a short period before its death. It seemed weak, and in pain, but otherwise it performed all its functions naturally. Death resulted from inflammation of the heart. The heart was found embedded in a cavity formed on the superior surface of the liver, and consisted of a single auricle, ventricle, and arterial trunk, which latter ascended into the thorax, and soon divided into two large branches, the aorta and the pulmonary artery. The aorta, ascending, formed its arch as usual. The pulmonary artery divided into its two branches. The circumference of the aorta, where it separated from the original trunk, was an inch and a quarter. The circumference of the pulmonary artery was only fifteen-sixteenths of an inch. No bronchial artery was sent to the lungs from the aorta or any of its ramifications. There were no bronchial veins. The lungs were, however, larger in proportion than in other children of the same age*.

A second case has been recorded by Mr. Standert†. This child lived only 10 days; it had cyanosis, but it is stated that the respiration, muscular action, and temperature of the body, were not materially affected. The heart consisted of one auricle, into which the pulmonary veins and the venæ cavæ entered, and which presented the rudiment of a septum, in the form of a muscular band, crossing the ostium venosum; and one ventricle, whence arose the aorta, which sent off a small branch in the situation of the ductus arteriosus. This branch divided into two arteries, which were distributed to the lungs. The pulmonary veins were four in number; but neither the area of these veins, nor that of the vessel which acted as the pulmonary artery, exceeded half the common dimensions. It does not appear very certain that the above case strictly belongs to the class in which I have placed it. It may, not improbably, have been one in which the ascending pulmonary artery had become obliterated, the ductus arteriosus only remaining to perform its functions, in which case the condition of parts, although in appearance almost identical with that observed in Wilson's and Tiedemann's cases, must of course have been, physiologically, altogether different.

It has very rarely happened, in the instances of this kind which have been recorded, that the infants have survived for more than a few days, but I shall hereafter have occasion to quote a case (by Dr. Mauran) in which, although there was merely a single auricle and ventricle, and the pulmonary artery was impervious at its origin, the child lived ten months and a half.

A single ventricle giving off a single artery (aorta), which distributes two branches to the lungs.

A malformation of this kind was observed by Dr. Farre*, in a male infant, which died seventy-nine hours after birth, having suffered for thirty hours from dyspnœa, and some coldness and livor of the surface. The heart was found distended to the utmost with blood: it consisted of an auricle and a ventricle. From the ventricle one artery—the aorta, furnished with semilunar valves—arose; its first two branches were pulmonary, very large, and situated close to each other.†

Otto describes that when there is a single arterial trunk from the heart, the pulmonary branch or branches are usually given off low down. Occasionally, however, higher up in the ascending portion of the aorta, as described by J. F. Meckel (Descriptio Monstrorum Nonnullor, p. 11).

Heart consisting of two auricles and two ventricles, giving off a single artery, which distributes pulmonary branches.

M. F. Tiedemann observed the following singular malformation in the heart of a male child, which died 9 days after its birth. The superior and inferior vena cava, and the coronary vein, opened into the right auricle. From the arterial part of the heart arose a single but very large trunk. After giving off the coronary arteries, there arose from the right side of this great trunk a large artery, which ascending gave off the left and right carotid, and the right subclavian. From the posterior part of the great arterial trunk the left and right pulmonary arteries originated; these were sub-divided and distributed in the usual manner. A little higher up the main trunk became very much contracted, but immediately afterwards resumed nearly its former calibre, and represented the descending aorta. From the arch of the aorta (beyond the contraction) the left subclavian artery arose. The foramen ovale was pervious. The right auricle communicated with the right ventricle, and the left auricle with the left ventricle. The ventricular septum was perforated at its upper part, so as to allow a communication between these two cavities. The great arterial trunk took its origin chiefly from the right ventricle, but there was a small communication of its orifice with the left ventricle also.

The child [when born] appeared well nourished, but wasted until the time of its death. It took the breast feebly; the warmth of the body was very sensibly less than that of other children of the same age. Its breathing was always short, but there was not any blueness of the surface. Death was preceded by convulsive attacks, and appears to have been due to inflammation of the spinal chord, and congestion of the meningeal vessels of the brain*.

Dr. Arrowsmith, who translated the above case, and appended some interesting physiological remarks, justly observes that it appears to be quite unique. The ventricular septum was in great measure formed, although the ventricular arterial trunk remained still undivided at its origin; in fact, nearly in its primitive state. It appears probable that, in all the cases where the cavities of the heart remain single, there exists, from a very early period of intra-uterine life, a certain amount of impediment to the pulmonary circulation, owing either to narrowness of the pulmonary vessels or mechanical obstructions of various other kinds†. The figure which accompanies this case, in the original paper, shows that here the afferent vessels of the lungs were of considerable, though apparently not of full, size; hence, probably, the more advanced formation of the septa.

The heart consisting either of a single auricle and ventricle, or of two auricles and one ventricle, the pulmonary artery and aorta arising by separate orifices from the ventricle.

There is another class of cases where, the cavities of the heart remaining single, the pulmonary artery is found to have become completely developed. The instance already quoted from the Ephemerides belongs to this class. The following is a nearly parallel example:—

In a case published in Hufeland's Journal‡, a man, æt. 24, who from his infancy had been subject to violent beating of the heart, suffered after an attack of pneumonia from great increase of palpitation and frequent paroxysms of suffocation, in which he found no relief, except by pressing the chest

* Except, of course, in those rare cases where a head or limb has been alone produced.
† Philosophical Transactions, vol. lxxxviii. p. 346.

* Mr. Wilson considered that "the pulmonary arteries and veins, in addition to their usual offices, performed those of the bronchial vessels."
There appears to be a tendency to displacement in cases where the heart is single. Ant. de Pozzi relates (in the Ephemerides Nat. Cur. Ann. 4 and 5, Obs. 40,) the somewhat marvellous case of a man, ætat. 27, who suffered, during four years, from palpitation, which could only be relieved by venesection, and who died in a sudden attack of syncope. The pulsation of the heart was not felt in the usual place, but low down on the left side, near the diaphragm. The heart was found very large, consisting only of one cavity or ventricle, containing sixteen ounces (?) of very dark blood. The whole substance of the heart was wasted, the arteries elongated and drawn together, so that the arterial blood (sanguis spirituosus) could not be fully expelled. In the place of the right ventricle the ascending vena cava was found raised into a large tumor (the single auricle).
In a specimen of Ectopia cordis, (preserved in the collection at Guy's Hospital, Prep. 1379 2 5,) where the ventricular part of a singularly malformed heart protruded from the central portion of the chest, the ventricle—a large part of which has been sliced off vertically—appears to me to have been single; and an excellent anatomist, who examined the preparation with me, coincides in that opinion. The auricles, which were situated within the thorax, communicate with each other by a very wide aperture. These, however, are not invariably the characters of congenitally displaced hearts, which, in several cases, have been found normally constructed.
† Philos. Trans. vol. xcv. p. 228.

* Pathological Researches, Essay 1, on Malformations of the Human Heart, p. 2.
† M. T. Weber (Observatio Anatomico Path. de Corde Univentriculari e quo unus tantum truncus arteriosus surgit. Bonnæ: 1832) describes and figures a similar malformation which he found in the heart of a calf. There were, however, two auricles in this case, and a large common coronary artery was given off from the ascending aorta.

* Zeitschrift für Physiologie, B. iv. H. ii.; and London Med. Gaz. vol. xiv. p. 704.
† See Cyclop. of Pract. Med. vol. iii. p. 66.
‡ Quoted in the Lancet, 1828-9, p. 137.

strongly against some resisting body. An incipient hæmorrhoidal discharge seemed, for some time, to mitigate the symptoms, but subsequently the results of thoracic obstruction became aggravated, anasarca and ascites supervened, and death ensued, apparently from suffocation. The cavity of the chest was found to be filled with several pints of bloody serum. The pericardium, to its whole extent, adhered to the heart, the cavities of which were filled with black blood. The *septum ventriculorum* was totally wanting; not the slightest trace of it could be found. The origin of the vessels was natural; the apertures of the veins were slightly enlarged, and the aorta was remarkably flaccid. No mention is made of the existence of cyanosis.

A child, æt. eleven days, died apparently of umbilical phlebitis, in the Foundling Hospital of Paris. The heart was found to consist of a single auricle and ventricle, and the aorta and pulmonary artery were given off from the latter by separate orifices. The child suffered from cough and great dyspnœa, but there was *no cyanosis**.

Tiedemann† describes the heart of a boy, eleven years of age, who had long suffered from cyanosis. This organ was found to consist of two auricles, but only one ventricle, from which the aorta and pulmonary artery sprung.

Chemineau‡ examined the heart of a fœtus consisting of three cavities; the right received the vena cava, the left the pulmonary veins, while from the central, or ventricle, the pulmonary artery and the aorta arose§. Other instances of this kind of malformation have also been recorded.

I have already referred to the probability that the undeveloped state of the septa in these as in many other cases of congenital malformation of the heart, depends, to a certain degree, either upon the narrowness of the pulmonary branches, or upon the existence of some cause of obstruction either in the lungs or systemic vessels; but, independently of this, more or less imperfection of the ventricular, as well as of the auricular septum, is in these cases rendered an arrangement of almost absolute necessity during the entire period of intra-uterine life, owing to a defect by which the whole of the above-described malformations appear to be characterised‖; *i. e.* the absence of a ductus arteriosus. In either of these kinds of malformation a ductus arteriosus could answer no useful purpose, but in either the closure of the ventricular septum, could it possibly occur, would at once produce a fatal alteration in the course of the circulation. It will be borne in mind that, although a great degree of livor may be present, cyanosis is not an essential or even a usual concomitant of the above kinds of malformation.

In cases of this description, as well as in those of the two classes previously alluded to, the abnormal condition of the heart and arteries is purely one of arrested development. Most of the other forms which I shall have to describe are to be considered either as the results of disease occurring during intra-uterine life, or as mal-conformations, in the strict sense of the term, combined to a greater or less degree with arrest of development. In many of these kinds of malformation the arrangements of parts have been regarded as almost identical with those which naturally exist in the circulating systems of several of the lower animals,—such as the ophidians, fishes, batrachians, birds, and inferior mammalia. In some of the cases, where the fault lies merely in arrest of development, or the malposition or excess of parts, these analogies, to a limited extent, hold good; but most anatomists of the present day will agree with Otto that a large proportion of the comparisons of this kind which have been drawn are fanciful and overstrained.

Obliteration of the ascending pulmonary artery, or congenital occlusion of its orifice.

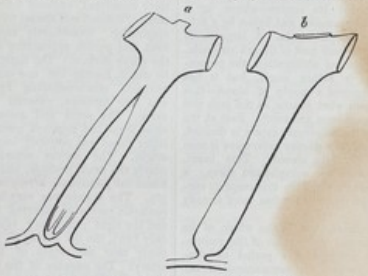
The cases of this class are, in reality, instances of obliteration of the pulmonary artery, not of absence or non-development of the vesssel. Careful examination will generally show that the ascending pulmonary trunk has existed as a separate, if not as a pervious canal—whether in some of the cases it has ever communicated with the ventricle must remain a matter of doubt. Its obliteration usually appears to be attributable to disease occurring at a very early period of intra-uterine life, probably antecedent to the second month, as shown by the imperfect state of the ventricular septum which is observed in a very large proportion of the cases.

The recorded instances of this remarkable species of congenital lesion are rather numerous; and I have myself had opportunities, within the last two years, of examining three recent specimens of the kind, exclusively of several well-marked examples which are to be found in the various pathological collections of this metropolis. Five cases were quoted by M. Gintrac, and many others have been subsequently observed. I find twenty-one cases of this description on record,

* Archives Gen. de Méd. Feb. 1843.
† Zoologie, Part I. p. 177.
‡ Vide Sandifort, Obs. Anatom. Path. T. iii. C. i. p. 38.
§ As quoted in the Cyclop. of Pract. Med.
‖ See remarks on Mr. Standert's case.

exclusively of the three which I have recently seen.

The pulmonary artery has been found obliterated in several ways. There may be discovered merely an indistinct band of condensed cellular tissue, or a thin impervious cord extending upwards in the usual course of the vessel, and attached to a small transverse arterial canal which sends branches to either lung; or a strong firm membrane may be observed stretched completely across the lower part of a narrow channel (the rudiment of a pulmonary artery) extending from the base of the right ventricle, and communicating with a branch from the aorta. The upper surface of this membrane may present some slight traces of a valvular structure (Fig. *a*), and it is sometimes dilated into a little shallow pouch which projects downwards into the ventricle. Occasionally the orifice appears suddenly constricted, as if a thread had been drawn tightly around it (as represented in the diagram, fig. *b*). Perforation of the ventricular



septum is a usual, but not invariable, accompaniment of congenital obliteration of the pulmonary orifice. Wherever life has been prolonged beyond a few weeks the ventricles have communicated. The ductus arteriosus is generally, but not always, pervious. The ovalian canal may or may not remain patent. In most of the cases where life has been considerably prolonged, this opening has been found either completely obliterated or greatly contracted.

In the whole of these cases the lungs receive blood either directly or indirectly from the aorta, or from arteries distributed by its primary branches. This supply is furnished in several modes, of which the following are the principal.

The orifice of the pulmonary artery obliterated, the arterial duct remaining pervious.

Permanency of the canalis arteriosus is the most frequent as well as the most natural means by which the lungs are supplied with blood in cases where the orifice or ascending trunk of the pulmonary artery is congenitally imperforate. The most remarkable instances of this kind on record are those observed by Dr. W. Hunter, Mr. Hodgson, Mr. Howship, Dr. Mauran, and Dr. Spittal.

In Dr. Hunter's case,* the infant, which was born at the eighth month, had cyanosis, dyspnœa, and violent palpitation of the heart, and died on the 13th day; the commencement of the *pulmonary artery* was converted into a solid substance or cord; but, except at its beginning, it was everywhere pervious, though small, and the *canalis arteriosus* had supplied it with a scanty share of blood. The right ventricle had scarcely any cavity left. The *foramen ovale* was very large.

There are two preparations, in the collection at Guy's Hospital, which bear a strong physiological resemblance to the heart described by Dr. Hunter.

In that marked 1383 (which is the heart of a fœtus or very young infant), the right ventricle is furnished with a diminutive tricuspid valve, but has become almost obliterated by an extraordinary concentric thickening of its walls. The *pulmonary orifice* is obliterated, the *ductus arteriosus* open. The *ventricular septum* is entire. The aorta, which is large, originates from the left ventricle.

The preparation 1382[a], is also the heart of a fœtus: its *pulmonary orifice* is closed. The auricles are small, communicating freely with each other. A very small chink, about a line in length, represents the right auriculo-ventricular orifice—below this, and rather to the left, is a little

* Med. Obs. and Inquiries, vol. vi., p. 291.

cavity (the right ventricle), furnished with the diminutive rudiment of a tricuspid valve. The right auricle communicates with the left by a large *foramen ovale*, guarded by a remarkably formed sigmoid valve which opens just above the left auriculo-ventricular orifice. The ovalian valve must have permitted a free passage of blood from right to left, but must have been efficient, or nearly so, in the contrary direction. The left ventricle, which is capacious, and communicates freely with the left auricle, is partially separated about its centre—above by a membranous expansion, and below by a network of pectinated bands. A large aorta arises from the back part of the left ventricle. Above the point of obliteration, the pulmonary artery is rather small, but it has evidently received a supply of blood—it is uncertain from what source, as the arch and its vessels have been removed. Just at the point where the pulmonary artery is divided, the orifice of the *ductus arteriosus* is seen apparently partially closed. This heart, notwithstanding its complicated form, is essentially single; and, in this respect, corresponds with those in the two cases already detailed.

It is probable that, in the whole of the above instances, obliteration of the pulmonary orifice occurred at a period of intra-uterine life, when the communication between the ventricles had been entirely or nearly closed by the septum, under which circumstances the right ventricle, destitute of an outlet, and reduced to the condition of an inefficient cul de sac, became contracted and almost impervious. This is the only class of cases with which I am acquainted where—the pulmonary orifice being congenitally imperforate—a free communication between the ventricles does not exist. This condition of heart appears to be incompatible with a prolonged duration of extra-uterine life.

Mr. Hodgson's case was published in the London Medical Review. The heart has also been described by Dr. Farre. The *foramen ovale* was widely open. Some of the fibres were wanting in the ventricular septum, and the lining membrane of the left ventricle had three foramina, giving it a cribriform appearance. In the place of the *pulmonary artery* there was an impervious filament leading to a very large *ductus arteriosus*, which might be traced from the aorta into two pulmonary branches. The trunk was much larger than an ordinary ductus arteriosus, but smaller than a pulmonary artery. The child lived seven days.

Two similar specimens are referred to by Farre, as contained in Mr. Langstaff's museum,—one from a still-born infant, the other from a child who lived 6 months. In this case there was a considerable perforation of the *septum of the ventricles*, but it was nearly closed by the fibres which almost filled up the right ventricle. The same author makes slight allusion to two other specimens.

A remarkable case, showing how completely the development of the ventricular and auricular septa may be prevented, when occlusion of the pulmonary orifice occurs at a very early period of intra-uterine life, is related by Dr. J. Mauran.* A female child, æt. 10½ months, of small stature and exceedingly emaciated, suffered from frequent attacks of difficult and almost totally suspended respiration, immediately succeeded by a livid and purple appearance extending over the whole surface. The heart was found to be single, having but one auricle and one ventricle, communicating by a very large and free foramen, guarded by a tricuspid valve. The aorta and pulmonary artery arose from the common ventricle, but the latter vessel was perfectly impervious at its origin. The *ductus arteriosus* occupied nearly the usual position, and was open, and hence the *pulmonary artery* received its supply. The auricle was very large; into its posterior part were inserted the two cavæ and the right pulmonary vein (there were only two), the left having an insertion anteriorly near the origin of the pulmonary artery. The parietes of the ventricle were unusually thick and strong.

Dr. F. Ramsbotham observed a very similar condition of parts in a child 6 months old. The heart was single, consisting of one auricle and one ventricle; it was turned to the right side, and the *ductus arteriosus* seemed to supply the place of the *pulmonary artery*. The pulmonary veins of the left lung terminated in the left subclavian vein—that of the right lung passed through the diaphragm and terminated in the vena portarum. There appeared to be an attempt to form a pulmonary artery and vein†.

The following instances represent the condition of parts which is most frequently observed in cases of this description, viz., the partial division of the heart by an imperfect ventricular septum—the aorta originating either from the right ventricle, or from a point immediately over the septum, mid-way between the two ventricles.

Dr. Farre relates‡ the case of a female infant, æt. 4 weeks, where the *ventricular septum* was perforated; the aorta arose from

* Philadelphia Journ. of Med. and Physical Sciences, vol. xiv., p. 253.

† So stated in the original account; but, as the course of the pulmonary veins is distinctly mentioned, this allusion is obscure: it probably refers to a rudiment of the fourth pulmonary vein, only one being described as passing from the right lung.—Medical and Physical Journ. vol lxi. p. 548.

‡ Pathological Researches, p. 27.

both ventricles, and was dilated. The *pulmonary artery* was imperforate as far as its bifurcation, and its branches received their blood from the aorta, through the ductus arteriosus. The right auricle and ventricle were larger than the left. The child was cyanosed.

Mr. Howship's case* was that of a female child, æt. 5 months, which had suffered from cyanosis and frequent suffocative attacks. The heart was found much larger than ordinary, the right ventricle was strong, the walls of the left wasted, the left auricle exceedingly small, and almost incapable of transmitting blood. Both ventricles communicated with the aorta, which, however, arose from the right. A rudiment of the *pulmonary artery* was with difficulty discovered—it arose in the usual situation of that vessel, but it was impervious, and had no communication with the ventricle. It terminated below in a small ligamentous cul de sac close upon the membrane lining the cavity of the ventricle. The upper part of the pulmonary artery, which was distributed as usual to the lungs, was freely supplied by a pervious *ductus arteriosus*.

Dr. Spittal† has recorded the case of a female child, born at the full period, which presented nothing anomalous in its external appearance, except that the integuments were observed to be of a bluish-purple colour, especially over the extremities. The colour of the skin varied in intensity, being sometimes darker than at others. The child took its nourishment freely, and, for the first few days of its existence, appeared to be doing well. This state, however, did not continue; the infant began to refuse its food, and became subject to frequent attacks of syncope, in one of which it was reported to have expired, having lived only 23 days. On examination, the heart was found to be nearly of the usual size. The walls of the right ventricle 3-16ths of an inch in thickness, those of the left 1-4th of an inch thick. The cavity of the former was greatly larger than that of the latter. The tricuspid and bicuspid valves were of their natural structure. A slight deficiency existed in the upper part of the *septum of the ventricles*. Immediately above this the aorta arose; it was larger than ordinary as far as the ductus arteriosus, being a quarter of an inch in circumference, but beyond this point the vessel was not examined. The rudiment of the *pulmonary artery* arose from the usual situation at the base of the ventricle close to the aorta, by a very small vessel of the usual structure, but capable of admitting only a very small probe, and quite impervious at its origin. In passing upwards, the vessel became rather abruptly enlarged in breadth, and, immediately beyond the ductus arteriosus, divided into its right and left pulmonary branches. The *ductus arteriosus* was open, and admitted a large probe freely. The auricles were naturally formed. The *foramen ovale* was capable of admitting a goose-quill. It is stated that auscultation revealed nothing unusual in the action of the heart.

Dr. Houston observed a good example of this kind of malformation in a male child which died at the age of 18 months. It was cyanosed from birth, and about the sixth month, when teething began, it had several convulsive struggles, one of which terminated in total insensibility and apparent death; it was recovered, however, by inflation of the lungs, &c. It finally expired in a convulsive struggle. The venæ cavæ and their leading branches were unusually dilated; the heart larger and firmer than natural; the right auricle double the ordinary capacity and thickness; the *foramen ovale* round and wide. The right ventricle was larger than the left. The tricuspid valve was perfect. The ventricle had no communication with the *pulmonary artery*; the passage from it into that vessel was totally blocked up. The left auricle was small and empty; the left ventricle exhibited nothing singular in its size or structure, but communicated with the right by a large round opening in the *ventricular septum*. The aorta at its origin was much dilated; the *ductus arteriosus* was large and pervious, and served as a trunk to the branches of the pulmonary artery. The pulmonary artery, though inconsiderable, was pervious down to its point of attachment at the ventricle.*

There are two specimens of this kind of malformation in the Museum of Guy's Hospital.

That numbered 1379 is evidently the heart of a very young infant. The aorta arises from both ventricles. The *pulmonary artery* is quite closed at its origin. This artery communicates above with the aorta by the pervious *canalis arteriosus*. There is an aperture in the upper part of the *ventricular septum*.

The preparation marked 1380^{50} is also from a young infant. A large aorta springs from both ventricles above the wide aperture in the *ventricular septum*. The orifice of the *pulmonary artery* is quite obliterated. There is a small opening in the *auricular septum*. The *arterial duct* is pervious. The right ventricle is not very powerful, but is stronger than the left. The right auricle greatly dilated; the left very small.

* Edinburgh Medical and Surgical Journal, vol. ix., p. 399.

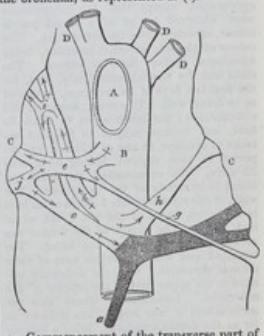
† Ibid. vol. xlix. p. 109.

* Dublin Hospital Reports, vol. v. p. 234.

Pulmonary artery closed, or obliterated. Lungs supplied by the ductus arteriosus and bronchial arteries, or by enlarged bronchial arteries only.

The following is an excellent example of the latter kind of malformation. In the heart of a girl, ætat. 16 years, presented to the Hunterian Society by Dr. F. Ramsbotham, there was no *pulmonary artery*: its deficiency appeared to be supplied by the *bronchial arteries*; the aorta, springing from a point between the two ventricles, ran on the right side of the trachea. There were two right bronchial arteries, and one left, all enlarged. The girl died of tubercular phthisis. The face had been very livid*.

A remarkable case of the former description occurred within my own observation, in a patient of Dr. Babington's, at Guy's Hospital, in October, 1844. The subject of this malformation was an old-fashioned looking boy, ætat. 10 years, born at the full period. His mother stated that she had suffered great mental disturbance during gestation. His intellect was precocious; he was always considerably cyanosed. An intense cardiac bruit was audible between the shoulders. Death resulted from an acute attack of thoracic inflammation. On inspection, some bloody fluid, containing thin shreds of fibrine, was found in the pericardium. The volume of the heart was large; its muscular parietes were dense and strong. The right auriculo-ventricular orifice was considerably narrowed, the tricuspid valve ill-formed, and imperfect. Both ventricles were of full size; the right was rather rounded in form, with strong muscular parietes: the walls of the left were equally powerful. The upper part of the *ventricular septum* was deficient, leaving an oblique, but free, interventricular opening, capable of admitting the first joint of the forefinger. This opening was formed in such a manner that the anterior curtain of the mitral valve on the left side formed the base of the left aortic sigmoid valve on the right. The aorta, perfect in all its parts, and about as large as it usually is in a person fourteen or fifteen years of age, arose from the right ventricle in the usual situation of the pulmonary artery, giving off two coronary arteries behind the sygmoids, and distributing the vessels of the arch in the usual manner. No distinct traces of a *pulmonary orifice* were observable; but, in the position usually occupied by the ascending pulmonary trunk, my friend Dr. Gull, who examined the parts with great care†, found a thin impervious cord (*a*) passing upwards to a vascular canal, (*c*, *c*,) transversely placed, which was evidently the upper part of the pulmonary artery, consisting of its right and left branches, passing to the lungs, and a portion of its ascending trunk (*d*), which, however, terminated below in a cul-de-sac. This latter portion of the vessel, as well as the whole of the left branch (*b*), was completely filled with a dense coagulum of some standing. At a short distance below the termination of its arch, the aorta gave off from its under part a large pervious *arterial duct* (*e*), which passed to the right lung, dividing into two branches just before it reached the bronchus; one, (perhaps both) of these branches communicated very freely with the branches of the pulmonary artery at (*j*). A very slender and long vessel (*g*), scarcely more than wide enough to admit a bristle, was also distributed from the arterial duct to the left lung. About half an inch below the duct two large vessels (*hh*), evidently *bronchial arteries*, greatly developed, passed upwards, one from either side of the aorta to the right and left lung. That on the left side appeared to have become narrowed in the middle: on the right very free communications may be observed between the pulmonary artery and the bronchial, as represented at (*i*).



A, Commencement of the transverse part of the aortic arch, the heart and ascending portion of the arch having been removed.
B, Descending thoracic aorta.
C, C, Lungs.
D, D, D, Vessels of the arch.

The above figure is merely a diagram, and by no means fully represents the extensive communications which existed between the large arterial branches at their entrance to the lungs.

* Med. and Physical Journal, vol. lxi. p. 548.
† And to whose suggestions I am much indebted for several of the details of the diagram.

The narrowing of the bronchial artery and of the branch from the duct on the left side must have greatly diminished the supply of blood to the left lung; but, while the left branch of the pulmonary artery remained pervious, that lung must have been freely supplied from the right, as may be at once seen on referring to the diagram.

The mitral valve was in an extremely diseased state; its orifice was somewhat narrowed, and the whole of the chordæ tendineæ of its anterior curtain were ruptured, their extremities being loaded with fibrinous deposits. Both auricles were large. The *foramen ovale* was closed; but, below it, there was a narrow circular opening through which a common director could be passed from either auricle, although it was furnished with a little imperfect valve. The left auricle presented a line of rough, granulated, fibrinous deposit, just above the origin of the posterior mitral curtain; an appearance frequently observed where there is disease of the left auriculo-ventricular orifice. The lungs were large, and extensively emphysematous on the surface; they contained several extensive recent apoplectic effusions. The liver was extremely congested, and bore traces of having suffered engorgement on previous occasions. The spleen was large and flesby; its tunic very opaque. The kidneys were much congested, and starred with minute spots of ecchymosis, as were also the mucous linings of their pelves.

The heart and aorta are preserved in the museum at Guy's Hospital. The former is numbered 1383$^{2\,3}$.

It is probable that the life of this child would have been considerably prolonged had not extensive lesion of the mitral valve been superadded to the malformation from which he suffered. The lungs were more freely supplied with blood than is usual in such cases, and that the pulmonary circulation had remained tolerably unimpeded until shortly before the date of the fatal attack is evidenced by the capacity and strength of the left ventricle, which is usually found small and weak in cases where arterial blood is scantily returned from the lungs. An attack of thoracic inflammation, in itself of rather a severe kind, became fatally aggravated by the obstruction offered by the diseased mitral, which also appears to have determined the occurrence of the pulmonary apoplexy and the great engorgement of the abdominal viscera.

The formation of a clot within the left branch of the pulmonary artery most likely tended to hasten the fatal event, the supply of blood to the left lung from the arterial duct and left bronchial artery being extremely scanty. The formation of a coagulum in the artery was in all probability attendant upon the pulmonary apoplexy, which was very extensive in this lung. The cardiac bruit heard between the shoulders was doubtless produced by the mitral lesions.

The pulmonary circulation supplied by supernumerary arteries from the aorta or its branches.

Otto and Andral state that the pulmonary arteries are sometimes given off from the subclavian.*

Although the preparation of the parts which were removed in the following case is imperfect, and it was not positively ascertained in what manner the pulmonary vessels received their supply, I think there are strong grounds for believing that blood was conveyed to these arteries by a branch from one of the large vessels of the arch, and, most probably, from the left subclavian. In May last, Dr. Thomas Chambers presented to the notice of the West London Medical and Surgical Society the heart of a boy between 9 and 10 years of age, who had laboured from his birth under the usual symptoms of morbus cæruleus, had several times suffered from hæmoptysis, and died rather suddenly with symptoms of pulmonary obstruction. Upon examination after death, the lungs were found extremely congested, and nearly airless throughout. The whole of the pericardial surfaces were inseparably united by old adhesions. By the kindness of Dr. Chambers and Mr. Prescott Hewett, I had an opportunity of carefully examining the heart. The right auricle was rather unusually large, the left (which received five pulmonary veins) was singularly small. I could not discover any opening in the *auricular septum*. The right ventricle was remarkably capacious and strong, and communicated with the left (which was unusually small), by an aperture in the *septum* large enough to admit the passage of the little finger. The aorta arose from the right ventricle; it was large and strong, gave off the two coronary arteries in the usual situation, and was complete in all its parts. The *ductus arteriosus* (which was attached to the ascending portion of the aorta about an inch and a half above its origin) was perfectly closed. A small depression was visible at the upper part of the right ventricle, to the left of the aortic orifice;—this hollow was evidently the rudiment of the *ventriculo-pulmonary opening*, but it was completely

* Otto quotes instances where this was observed in the lower mammalia. Legallois noticed it in a rabbit. Otto also saw, in a double monster, an artery from the innominata trunk pass to an accessory lung. (Selt. Beob. Part I. p. 16. I shall have to allude to other instances of this kind).

occluded by a dense membrane of fibrous structure. Immediately above this partition a narrow passage (the ascending portion of the pulmonary artery) extended upwards to the back of the aorta, where it divided into two branches; one of these appears to have passed to the right lung, and the other is again subdivided into two. I believe that the lower of these went to the left lung, and that the upper received blood from some aortic branch. The arterial ligament passed directly from the upper and inferior part of the ascending aorta to the point of divergence of the two main pulmonary branches. In the portion of the aorta which remained, there were not any vessels which could have conveyed blood to this rudimentary pulmonary artery, but this must, in all probability, have been supplied by branches arising either from some of the large vessels of the arch, or from some part of the descending thoracic aorta. That the supply was received from the latter source is rendered extremely improbable by the fact that, immediately distal to the left subclavian artery, the tissues of the aorta were considerably thinner and weaker than they were a short distance above this point, rendering it probable that a somewhat diminished current of blood passed through the descending part of the main artery. Two or three of the orifices of the superior aortic intercostal arteries were left attached to the vessel, (which had been divided obliquely immediately anterior to the origin of the left subclavian): these were of the natural size: hence the further improbability that the pulmonary artery received any supply from the aorta below its arch. The origins of the brachio-cephalic and left carotid arteries appeared of the ordinary dimensions, and did not seem to have been capable of admitting more than the usual quantity of blood. The left subclavian had been severed, and therefore it was impossible to judge of either its dimensions or its course, but the obliterated state of the arterial duct, the undilated condition of the brachio-cephalic, left carotid, and intercostal arteries, and the thinness of the coats of the descending aorta, appear to render it a perfectly fair conjecture that the pulmonary artery could not have received its supply of blood from any other source than the left subclavian.

Whatever uncertainty may attach to the manner in which the pulmonary artery was supplied with blood in this remarkable case, its detail affords one or two extremely interesting facts—as shewing for how long a time an individual may survive, although suffering from so extreme a malformation, and as proving that the natural closure both of the arterial duct and the ovalian foramen may be effected, even in a case where there is complete occlusion of the pulmonary ostium. The heart is preserved in the Museum of St. George's Hospital.

The following case, by M. Bigger,* affords a rare example of absence of the pulmonary artery, the lungs being supplied by a supernumerary vessel from the arch of the aorta. An infant which, from the fifth day after birth, had been affected with cyanosis, died at the age of 5½ months, of convulsions and dyspnœa. The left ventricle and auricle of the heart were found to be much smaller than the right. The *foramen ovale* was still patent. The pulmonary veins were much contracted, but no traces of the [true?] *pulmonary arteries* could be detected. The aorta arose from the septum of the two ventricles, and communicated with both. A considerable *arterial branch*, about the size of the arteria innominata, was given off from the arch of the aorta, and was distributed to the lungs.

In January 1842, Mr. G. Smith detailed, at the Westminster Medical Society, the case of an infant which attracted attention soon after its birth by the dark colour of its skin. It, however, went on pretty well, and appeared in good health, except that it was subject to attacks of crying, with shortness of breath, at which time the blueness of the surface was very much increased. It died suddenly at the age of eight months. The body was plump and fat. The heart was large, the right auricle and ventricle were of great extent, and composed the greater portion of the organ; the right ventricle was more muscular than natural, and the left had the small proportions proper to the right. The *pulmonary artery* was wanting, with the exception of what appeared to be the small rudiment of one going in a direction towards the ductus arteriosus. The aorta, which was very large, arose from both ventricles, the *ventricular septum* being incomplete. The vessels branched off from the arch of the aorta as usual; but, in addition to these, there was an artery about the size of the subclavian arising from the under surface of the arch. This shortly divided into two branches, which were distributed one to each lung. There were three or four small pulmonary veins, which passed to the left auricle. The foramen ovale was open sufficiently to admit a goose-quill in an oblique direction.†

As the precise spot from which the supernumerary branch arose in the two cases just quoted is not mentioned, and as no reference to the arterial duct is made in either, it would be difficult to judge whether the

* Wochenschrift fur die Gesammte Heilkunde, 1839, No. 36; and Edinburgh Med. and Surgical Journal, vol. lv. p. 25.

† Lancet, Jan. 15th, 1842.

lungs were, in reality, supplied by an accessory vessel formed especially for that purpose, by an enlarged bronchial artery, or by the ductus arteriosus, which, owing to the complete obliteration of the ascending pulmonary trunk, would have had the appearance of arising as a distinct branch from the aorta. The latter is most probably the true explanation. At all events, these cases bear the closest physiological relation to those which have been previously quoted.

A few other cases of obliteration of the pulmonary orifice have been recorded, but these are rendered imperfect in their details from the circumstance that the precise manner in which the lungs received their supply of blood was not ascertained, the aorta having been cut through a short distance above its origin. I have recently seen an instance of this kind; but, as I understand that the gentleman in whose practice it occurred intends to publish the case, I shall make no further allusion to it at present.

Dr. Shearman, of Rotherham,* details the case of a delicate female child, ætat. 8 years, who suffered from slight cough, hæmoptysis, and some lividity about the mouth and eyes. The chest was well formed, except that the left side was more prominent than the right. "There was a *flat* sound over a much greater extent than natural in the cardiac region; on a level with the superior margin of the third rib on the sternum, a loud bellows sound with the first or systolic beat of the heart, which was heard louder under the right clavicle, and not under the left; another loud rasping bellows sound at the ensiform cartilage, with the first beat, which decreased in intensity as you left that spot; and another systolic bellows sound at the apex, more feeble than the others, which decreased in intensity higher up the ventricle. There was a slightly perceptible purring tremor over the apex, and very great jugular pulsation in the neck." Every three or four weeks she was seized with hæmoptysis to a greater or less extent, accompanied by symptoms of cyanosis; during the intervals she had very little if any cough, but she was not capable of taking any exercise without producing very rapid respiration and a violet colour of the skin, on which occasions the jugular pulsation was very great. She died of hæmoptysis about nineteen months after she was first seen by Dr. Shearman.

Both lungs were found very full of grey tubercle. The heart, with its pericardium attached, weighed ten ounces. The right ventricle was considered by Dr. S. to be on the left side of the heart, and the right side of the heart constituted the apex, instead of the left. The auricle connected with the tricuspid valve was very large, and had two large venous openings; the other, connected with the mitral valve, was very small and thin, and only one very minute orifice* could be found. The *foramen ovale* was open. The ventricles communicated by an aperture in the septum, large enough to admit the little finger, just at the root of the aorta. The *pulmonary artery*, like a very small vein, opened into that ventricle which was divided from the auricle by the tricuspid valve. The semilunar valves of the pulmonary artery were quite rudimentary, and the author appears to think that the lungs did not receive blood by that route. As the upper part of the aorta was not preserved, the precise manner in which the circulation through the lungs was carried on could not be ascertained.

* Provincial Med. and Surg. Journal, July 30th, 1845.

Lediberder† (quoted by Hasse) found the pulmonary artery closed by a transverse membranous expansion without any aperture whatever. I have failed to obtain a sight of the original narrative.

Mr. Fearn‡, of Derby, relates the case of an infant which had been affected from the time of its birth with considerable difficulty of breathing, drowsiness, and lividity of the surface. It died at the end of the 7th week. The aorta was nearly twice its ordinary calibre. The *pulmonary artery* was much smaller than natural; it did not cross the root of the aorta, but ran along its right side, giving off its branches to the lungs in the normal way. It contained some fluid blood, and its communication with the ventricle was cut off by a thin membrane stretching across its commencement. The upper part of the *septum ventriculorum* was deficient. The *foramen ovale* was open, and as large as in the fœtal state. The right auriculo-ventricular opening would scarcely admit the passage of a probe. The left auriculo-ventricular opening was natural. The ductus arteriosus was closed. This interesting case appears to have been in many respects similar to that observed by Dr. Thomas Chambers. Unfortunately no clue is afforded to the precise manner in which the upper part of the pulmonary artery was supplied with blood—whether from dilated bronchial vessels, or supernumerary branches from the arch or elsewhere.

M. Gintrac cites§ the instance (observed by M. Lordat) of a child, six weeks old,

* In a subsequent communication Dr. Shearman mentions "that the very small orifice, like a minute vein, connected with the small auricle, had, he feels convinced, never been used as a pulmonary vein."

† Bulletin de la Soc. Anat. t. ii. p. 68.

‡ Lancet, Nov. 22, 1834, p. 312.

§ Observations et Recherches sur la Cyanose, ou Maladie Bleu, par E. Gintrac, M.D. p. 201.

which, while tranquil, performed its functions naturally, there being merely a bluish appearance of the upper eyelids; but when it cried, or was otherwise excited, the surface of the body became blue. It died in a paroxysm. The heart was small, and nearly of a spherical shape; the walls of the right ventricle were extremely thickened, its cavity was exceedingly small; the size of the right auriculo-ventricular orifice corresponded with that of the ventricle. The auricles were well shaped, and the *foramen ovale* was large. The *pulmonary artery* was very small, and entirely obliterated at its commencement. The manner in which the lungs were supplied does not appear to have been observed. The case was probably, in most respects, similar to that by Dr. Hunter, already quoted.

In the whole of the recorded instances of congenital obliteration of the ascending pulmonary artery, where the symptoms have been described, there were, to a greater or less degree, cyanosis and other signs of pulmonary obstruction. In the three cases which have occurred recently in some measure within my own observation, life was, I believe, more prolonged than in any other recorded instances, with the exception of that by Dr. Ramsbotham. In these three cases there was anatomical evidence of the fact that a considerable quantity of blood circulated through the lungs. In two the left cavities of the heart were equal in capacity to the right. In Dr. Thomas Chambers's case they were much smaller, but not extremely contracted. In nearly all the other examples which I have cited there were evidences of a very greatly impeded pulmonary circulation. This obstruction is evidently due less to the indirectness of the course by which the blood reaches the lungs, than to the unnatural narrowness of the pulmonary vessels, which, although greatly varying in dimensions in different individuals, are very rarely, if ever, equal in capacity to the pulmonary branches of a well-formed organ. In all such cases the occurrence of slight pulmonary disease may at once determine the fatal issue; but it is certain that—other things being equal—the duration of life in this, as in most other forms of congenital heart disease, depends mainly upon the degree of freedom with which the blood gains access to, or returns from, the lungs.

To render this part of the subject complete, it will be necessary to devote a few observations to the

Offices of the Bronchial Arteries in health and disease.

The physiological properties of these vessels are exceedingly curious, and appear, even in the present day, to be in great measure overlooked by anatomical writers. It has generally been considered that the bronchial arteries are merely intended to convey nutrient material to the pulmonary tissues, and to supply the external and internal secreting surfaces, and the glandular apparatus, of the lungs: and, although cases have been alluded to by authors in which these vessels were absent*, it cannot be questioned that, under ordinary circumstances, their duties may be almost exclusively of this nature. Still, the capillary anastomoses of the pulmonary artery with the bronchials, as described by Ruysch, Petsche, Haller, and Reisseisen, are so free, and the blood contained in the extreme pulmonary branches is so well fitted for all the purposes of nutrition and secretion, that it is difficult to resist the conviction that the true pulmonary vascular system would be alone sufficient to maintain the organic integrity of the parenchyma and other structures of the lungs, and that the bronchial arteries have been superadded for the purpose of performing certain additional and peculiar functions. Indeed, it cannot be questioned that these vessels are designed to fulfil another very curious and perhaps very important office; they appear to be almost equally calculated either to supply the pulmonary artery, where the entrance of blood to that vessel is mechanically impeded, or to afford, to a limited extent, a means of exit to its branches, when the passage of its contents through the veins is rendered difficult by extreme pulmonary or cardiac obstruction†: thus affording a physiological parallel

* That the bronchial arteries may be totally absent, and their functions be efficiently supplied by the pulmonary capillaries, is an opinion fully in accordance with my own views; still, in such cases as those related by Wilson and others, where no bronchial arteries can be discovered in their usual situation, it is scarcely admissible to decide that these vessels are certainly wanting. There are so many ways in which the bronchials may be supplied by anastomoses with other branches in their vicinity; and, as will be presently shewn, so many modes in which additional vessels may be given off to compensate for defect either in the bronchial or pulmonary system, that it is almost impossible to prove the total absence of the latter vessels in any given case, unless the whole of the arteries of the chest, neck, and abdomen, have been minutely examined. Several instances of abnormal distribution of the bronchials have been recorded. Christopher Fichet (Haller, Disputat. Anatom. vol. iii. p. 6) saw a common bronchial artery given off from the subclavian distal to the origin of the mammary; and Ruysch states that he very frequently met with this arrangement. The latter authority also describes that the termination of the upper intercostal artery may pass to the lungs. Cruveilhier has seen a bronchial artery given off by the inferior thyroid, and various other arrangements of these vessels have been observed.

† Reisseisen always found that injection was conveyed from the pulmonary artery into the bronchials with greater facility than from the bronchials into the pulmonary artery. He also observed that by far the greater number of bronchial arteries end in the pulmonary veins.

to the duties performed by the external epigastric and external thoracic veins, and the internal mammary and epigastric arteries, which, under ordinary circumstances, are merely engaged in conveying blood to and from the integumentary and muscular structures in their vicinity, but which are also subsidiary to the cavæ and the aorta: communicating with the great venous and arterial trunks by free though indirect anastomoses, they are capable of undergoing extraordinary dilatation whenever those important vessels become obstructed, of conveying large quantities of blood in a direction contrary to that of their ordinary current, and, in short, of almost entirely assuming the important functions of the main systemic vessels. Hence we have seen that where the pulmonary artery is congenitally occluded, the bronchials, extremely dilated, may, in great measure, fulfil its office; and, on the other hand, where the aorta is congenitally obliterated at the lower part of its arch, the bronchials, and upper intercostal arteries, into which they frequently open, are found remarkably dilated, having evidently taken an important share in conveying blood to that portion of the aorta which is distal to the stricture. It is, of course, evident that, where obstruction of the pulmonary artery or aorta occurs late in life, functions of this kind cannot be fully assumed by the slenderly developed bronchials. Still, it is probable that, even at the adult period, the supplementary action of these arteries is occasionally, and to a limited extent, called into play; and it appears highly probable that, in many cases of great and long-continued pulmonary obstruction, the relief afforded to the over-distended pulmonary artery by its anastomosing vessels is by no means inconsiderable.

With regard to the general pathological relations of the bronchial vessels little is known; and the subject is one which promises to afford an interesting field for diligent inquiry, especially as regards the conditions of these vessels in various forms of pulmonary and cardiac diseases.

It has been argued that hypertrophy of the left ventricle occasionally produces pulmonary apoplexy by the violence with which the blood is impelled through the bronchial arteries; but this appears to be physically impossible: and, in such cases, the rupture of the vessels within the lung is probably due to very different causes: I have, however, occasionally been inclined to attribute the determination of pulmonary congestion, apoplexy, and œdema, to an obstructed condition of the bronchial orifices consequent upon a diseased state of the internal tissues of the aorta. The smallness of the vessels however, renders it difficult to bring this to absolute proof without injecting the parts *in situ*, which can rarely be done. Still I think that this suggestion deserves investigation, as it is evident that any cause which tends to promote congestion or inflammation of the lungs must be greatly aggravated where the vessels which anastomose with the pulmonary arteries and veins are in an obstructed condition*.

In the following case, which I saw in June last, it appeared probable that the great extent of the pulmonary inflammation, and the rapidity with which it involved the whole of both lungs, was to a certain, though probably to a minor, degree, owing to the lesion in question.

A man between 30 and 40 years of age, who appeared to have been previously the subject of tubercular deposit in the lungs, died, after a fortnight's illness, of suppurating pneumonia, involving nearly the whole of the pulmonary tissue, and in some parts assuming almost the characters of gangrene. There was an aneurism of the descending arch of the aorta, which had obliterated the left subclavian artery; and, below this, the sub-epithelial tissue of the vessel was greatly thickened, and otherwise diseased. The orifice of the principal bronchial artery was almost obliterated, scarcely admitting a bristle; externally to the aorta, this vessel was unusually wide.

In a case of remarkable obstructive disease of the aortic valves related by M. Fenema, and quoted by Professor Tiedemann, it is mentioned, that "the first intercostal arteries were narrowed and contracted, and then unusually dilated. The patient—a soldier, æt. 43—had suffered from asthma, and symptoms of great oppression in the chest."

Otto mentions that we sometimes find these arteries very much enlarged in persons affected with cyanosis. He has seen this once. Tiedemann† and others have made the same observation. Otto also refers to a case of aneurism of the bronchial artery‡—a very rare lesion—but an evidence of the activity of the circulation through those vessels.

CONGENITAL NARROWING OF THE PULMONARY ORIFICE.‡

The great frequency of disease of the pulmonary orifice occurring during intra-uterine

* In cases of pulmonary congestion, where the aortic orifices of the bronchial and upper intercostal arteries were almost obliterated by thickening, &c., I have found the trunks of those arteries externally to the aorta unusually wide: but this is an uncertain condition, as it may probably depend upon delay to the passage of the blood through these vessels consequent upon diminished cardiac impulse.

† Zeitschrift f. Physiol. vol. i. part i. p. 111.

‡ A Leprous in Comment. Bonon, vol. i. p. 343 and 353.

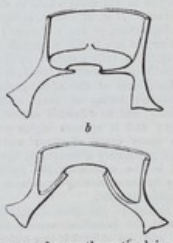
‡ In quoting a very extended series of cases, a

life, compared with similar lesion of the aortic ostium, cannot fail to be observed.* The aorta, it is true, is liable to several lesions of congenital origin, among which the fusion of its valves into a funnel-shaped channel is one of the most striking; but, during intra-uterine life, the functions of the aorta are light compared with those performed by the pulmonary artery, upon which mainly devolves the office of transmitting blood to the lower part of the body, and it is not surprising that, during the earlier months of fœtal existence, obstruction to the circulation, depending, it may be, either upon malposition of the body of the infant, pressure upon the umbilical cord, &c., or upon faulty conditions of the lungs, should determine the occurrence of imperfection or irreparable lesion in the delicate tissues about the pulmonary orifice, while the aorta, being less influenced by these or any other causes of obstruction, escapes uninjured.

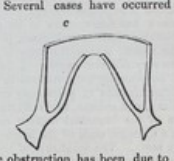
Whenever, in more advanced life, the pulmonary artery is called upon to perform an unusual † or too active function, it is found to be as liable to various forms of structural disease as is the aorta.

Congenital narrowing of the pulmonary ostium has been noticed by a large number of observers; and several writers have, to a certain extent, discussed the pathological relations of this lesion. Among others, Sandiforth, Hein, Louis, Dr. Craigie, and Dr. Stillé, have made it the subject of especial notice. The lesion is not by any means one of very common occurrence, as is proved by the fact that more than two instances of the kind have rarely been noticed by the same observer; relatively, however, it cannot be regarded as by any means rare, for it presents itself more frequently than does any other of the graver kinds of malformation to which the heart is liable. I have met with notices of about 60 well-marked instances of the kind, and might, doubtless, have obtained histories of many others, had it appeared necessary to extend my search still further.

Congenital arctation of the pulmonary orifice may occur in an almost infinite variety of forms. Occasionally there is found a very narrow passage, either confined merely to the ostium of the vessel, or involving also a portion of its ascending canal. This strait is generally rendered uneven by warty or ossific elevations. In other cases the orifice is less narrow than in the preceding instances, its sides being furnished with indistinct traces of rudimentary valves. Cruveilhier, Craigie, and many other observers, have described cases where the pulmonary orifice was furnished with an imperfect valvular apparatus, in the form of a transverse or conical partition, having an aperture in its centre, and presenting a smooth surface below; but above, three slightly elevated ridges which are apparently rudiments of the free edges of sygmoid valves (diagrams *a* and *b*). This is the state



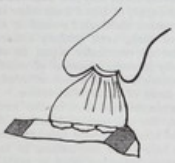
of parts most frequently noticed in cases of congenital narrowing of the pulmonary orifice. A very similar appearance is occasionally seen in the aorta. In some instances the mouth of the artery is occupied by a thimble-shaped membrane, perforated at its apex, and smooth on both surfaces (Fig. *c*). Several cases have occurred in



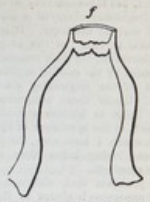
which the obstruction has been due to excrescences adhering to the valves. An instance is noted by Kreysig,* where the pulmonary artery was narrowed only on one side. In an example by Nasse, quoted by M. Hasse, the pulmonary orifice was blocked up by fleshy columns hardly separable from each other. A similar condition is described by Dr. Elliotson, as occurring in a case where the walls of the ventricle appeared to have grown up around the mouth of the pulmonary artery, forming a short narrow canal, leading to the orifice of the vessel, which was of the usual size (an appearance which may be represented by the diagram *d*).



In a case related by Mr. J. Marshall, the valves were found in a contracted and inefficient state, and the vessel above presented a singular contraction. The diagram (*e*) is copied from Mr. Marshall's paper. In some instances, the vessel has been found considerably, but not extremely, narrowed, and either destitute of valves or furnished with only two sygmoid curtains.



In cases where the opening is extremely narrow, and life is prolonged for several years, the constricted part usually has a tendency to be forced upwards by the impulse of the ventricle. We have seen that in some of these instances, where the orifice is closed by a perforated membrane, this becomes extended into a funnel-shaped tube; in others the structures at the base of the vessel become rather elongated, and then the rudimentary valvular ring, which is the seat of obstruction, has an appearance of being situated unusually high up in the canal. In other cases a portion of the muscular tissue of the ventricle immediately at the root of the artery becomes stretched upwards, and may form a little supplementary muscular cavity below the pulmonary orifice, as figured in diagram *f*.



Lastly, the artery may simply be diminutive in size, but complete in all its parts. It is probable, however, that, in the cases of this kind which have been observed, the tissues of the arterial orifice have been rendered contracted and unyielding by previous disease; as it is clear that, providing the whole structures were healthy, the size of the artery would not fail to be proportioned to that of the ventricle.* Several other forms of contraction of the pulmonary orifice have been observed. These will be noticed in the details of the several cases.

The whole of these morbid conditions are evidently due to lesions of the vessel occurring at a very early period of intra-uterine life; the obstruction thus produced checking the further development of the parts, and frequently preventing also the due formation of the ventricular and auricular septa, and other portions of the cardiac apparatus, which are at that period either wholly undeveloped, or but imperfectly formed.

It has been remarked by several authori-

large number of which correspond in many particulars with each other, I have, of course, been compelled to abridge many of the narratives, merely retaining, in each, those details which best illustrate the nature and effects of the lesions observed in the pulmonary artery and remainder of the circulatory system, and omitting many facts which certainly add much to the pathological value of most of the cases as they appear in the original works. The utmost that I have been able to effect, consistently with due brevity, has been to retain the most prominent and essential features of each case in such a manner that the perusal of the whole may furnish the reader with a complete view of all the symptoms which attend cyanosis, and of all the effects which have been noticed to result in the system from congenital malformation of the pulmonary artery. For the entire details of each case I must refer my readers to the original narratives.

* In nineteen cases of congenital malformation of the heart, described by M. Louis, there was contraction of the pulmonary artery in ten; but of the aorta, only in one.

† As in cases where the main arteries communicate freely by the ductus arteriosus.

* Herzkrankheiten, vol. iii. p. 104, quoted by Otto.

* It may be supposed that the small size of the pulmonary artery is here the result of the diminished quantity of blood which passes through it to the lungs in consequence of the open state of the ventricular septum; but this view would generally be erroneous—as, in those numerous cases where, the pulmonary artery being contracted, the aorta is given off either from the right ventricle, or midway between the two cavities, there is not the slightest reason to believe that blood ever passes completely through the septum from right to left, however freely it may do so in a contrary direction; in these cases also the ovalian foramen is not unfrequently closed. In hearts which I have seen, where a free opening—apparently the result of ulceration—had long existed between the ventricles, the pulmonary artery was not diminished in size. In a few instances, however, (as for example in that which will presently be quoted from M. Schuler, where contraction of the pulmonary orifice was attended with an open state of the foramen ovale and arterial duct, the ventricular septum being complete), the narrowness of the vessel may be, in part, owing to the diversion of the stream into another course.

ties, that, where the pulmonary valves are concreted, rendering the orifice of the vessel extremely narrow, the artery above is not contracted, but is occasionally rather dilated. I have frequently observed a similar condition of parts in the aorta. It is evidently attributable to delay of the blood in that part of the vessel, the fluid remaining almost uninfluenced by the cardiac impulse in consequence of the unyielding state of the ostium. Under these circumstances the aorta is liable to become considerably dilated; but, in the specimens of the lesion in question which I have seen, the pulmonary artery was rather below than above the ordinary standard.

It is probable that, although a normal valvular apparatus is absent in these cases, regurgitation of blood into the ventricle occurs in but a very small proportion of instances. The arterial aspects of the contracted orifices are occasionally clothed with small distinct masses of organised vegetations, which freely admit of the passage of blood into the artery, but afterwards close together and prevent reflux. A very curious arrangement of this kind is observable in a heart preserved in the Museum of Guy's Hospital. (fig. *g*.) The details of this case



will be mentioned hereafter. In most of the examples where the rudiments of the sygmoid curtains form an elevated cone projecting upwards into the vessel, this apparatus in reality constitutes a very efficient valve. In many of these cases the mechanism of the parts is very similar to that of the small glass ink bottles which are contrived in such a manner as to be carried open in the pocket without escape of the fluid.

The only difference is that the sides of the funnel-shaped valve of the artery (*a*, *a*), require to be pliable, and capable of



being pressed together; were they wholly inelastic, like those of the conical glass tube, (*b*, *b*), they would admit of some degree of



reflux at every ventricular diastole. It, however, rarely, if ever, happens in the pulmonary artery that the malformed valve altogether loses its pliability. In a few cases, where the communication with the ventricle is direct, or where the orifice of the artery is formed by a large circular opening in a transverse membrane, regurgitation may occur; but these are, I believe, exceptional and rare instances.

It is remarkable how extreme a degree of narrowing of the pulmonary orifice may exist without producing fatal interruption to the circulation. I have never seen or heard of any instance where the area of the aortic opening had become reduced to less than the diameter of a common writing-quill, but a considerably greater degree of arctation of the pulmonary orifice has been noticed even in persons above the age of ten years, and this too in cases where it was not observed that any supernumerary arteries communicated with the lungs. There frequently, however, remains a doubt, in such cases, whether the lungs may not have received an indirect supply of blood from other sources, which passed undetected;—and it must be observed that, here, patency either of the auricular or ventricular septa, or of both, is usually present; a means of relief to the right ventricle which is not afforded to the left in cases of aortic obstruction. It will also be remembered that, in the former instances, the entrance of blood to the obstructed vessel is greatly aided by the action of the lungs—the respiration, in many of these cases, being increased in rapidity evidently for the purpose of, in some measure, compensating for the scantiness of the supply of blood to the lungs by the quickness of its transit,—a point which has been very ably argued by my friend Dr. Lloyd. It will be observed that, in cases of this description, the duration of life depends far more upon the strength of the right ventricle, and the expansibility of the lungs, than even upon the size of the vessels by which the pulmonary circulation is carried on—a fact of no small practical moment.

In classifying the cases where the pulmonary orifice has been found congenitally narrowed, reference should especially be had to the relative position which this vessel, the aorta, and the ventricles, are found to occupy in the several instances; as the va-

riations which occur in this respect are extremely marked, rendering the degree of malformation very far indeed more salient in some cases than in others; and especially as it is found that such variations produce considerable difference in the state of the circulation, and distinctly affect the viability of the individuals who suffer from these malformations.

Still it appears preferable to arrange the cases according to the degree of abnormal communication which is observed to exist in the several instances, premising that the modes in which the large vessels are found to be given off from the heart, in cases of arctation of the pulmonary orifice, are as follow:—

1. The aorta arising from the right ventricle, the narrowed orifice of the pulmonary artery being situated to its left, and anteriorly. The septum of the ventricles pierced by a rounded opening, which may either be confined to the upper portion of the partition, or be so extremely wide as to leave the ventricles separated merely by a slightly elevated falx.

2. The aorta, originating behind the pulmonary artery, nearly midway between the ventricles, and above the ventricular septum, which terminates by a crescentic margin below the ostium of the vessel; thus leaving an aperture of greater or less size through which both ventricles communicate alike with the arteries and with each other.

3. The narrowed pulmonary artery and the aorta arising, as usual, from the right and left ventricles, with or without a rounded aperture in the ventricular septum.

Although by far the larger proportion of instances in which these malformations occur are congenital, it occasionally becomes a matter of difficulty to decide whether many, if not the whole, of the abnormal appearances are not the results of comparatively recent disease. In the cases of the first and second orders, just described, such a doubt cannot for an instant be maintained; a single glance will shew that the principal lesions are congenital, although morbid changes of later date may have been superadded; but with regard to the third class, it is often by no means easy to decide;—in such cases an opinion can only be founded upon a minute examination of the structures, and a knowledge of the duration and character of the symptoms during life.

The following arrangements of parts have been observed in cases where the orifice of the pulmonary artery was congenitally narrowed. (1.) The pulmonary circulation supplied by enlarged bronchial arteries. (2.) The auricular and ventricular septa imperfect; the ductus arteriosus remaining permanently open. (3.) The foramen ovale and arterial duct permanently open; the ventricular septum being complete. (4.) The arterial duct and ventricular septum pervious; the foramen ovale perfectly obliterated. (5.) The arterial duct closed, the ventricular and auricular septa perforated. (6.) The ventricular septum, only, incomplete. (7.) Patency of the foramen ovale only. (8.) Entire absence of any unnatural communication either between the cavities of the heart or its great vessels. A collection of examples of each of these kinds of malformation will furnish some interesting data illustrative of the pathology of the circulation.

(1.) *The pulmonary orifice contracted—the lungs supplied by enlarged bronchial arteries.*

Reference has been already made to a class of cases where—the ostium of the pulmonary artery being congenitally occluded,—the supply of blood to the lungs is in part or wholly furnished by dilated bronchial arteries, arising from the descending thoracic aorta. The following case proves that a similar arrangement may obtain where the entrance of the pulmonary artery is merely extremely narrowed. It is, however, the only well-marked instance of the kind with which I am acquainted.

M. Jacobson describes * the heart of a boy who died with morbus cæruleus, and in whom the aorta, which was considerably enlarged, was given off from the right ventricle. The *pulmonary artery* was not more than one-fourth the size of the aorta, its ostium being greatly contracted by thickening of its valves. But, to compensate for this, the *bronchial arteries* were very much enlarged; three of these vessels passed off from the right and left sides of the aorta to the lungs, and a branch was distributed to either lung from the pericardial artery. A large opening existed in the *septum of the ventricles*; the *foramen ovale* was almost closed.

II.—*With patency of the auricular and ventricular septa and ductus arteriosus.*

We have observed that, in by far the larger proportion of instances of non-development or congenital occlusion, of the pulmonary orifice, the arterial duct remains permanently open throughout the life of the individual; still, such an arrangement does not invariably obtain, even in those extreme instances; and, in cases where children are born with great narrowing of the pulmonary ostium, it may be stated, as a general rule, that the obstruction to the circulation thus produced (however extreme it may be) is usually not sufficiently great to determine the permanency of the arterial duct. This is pro-

* Meckel, Archiv. für Physiologie, B. ii. St. i. 13.

bably due to the fact that, to whatever extent the aretation of the pulmonary artery may have gone, the entrance of blood through its narrowed orifice is rendered considerably more free immediately the child is born, and respiration commences. Aretation of the pulmonary orifice is doubtless a source of far greater obstruction to the passage of blood from the right ventricle during intra-uterine life, while the respiratory organs are inactive, than it is subsequently to birth; hence we may probably account for the fact that a degree of obstruction in the pulmonary artery which has been sufficient to determine the persistence of an opening in the ventricular septum, has, in many cases, not been capable of preventing the foramen ovale and the arterial duct from becoming naturally closed. There are, however, several instances on record in which permanence of the arterial duct was attendant upon the form of lesion now under consideration: still, they are few compared with those in which the duct has become obliterated; not amounting, in fact, to more than one case in ten. And it will be observed that in the whole of the following instances, although the canal was pervious, its channel had become, to a certain degree, contracted. And it is probable that in cases of this kind it never retains that width relatively to the pulmonary artery which is observed in the naturally-constructed fœtal heart. This last observation also applies to the cases where permanence of the arterial duct is combined with an obliterated state of the pulmonary orifice. I believe that, neither in these nor in any other class of instances where life has been much prolonged, has the arterial duct been found to exceed, if ever it has been seen to equal, one of the main pulmonary branches of a well-formed individual of the same age.

A remarkable instance of this kind of malformation is recorded by Dr. Cheever*. The patient was a boy, who died at the age of 13½ years. During the first months of infancy his skin began to assume a purple tinge, and his respiration became, at times, laborious. As he grew up, his chest presented an appearance of deformity. Dyspnœa, attended with slight cough and occasional expectoration of blood, continued to increase until Dr. C. saw him: he was then 12 years of age. He was tall and slender, having little flesh on his extremities. The last joints of his fingers and toes were unusually long, broad, and bulbous; the nails hooked, and of a dark purple colour. The surface of the body generally was of a leaden hue; but, on the accession of a paroxysm, some parts, such as the lips, nose, and ears, acquired a very dark purple tint. He was very sensible to cold, and the temperature of his body, when at rest, was below the natural standard, though easily raised by the slightest exertion. There were no marks of a diminished energy of the brain; his intellectual powers were good. Attempts at active exercise produced very severe paroxysms of palpitation and dyspnœa, blackness of the tongue and fauces, and all the most vascular parts of the surface; a turgidity of the vessels of the neck, an engorgement of the countenance, and great heat of the surface*. These symptoms also often occurred unexpectedly when he was not using exercise. The paroxysms above described continued to increase in force and frequency, and were latterly complicated with symptoms of hydrocephalus. His cough became incessant, attended with frequent expectoration of black blood, and great difficulty in breathing. His eyes, before prominent and suffused, now seemed starting from his head; he was paralytic on one side, extremities œdematous, bowels torpid. During his last illness the heat of his body was very variable, often great. His pulse was quick and small, but regular. Deglutition was often suspended, and he had frequent hæmorrhages from the mouth and nose. He died reduced to the last degree of emaciation. Upon examination, the thymus gland was found large and distinct, the lungs very small and dark coloured, and adherent to the ribs on either side, and very firmly to the diaphragm. Tubercles were pretty generally diffused throughout their substance. The cavities of the thorax and pericardium appeared black and gangrenous†, but free from serous effusion. The heart, generally, appears to have been smaller than is usual in persons of that age; its apex was obtuse, and there was a collection of fat upon its surface, which was remarkable, considering the emaciation of the subject. The auricles were distended with blood. The parietes of the right ventricle were so much increased in hardness and muscularity as to render it incapable of containing more than half an ounce of blood. The *pulmonary artery* was nearly impervious; its valves were thickened into a triangular form, and so much constricted as to admit with difficulty the eye of a small probe. The *ductus arteriosus*, about half an inch in length, was pervious, and of the size of a crow-quill. The sides of the left ventricle were thin, and its cavity proportionally increased. There was a perforation in the *septum of the ventricles*, just below the origin of the aorta, large enough to admit the little finger. Through this communication ligamentous bands, resembling the chordæ tendineæ, passed to either ventricle, proving that the perforation was the effect of malformation, not of disease. The *septum* of the *auricles* was entirely wanting.

M. Obet*, describes the case of a boy who became affected with cyanosis in his second month, and died at the end of his 3d year. The lungs were found to be sound, the right auricle was greatly larger than the left; the rest of the heart was of usual size. The *foramen ovale* was open; the *pulmonary artery* very small; its tunics were thin, like those of a vein, and its orifice so contracted that it would scarce admit a probe; it arose a little in front of the septum of the ventricles. The *canalis arteriosus*, which was very narrow at its origin, opened into the left subclavian. The *septum of the ventricles* was deficient towards the base of the heart. The aorta—larger than natural—arose above the septum in such a manner that one of its sigmoid valves corresponded with the right ventricle. The aorta gave off its primary branches in an irregular manner, and passed to the right of the trachea, the pulmonary artery passing to the left.

III.—*The ductus arteriosus and foramen ovale patent; the ventricular septum complete.*

In the following cases the narrowing of the pulmonary orifice must have occurred much later than in the instances just quoted.

M. Bapt. Schuler† mentions a male infant whose nails and lips became blue a few days after birth, and who lived only 7 weeks. Upon examination, the lungs were found of a blackish purple colour. The right auricle was distended with blood, and its walls were thicker than natural. The venæ cavæ and coronary veins were also distended. The *foramen ovale* was widely open, and its valve reticular. The walls of the ventricles were very fleshy, especially those of the right, the cavity of which would scarcely admit a small nut. The valves of the *pulmonary artery* coalesced and projected upwards, leaving but a very contracted opening. The orifice of the aorta was much wider than natural, and its valves thickened. The *arterial duct* was narrow. Many of the mesenteric glands were in a state of scrofulous degeneration.

The above case nearly resembles those already detailed in which closure of the pulmonary orifice was followed by extreme contraction, nearly approaching to obliteration, of the cavity of the right ventricle; the larger proportion of the blood, finding no early exit from that receptacle, passed freely through the wide foramen ovale into the left cavities, whilst the pulmonary ventricle became extremely small. If, however, this contracted state of the ventricle was not rather apparent than real, it is probable that the lungs received a supply of blood from accessory vessels which escaped M. Schuler's observation, as the pulmonary circulation could scarcely have been adequately maintained through the narrow ventricle and slender arterial duct.

M. Seiler* observed the case of a man, æt. 29, in whom the chest was found to be deformed, its cavity being much narrowed. The lungs were small, collapsed, of a livid colour, almost approaching to black, and pressed into the posterior part of the cavity by the enlarged heart. This organ was three times larger than natural; its walls were much thickened, those of the left ventricle were thicker than the thumb, and presented traces of ossification. The cavities were full of blood. The *foramen ovale* and *arterial duct* were pervious, the former very freely. The *pulmonary artery* was very narrow, its valves ossified. The aorta was very large; its tunics were not thinned.

IV.—*The foramen ovale obliterated, the ventricular septum and arterial duct pervious.*

An instance of this description is related by Dr. Houston†. The subject of his case was a boy, æt. 3 years, who died of phthisis and bronchitis. There was violent action of the heart, but the pulse was feeble and the skin cold. The face, hands, and feet, were of a dark livid hue; this colour had not been habitual, and "had only come on when he laboured under pectoral affections." It could not be ascertained whether violent exercise produced the same effect. The right auricle was found to be large, the *foramen ovale* closed. The *pulmonary artery* was about half the usual size, and furnished with two semilunar valves. A smooth defined hole, large enough to admit the little finger, and twice as large as the pulmonary orifice, led through the *septum*, from the right ventricle into the upper part of the left ventricle. The left auricle was small. The left ventricle of the same size and thickness as the right. The aorta was unusually capacious; its valves were perfect. The

* New England Journal of Medicine and Surgery, vol. v. p. 218, 1821.

* In some other cases of cyanosis the body has been noticed to be liable to intense flushes of heat.

† This was probably an excessive degree of the ecchymosed appearance of the thoracic serous membranes which has been observed in some other instances of cyanosis.

* Bulletin des Scien. Med. réd par Grapenon, t. iii. p. 65, 1808.

† Diss. de Morb. Cœrul. Œniponte, 1810, quoted by Hein.

* Horn. Nues. Archiv. Bd. ii. Heft. ii., 1803, quoted also by Corvisart as from "a thesis read at Wittenberg."

† Dublin Hospital Reports, vol. v. p. 322.

ductus arteriosus was diminished in size, but not obliterated.

These are the only examples with which I am acquainted where permanence of the ductus arteriosus has been observed coincidentally with narrowing of the pulmonary orifice. In the whole of the instances of this lesion which I shall next have to cite the duct was found impervious.

V.—*The pulmonary artery small. An abnormal communication existing between one of its branches and the subclavian.*

It may be considered that, in the following instance, as well as in the case by M. Obet already cited, the arterial duct remained pervious; but it appears certain that, in both of these hearts, the communicating vessel was not in reality the canalis arteriosus, but the remains of the communication which, at a very early period of intra-uterine life, while the circulation of the upper portion of the body is carried on through the branchial arches, naturally exists between the vessels which afterwards form the right and left pulmonary and subclavian arteries. The occasional permanence of this communication between the canals in question has been very clearly described by Dr. Paget.*

M. Holst† relates the case of a girl who enjoyed perfectly good health until she was two years old, when her skin began to assume a blue appearance, especially at the parts most distant from the heart. She then experienced feelings of suffocation, giddiness, and spasms. These attacks were usually followed by sleep, after which the blueness of the skin was less. Violent palpitations of the heart, with difficulty of breathing, coldness, and frequent epistaxis, gradually succeeded; and though the frequency of the seizures diminished, yet their violence and duration gradually increased. During the paroxysms, the left side became colder than the right, and the pulsations of the arteries at the wrist of the left arm could not be felt. She died suffocated in one of these attacks. The heart was found very large; the right side larger than the left. There

* Since the appearance of the preceding section of my paper I have met with a report of the following case of extreme *narrowing of the pulmonary orifice attended with an unclosed state of the auricular and ventricular septa and arterial duct.* The subject of this rare malformation was a boy, who, for the first two years of his life, did not manifest any peculiarity of appearance or disorder of function beyond occasional attacks of bronchitis, during which his face appears to have assumed a congested appearance. When he had entered his fourth year Dr. John Bell, who narrates the case, found him suffering from the usual symptoms of the blue disease, but apparently not in a very extreme degree. The sternum and anterior part of the thorax were unduly prominent. The pulse was frequent, and rather full, but easily compressed. "Impulse and the bellows sound well marked over the sternum. Dulness on percussion was of limited extent." The respiration was hurried and panting, but he usually ran about and amused himself like other children of his age. From this period up to the time of his death, which occurred when he was somewhat under the age of 8 years, he continued to suffer from repeated attacks of "pulmonary oppression and congestion," which, within the last two years of his life, became more violent. The prominence of the sternum and left side of the chest went on increasing; the distension of the jugulars and their pulsations were still more perceptible. Usually, the [heart's] sounds were not strong, although those of regurgitation were quite distinct. During a paroxysm there was, however, a loud bellows and rasping sound, with another less evident, and comparable to a subdued gurgling or churning. The pulse all the while was frequent, but without force. At two different periods, together with great præcordial and pulmonary compression, dyspnœa, &c., there was tympanitis, with obstinate constipation, and anasarca, against which all the usual diuretics and means of indirect reduction were utterly powerless. But the abstraction of a few ounces of blood, either from a vein or locally, exerted an immediate and beneficially controlling influence over the disease. The common purgatives, diuretics, and expectorants, would then manifest their customary effects. Little or no benefit was derived from digitalis. The appetite was generally good, and the craving for various articles, some of them of an indigestible nature, inconveniently great. He died somewhat unexpectedly, after having in great measure recovered from two very severe attacks, which were relieved by venesection. It was found that each pleural cavity contained five or six ounces of serous fluid, the pericardium a small quantity. The heart was very much enlarged in consequence of the hypertrophy of its right ventricle, the parietes of this cavity being twice their ordinary thickness. Nothing peculiar in the appearance of the right auricle. The left cavities were atrophied, but otherwise natural. An opening about half an inch in diameter existed at the upper part of the *ventricular septum* common to the two ventricles and the aorta. The ascending aorta was nearly twice its natural size. The *ductus arteriosus* was open, and large enough to admit a goose-quill. The pulmonary artery was of its natural size at the entrance of the arterial duct, but gradually tapered towards the right ventricle, with which it communicated *directly* by an opening so small as scarcely to admit the introduction of a common probe. The *foramen ovale* was not closed, but evidently not sufficiently open to allow of any deleterious admixture of venous and arterial blood.
(As quoted in the Bullet. de Sci. Méd. August 1845.)

The extreme minuteness of the right ventriculo-arterial opening, and the absence of any valvular appearance at its mouth, led Dr. Bell to consider that, during life, blood passed from the pulmonary artery into the ventricle, and not in the contrary and usual direction. This opinion is certainly favoured by the fact of the existence of a regurgitant sound during the paroxysms.

* On the Congenital Malformations of the Heart, Edinburgh Medical and Surgical Journal, vol. xxxvi. p. 287; an essay to which (in common with most recent writers on malformations of the heart) I am indebted for much valuable information.

† Hufeland's Journal, Jan. 1837; quoted in Amer. Journ. of Med. Science, vol. xxi. p. 457.

was communication between the two auricles, not only by the *foramen ovale*, but also by an abnormal opening. The aorta and pulmonary artery arose from the right ventricle. These arteries and all the others were smaller than natural. The left subclavian artery arose from the upper angle of a triangular sac, into the two other corners of which opened the left vertebral artery and an abnormal canal, considered by M. Holst to occupy the place of the *ductus arteriosus*. By this last the subclavian artery was in communication with the left branch of the pulmonary; but, as this canal was very small, but little blood could enter into the subclavian artery, which seemed to have received most of its supply from the left vertebral: so that the blood, in getting from the aorta to the left subclavian, must have passed through the carotids and the circle of Willis. This condition of parts was considered to account for the occasional cessation of the pulse, and the diminished temperature of the left arm.

VI.—*The auricular and ventricular septa perforated—The arterial duct closed.*

Many instances of this kind of malformation have been observed; and, in these, although the arrangement of parts has been in all physiologically similar, the degree and form of the congenital lesions have varied in an extremely remarkable manner. Thus, in one case, the arterial duct was pervious at its aortic end, though closed towards the pulmonary artery. In several instances there has not existed the slightest trace of an arterial canal; in others, the septum of the auricles has been found nearly complete, the foramen ovale being furnished with an efficient valve, while the ventricular septum was almost totally absent. In a fourth set, the main imperfection has existed in the auricular partition; and in a fifth, great contraction of the pulmonary orifice has been coincident with a very moderate amount of communication between the right and left cavities. These diversities of structure have clearly depended, in some cases, upon the period of intra-uterine life at which the lesion of the artery has occurred; in others, upon the degree of obstruction established in the vessel.

In the following example, observed by M. Caillot*, the malformation was only in a small degree removed from that which was found in the instances by Dr. Cheever and Dr. Bell, already cited; there the auricular and ventricular septa were imperfect, and the arterial duct pervious, but greatly contracted: here the heart was similarly malformed, and the canalis arteriosus was barely closed.

* Hasse, Diss. de Morb. Cærul. Lips. 1813; quoted also by Corvisart and M. Hein.

A girl, who had been cyanosed from birth, died with hydrocephalic symptoms at the age of 11 years and 7 months. The heart was found obtuse at its apex, and with a considerable deposit of fat on its surface; it was but little larger than natural. The *pulmonary artery* arose rather more to the left than usual; its coats were thin; its valves were cartilaginous, and so contracted together as scarcely to leave an aperture half a line in diameter. The aorta rose above the *ventricular septum*, which was deficient at its upper part; the vessel principally communicated with the right ventricle. This cavity was larger than the left, and its walls were stronger. The free edges of the tricuspid valve were cartilaginous. The *foramen ovale* was widely open. The *ductus arteriosus* was patent as far as the pulmonary artery, where it was closed by a cicatrix. All the veins were distended with blood; one of the cavæ was three times larger than the aorta. The upper part of the chest was contracted. The structures of the lungs were condensed, and were in an advanced condition of phthisical degeneration.

In the next examples which I shall quote there were found traces of a malformation similar to that observed by MM. Obet and Holst; viz. a communication between the pulmonary and left subclavian arteries. In these instances, however, the abnormal channel had become completely obliterated.

M. Caillot observed* the case of a boy, ætat. 3 years, who began to shew indications of morbus cæruleus at the age of two months. After death, the ends of his fingers only were of a livid colour, approaching to black, but spots like ecchymoses were spread over various parts of the body. The heart was placed transversely, its basis being situated unusually to the right. The right auricle was so distended as to equal the remainder of the heart in volume. The cavity of the right ventricle was small, and its walls of due thickness. The *pulmonary artery* was very narrow. There was a large opening in the *ventricular septum* above which the aorta originated. The *foramen ovale* was closed. The *ductus arteriosus* (branchial canal?), which was closed, extended towards the left subclavian. The aorta took a course above the right bronchus, and behind the trachea, towards the left of the vertebral column. The right carotid and subclavian arteries arose by separate orifices.

M. Duret† narrates the history of a child, 11 years of age, who was cyanosed from his earliest infancy; and, when sixteen months

* Bullet. de l'Ecole de Méd. 1837, p. 21.
† Quoted by Corvisart.

old, began to suffer from convulsive movements of the body, and fainting, during which attacks the face became completely of a violet hue. These paroxysms were subsequently produced by any unusual effort or mental emotion. Upon examination, the heart was found to be larger than natural; the aorta arose from the *septum of the ventricles*. The commencement of the *pulmonary artery*, which had but two sigmoid valves, was much contracted. Just above the valves the vessel was still more contracted; but, higher up, its diameter was larger: its coats were thinner than natural. The *foramen ovale* was open, and about four lines in diameter. The *canalis* arteriosus (branchial canal?), which was completely obliterated, terminated in the left subclavian artery, from which arose the carotid of the same side, while the right subclavian and carotid originated from the aorta by distinct openings.

Several cases of this kind have occurred in which no trace of an arterial duct could be discovered; a negative fact, it is true, but one of great interest and weight, coming, as it does, from a number of very accurate observers.

Sandifort has given* the history of a boy who suffered from cyanosis, dyspnœa, pulsations in the neck, a tumid and livid condition of the face, constant shiverings, occasional epistaxis, which relieved his symptoms, a foul state of the tongue, and fœtid breath. The urine was red, and the bowels costive. These symptoms commenced at the beginning of the child's second year, and continued to increase until the time of his death, which occurred at the age of 12 years. The lungs are described to have been found compressed by the enlarged heart, which was full of blood. The pericardium contained some serum; the right cavities were greatly enlarged. There was an aperture in the *fossa ovalis* which would admit a probe. The aorta arose from both ventricles over an aperture in the *septum*; the right ventricle was dilated. An excrescence on the valves of the *pulmonary artery* had so far closed the orifice of that vessel that it would only admit a small probe. There was no *ductus arteriosus*. The lungs were small, but not diseased.

Dr. Knox† relates the case of a female child, 4 years of age, who had remained in good health until six months after birth, when a blueness of the surface was remarked, particularly after any exertion, with constant difficulty of breathing. On inspection, the lungs appeared to be sound, though dark coloured. The *foramen ovale* was pervious and pretty large. The *pulmonary artery* arose from the right ventricle, its coats were thin, and it was about the size of a goose-quill in its diameter: it possessed all the characters of the artery, such as its valves, and its division into two branches. The aorta arose from both ventricles in such a manner that the ventricles communicated with each other, and with the cavity of the vessel. Not the smallest vestige of the *ductus arteriosus* could be discovered; it seemed never to have existed.

Dr. E. Lexis records* the case of a girl who was born apparently sound and strong, and who presented nothing remarkable for the first year after birth, except, now and then, a difficult asthmatic respiration. Afterwards, severe symptoms of morbus cæruleus presented themselves, the little patient becoming liable to paroxysms of strong suffocation, which she endeavoured to relieve by bending the body forwards. These symptoms were subsequently aggravated by convulsive movements, and painful darting sensations in the limbs. The pulsation of the heart was very strong, but irregular in quality and frequence, and sometimes so small that certain pulsations could not be distinguished. The breath was very foul. The appetite was irregular, and the thirst increased. She died at the age of 5½ years. The left lung was adherent to the costal pleura; the pulmonary tissue contained a few tubercles. The heart was enlarged, the parietes of the right ventricle thicker than those of the left. There was an opening in the *auricular septum* large enough to receive a swan's feather, but closed on the left side by a valve. The aorta, of the usual size, originated above an opening in the *ventricular septum* large enough to admit the tip of the index finger. A probe passed into the *pulmonary artery* entered the ventricle with difficulty. Between the edges of the artery and the semilunar valves there were found a number of blind sacs, into which the probe passed more than half an inch. The communication of the pulmonary artery with the ventricle was about the diameter of a crow-quill. The *arterial duct* was absent.

In a fœtus of about 8 months, Otto† found the heart placed perpendicularly in the chest, and rather to the right. It was of the usual size, except that the right auricle was much dilated. The *foramen ovale* was large enough to admit a small pea. The left auricle was about a quarter as large as the right, and had no communication

* Obs. Anat. Path. cap. i.
† Edinburgh Medical and Surgical Journal, vol. xi. p. 37.

* Lancet, 1845-6, vol. ii. p. 433.
† Seltene Beob. z. Anat. Phys. u. pathol. gehör. Bresl. 1816, p. 11. Quoted by Hein.

with the left ventricle. There was no *ventricular septum*; both sides of the heart were of equal strength. The auriculo-ventricular valve was well-formed. The aorta was situated more than usually to the right. The *pulmonary artery* had three semilunar valves, was about one-third as large as the aorta, and arose to the left of that vessel; it gave off two branches to the lungs, but did not form a *ductus arteriosus*.

It will be observed that in at least three of these four cases in which the ductus arteriosus was absent, the pulmonary artery appears to have been imperfectly developed and generally diminutive, and not merely contracted at its orifice, as is usual in most of the forms of arctation of this vessel. The same peculiarity will also be noticed in some other instances of absence of the arterial duct, to which reference will be made in the next section.

It can scarcely be doubted that, in these cases, the contraction of the pulmonary artery occurred less as a result of structural lesion in the vessel, than as a consequence of the non-developement of the arterial duct; in fact, as a natural provision by which the entrance of an undue quantity of blood to the lungs during intra-uterine life was entirely precluded. In a fœtus where the pulmonary artery was extremely narrow, and the auricular and ventricular septa remained pervious, a ductus arteriosus would be scarcely at all required, of whatever importance such a communication might prove subsequently to the commencement of respiration. And there can be little doubt, that in the four cases related above, this canal became obliterated and absorbed at a very early period of intra-uterine existence.

I believe that the fact of the duct being absent in these instances, and pervious in those related by Dr. Cheever and Dr. Bell, is explicable upon the grounds which I have already hinted at:—in the class of cases just described, the non-development or early closure of the duct appears to have been the primary lesion, whence resulted the formation of a very narrow pulmonary artery, and the persistence of the auricular and ventricular openings; while, in the other examples, the principal lesion seems to have been disease of the pulmonary osteum occurring at a time when the arterial duct was fully formed, but while the inter-ventricular partition was still imperfect.

The condition of the ventricular portion of the heart in Otto's patient shews that the case was nearly allied to those instances already cited, in which absence or obliteration of the pulmonary orifice determined the non-development of the septum cordis—leaving the efferent portion of the organ essentially in the condition of a single ventricle. In the four following cases there is every reason to believe that the arterial duct had been open previously to birth, though the obstruction to the pulmonary circulation subsequently to the commencement of respiration was not sufficient to prevent its closure at a later period. In this particular these cases differ from that by Dr. Mauran already noticed. I have before shewn the reasons why patency of the duct should be far more frequently observed in cases of obliteration of the pulmonary orifice, than in others where this opening is much contracted.

Dr. Farre* narrates the case of a person of the male sex, 22 years of age, whose figure was diminutive, and appearance and mental faculties puerile. The face was of a deep violet colour. In proportion to his height, the size of his abdomen and capacity of his thorax were large. When at rest, his breathing was easy, but rather quicker than natural; he occasionally took a full inspiration with perfect freedom. Whilst capable of taking exercise, slight exertions sufficed to bring on dyspnœa and cough. There was jugular pulsation; the superficial veins were enlarged and varicose. His ankles were œdematous, and there was sometimes vertigo. The pulse was observed, for a few weeks previously to his death, to range from 100 to 120; respirations from 26 to 28. The temperature of the hand was 98°, beneath the tongue 100°; while that of the room was 76°. The bowels were stated to be regular, except during the winter previous to his death, when some blood was discharged without any relief of symptoms. His urine was high coloured, offensive, and scanty. Death occurred rather suddenly (after a gradual failing of the muscular power), subsequently to taking a rather fuller meal than usual. On examination, the extremities were found to be slender: height 4 feet 3 inches. All the characters of puberty were wanting. Gangrene had commenced in one of the great toes. The pleuræ were universally adherent; the lungs were free from tubercles; their cellular tissue was coloured with blood. The veins of the chest were unusually congested, but the inferior cava and coronary veins only were dilated. The auricles were of due size; the valve of the *foramen ovale* was of due size, and efficient. The auriculo-ventricular orifices were narrowed; the right admitted the points of two fingers, the left the point of only one finger. The tricuspid and mitral valves were otherwise malformed. Both auricles opened into a ventricle, which was not only more capacious, but thicker in its walls than natural. The *pulmonary artery* arose from the superior and central part of this ventricle by a very narrow mouth

* Op. citat. pp. 37-40.

of dense structure; its semilunar valves were concealed by a warty excrescence which grew from them, leaving a passage barely large enough to admit a small probe. The rest of the artery was of its proper size. The *ventricular septum* was wanting, and instead of the left ventricle, a pouch, impervious towards the left auricle, was continued from the right ventricle, and opened anteriorly into the aorta by a free orifice. The *ductus arteriosus* was closed. The left vertebral artery arose from the aorta between the left subclavian and carotid arteries. In the descending aorta no branch remarkable for its size appeared as low as the fifth intercostal. All the abdominal viscera were so greatly congested as to appear of a deep claret colour.

M. Vallete has related* the case of a female child born at the full period, who, for six months, remained in appearance healthy, when, all at once, the skin presented a blue tint, and the respiration became difficult. When six months old, convulsions took place, and were succeeded by hemiplegia of the right side, which, however, gradually decreased and finally disappeared, still leaving that side somewhat weaker than the opposite. The cyanosis persisted, increasing when the patient ran or walked quickly, and being deeper on the right than on the left side. At the age of 6 years the patient caught cold, which produced bronchitis and fatal pulmonary congestion. It was found that the ventricular parietes were, on both sides, about two lines in thickness. A large communication, through which the thumb could easily be passed, existed between the ventricles—the circumference of this opening was rounded and smooth; the *septum* was only four lines high, the aortic orifice was normal, and situated at the internal and upper part of the circumference of the inter ventricular opening. The orifice of the *pulmonary artery* was separated from that of the aorta by a small eminence which formed the upper part of the circumference of the inter-ventricular opening. The *pulmonary artery* was about half the size of the aorta; neither it nor its orifice presented anything irregular. The auriculo-ventricular orifice was single, common to the auricles and ventricles, situated at the base of the cavity of the ventricles, behind the aorta and pulmonary artery; it was furnished with a large triangular valve, the base of which was inserted into three-fourths of the circumference of the opening, and the summit of which was fixed, by means of small fleshy columns, to the posterior walls of the ventricles. The valve was large enough to close the orifice completely. The auricles communicated freely by two openings.

* Gazette Médicale, and Lancet, cir. Feb., 1845.

M. Hein relates* the case of a lad, æt. 16, who was very severely affected with cyanosis, having been livid from the earliest period. His chest was narrow, and his breathing short and difficult. There was strong palpitation of the heart, the pulse was 100 and hard, becoming weak and still quicker when oppression was greater, and latterly increasing still more in rapidity when the symptoms were alleviated. He had suffered from copious hæmoptysis, but died from accumulation of mucus in the air-passages. The lungs were found extremely congested, flaccid towards their bases, and small compared with the size of the heart. The left contained a vomica. The bronchial glands were hard and enlarged. The heart was unusually large. There was merely a vestige of a *ventricular septum*. The two sides of the organ were of nearly equal thickness. The valves of the *pulmonary artery*, coalescing, formed a little ring, about three lines in diameter, which projected into the artery. There were three openings in the valve of the *foramen ovale*. The *arterial duct* was closed.

The most extreme degree of this kind of malformation was observed in a heart shewn to Dr. Farre by Mr. Lawrence (in the collection at St. Bartholomew's). Here the venæ cavæ and pulmonary veins opened into their respective auricles, but the *auricular septum* was very imperfect, consisting only of a small muscular band, which left a large foramen ovale without a valve. The *septum of the ventricles* was altogether wanting, so that the ventricle communicated with the auricles by a single ostium, around which a valve was attached. The aorta and pulmonary artery, the entrance of the latter being somewhat contracted, arose side by side from the left part of the ventricle. The history of the case was unknown; but, from the size of the heart, it appeared probable that the child had lived some months.†

Cases of this class are found to vary exceedingly with respect to the size and form of the inter auricular communication: in some, the septum has been found either wholly deficient or merely in a rudimentary state, while in many others it was evident that the process which closes the foramen ovale subsequently to birth had been commenced in the natural manner, and had been arrested only at the period when it was just verging upon completion. The two following cases are fair examples of the extreme degrees of this species of malformation.

In a female child whose complexion was always more or less blue, who suffered from difficulty of breathing, and died at the com-

* De istis Cordis Deformationibus quæ Sanguinem Venosum cum Arterioso Misceri permittunt. Gotting. 1816, p. 37.

† On Malformations of the Heart, pp. 30-1.

pletion of her first year, Mr. Ring found that the heart was furnished with two descending venæ cavæ, one of which terminated in the right, the other entering the left side of the auricle (which was single, the *auricular septum* being totally absent), with the ascending cavæ. The *septum of the ventricles* was also remarkably imperfect. The aorta arose from both ventricles. The *pulmonary artery* was much smaller than usual.*

As a contrast to this case we may take that described by Dr. Nevin.† A boy, æt. 10 months, first began to suffer from symptoms of blue disease about a month subsequently to birth. Death occurred during a violent paroxysm. The heart was found larger than usual; the coronary veins were remarkably turgid; the aorta, at its origin, was considerably enlarged, and arose equally from both ventricles. The *pulmonary artery* was remarkably contracted, and its valves were firmly attached by their apices to the internal coat of the artery, and appeared somewhat ossified at their bases. The *arterial duct* was closed. The right ventricle was considerably enlarged. The *foramen ovale* admitted a large probe. The carotid and subclavian arteries on each side arose by separate trunks.

In other examples narrated in this section, the inter-auricular communication seems to have been as narrow as in the instance by Dr. Nevin, and perhaps even narrower. Indeed, the existence of a very wide communication between the auricles appears in these cases to be a somewhat rare exception to the general rule, and the great width of the aperture may, in some instances, be attributed rather to dilatation occurring subsequently to birth, than to an original arrest of development.

The following are interesting examples of the varieties, both in the anatomical condition of the parts, and in the resulting symptoms, which cases of this description are wont to present.

Dr. John Crampton‡ relates the following instance:—

A boy had, from his birth, been subject to palpitation, attended with pain in the left side. His complexion was livid and sallow; the whole surface of his body was extensively anasarcous; his breathing was very laborious, and he could not lie on either side, but was easiest sitting up. The pulse at the wrist was 140, small, and synchronous with the heart's action; but latterly both became irregular and intermittent. The measurement of the right side of the thorax exceeded that of the left by half an inch. A very loud *bruit de soufflet* was distinctly audible in every part of the thorax, and in the neck. Latterly he suffered from diarrhœa. His symptoms were, on one occasion, temporarily relieved by a profuse perspiration. Death occurred from exhaustion at the age of ten years. Attached to the right ventricle there was found a supernumerary cavity which communicated with the pulmonary artery. This cavity also communicated with the right ventricle by an opening large enough to admit the little finger, and formed under the columnæ carneæ of the ventricle. The *pulmonary artery* was perfectly destitute of valves; its lining membrane, at the usual situation of the valves, appeared a little puckered; at this spot the artery was contracted, but above, it was considerably dilated. The *septum of the ventricles* was imperfect, and the *foramen ovale* patent.

Dr. Todd, who also examined this case, attributed* the formation of the supernumerary cavity to a partial dilatation of the infundibular portion of the right ventricle in consequence of the obstruction at the pulmonary orifice. I shall presently have to quote other cases which bear a very close resemblance to this.

Mr. A. Napper has related† the case of a boy, æt. 5 years and 7 months, who began to be the subject of cyanosis and its attendant symptoms subsequently to a fall which he suffered at the age of six months. He died with symptoms of acute hydrocephalus. On examination of the brain, extensive ramollissement of the right hemisphere and serous effusion were found to exist. The lungs were extremely small and flaccid, and of a bright crimson colour throughout. The heart was of normal dimensions; both ventricles were contracted; both auricles were extremely distended with blood. In the situation of the *foramen ovale* there was an opening about half an inch in length and a line in breadth; its valvular form rendered it probable that this opening did not ordinarily admit of the passage of the blood. The aorta communicated equally with both ventricles. The ventricular orifice of the *pulmonary artery* was much contracted, scarcely admitting the large end of a common blowpipe. The sigmoid valves were exceedingly small, but otherwise perfect.

Mr. John C. Bloxham describes‡ the case of a girl who died at the age of 3 years, having been the subject of cyanosis almost from birth. The heart was found lying transversely in the chest, and of an enormous size compared with the growth of the child.

* Medical and Physical Journal, vol. xiii. p. 120.

† Duncan's Medical Commentaries, vol. xix. p. 326.

‡ Trans. of the Dublin College of Physicians, 1830.

* Cyclopædia of Anatomy and Physiology, vol. ii. p. 634.

† Med. Gaz. vol. xxvii. p. 793.

‡ Ibid. vol. xv. p 435.

The right auricle was enlarged and hypertrophied; the left appears to have been small; the *foramen ovale* would have admitted a goose-quill, but was furnished with a valve. The thickest portion of the walls of the right ventricle measured a quarter of an inch; of the left nearly half an inch. The aorta took its origin nearly equally from both ventricles, and was unusually large. The *pulmonary artery* communicated with the right ventricle; its parietes were extremely thin, and it was altogether so diminutive that its existence was discovered only by tracing its branches backwards from the lungs. "It was perceived on the posterior surface of the heart collapsed, and bifurcating immediately on its exit, so that the trunk was not, in any part of its circumference, above a line in length." It was only furnished with two valves. The aorta, which was unusually large, arose above the septum, and communicated more freely with the right ventricle than with the left. The tricuspid valve was thickened, and callous at its margin. The pericardial, pleural, and abdominal cavities contained serous effusions. The lungs were tubercular, contracted, and adherent to the ribs and spine. The liver was much enlarged, encroaching on the chest.

Dr. Hope details the case of a girl, æt. 8 years, whose lips, nose, cheeks, palpebræ, hands, and feet, were of a violet colour; the tongue and mouth still darker. There was dyspnœa on the slightest exertion, and cough only when hurried; the sternum was very prominent; great sensibility to cold, headache, vertigo, drowsiness, and sluggishness. Pulse very small and weak; and when the patient was hurried it was irregular, intermittent, and unequal. A very loud, hissing, superficial, bellows murmur was heard in the præcordial region, synchronous with the first sound of the heart; the diastolic sound was healthy, or nearly so. The child died of a chronic abscess in the brain. The heart was one-half larger than natural; the walls of the right ventricle were thickened to half an inch; the cavity was slightly dilated, and the *pulmonary orifice* contracted to the size of a goose-quill, whilst a common opening from the right ventricle, admitting the index finger, existed into the aorta and left ventricle. The left ventricle was one-third of an inch thick, and its cavity about natural. An oblique passage, admitting the handle of a scalpel, existed between the auricles. The lungs were rather flaccid, imperfectly crepitant, and universally gorged with black blood*.

M. Huet furnishes an instance† where he found the *pulmonary artery* very narrow in a patient 13 years of age. The aorta arose from both ventricles. The *foramen ovale* was open; the arterial duct closed.

The preparation numbered 1382[a*] in the collection at Guy's Hospital affords an interesting type of this kind of malformation. It is the heart apparently of a young adult in which there are evident traces of universal pericardial adhesion and of inflammation (probably of old date) affecting the substance of the organ, the presence of interstitial deposit being very apparent, especially in the walls of the right ventricle. The valves of the *pulmonary artery* are fused into a conical canal, upon the upper surface of which slight traces of the free edges of the curtains are perceptible; the artery above is about the natural size. A singularly free communication exists between the ventricles; the right ventricle is rather larger than the left, and its walls are slightly thicker. The muscular tissue of the imperfect ventricular septum is remarkably developed. The right auricle is very large; the left greatly contracted. The *foramen ovale* appears to be unclosed, but well defended by a strong valve.

It will be observed that, in a considerable proportion of the preceding instances, cyanosis is mentioned to have occurred (as it generally does in cases of congenital imperfection of the heart) at a period of some days or months subsequently to birth*. Dr. Quain has very justly argued† that, in cases of this description, the accession of cyanosis shortly after birth is immediately determined by the commencement of the process of closure in the arterial duct: it might also have been added, and in the foramen ovale. A large proportion of the instances above related, as well as many of those which will be subsequently adduced, afford strong corroboration to this view. In the five cases where permanence of the arterial canal was coincident with arctation of the pulmonary orifice, the passage of the duct had become narrowed: in the two instances where the branchial canal remained pervious, this channel was also remarkably narrow. In 13 out of the 27 cases in which patency of the foramen ovale was found to coexist with narrowing of the pulmonary orifice, there were found evidences that a process of contraction in the inter-auricular opening had proceeded to a certain, and, in most of the instances, to a very considerable extent. Besides the instances in which the arterial duct was observed to be extremely contracted, there were also twenty cases where the auricular septum remained to a greater or less degree pervious, in which no direct communication existed between the aorta and pulmonary artery. It is certainly observable, that, in the majority of cases where the foramen ovale and ductus arteriosus remain permanently open in consequence of congenital obstruction to the circulation, an effort is made by nature to close these apertures by the usual vital processes; and, although this effort proves abortive, it is sufficient to produce a certain amount of contraction in the abnormal passages; to which contraction the accession of the symptoms of obstruction to the circulation, and of morbus cæruleus during the earlier months of infancy, is, I believe, in most cases referrible. This proposition will receive still further confirmation when we arrive at the consideration of the facts upon which the now generally recognised doctrine of the causes of cyanosis rests.

In the next class of instances of narrowing of the pulmonary artery to which I shall have to refer, the ventricular septum was perforated, but no communication existed either through the foramen ovale or the arterial duct.

VII.—*The pulmonary orifice narrow—the ventricular septum imperfect—the foramen ovale and arterial duct closed.**

This species of malformation, like those which have been previously described, presents several remarkable varieties, the most interesting of which consists in an entire *absence* of the *arterial duct*, as observed in the following instances.

Professor Huss, of Stockholm, attended a boy 6 years of age, who had from birth exhibited the usual symptoms of cyanosis. When he entered the Hôpital des Séraphins the colour of his skin was dark blue, that of the mucous membrane of the mouth clear blue;—he had frequent attacks of palpita-

* Treatise on Diseases of the Heart, p. 468.
† Bulletin de Sc. Méd. t. ii. p. 72, 1808.

* This happened in twelve of the cases. In most of the other instances the precise time at which cyanosis appeared is not mentioned. In the remaining examples (four) it is reported to have existed from birth; but this statement does not appear to have been, in all those cases, the result of actual observation on the part of the reporters. I believe it to be an extremely rare occurrence for an infant to be born in a condition of true cyanosis, if, indeed, such a circumstance has ever occurred at all; but cases have been observed in which lividity, which afterwards became permanent, appeared very shortly after the commencement of respiration.
† Dublin Medical Press, vol. viii. p. 88.

* The following remarkable instances of *contraction of the pulmonary ostium, associated with perforation of the auricular and ventricular septa*, were accidentally omitted when describing the several conditions of parts with which that malformation is found to be associated.

The first and second of these correspond with the cases observed by Sandifort, Knox, Lexis, and Otto, in which narrowing of the right ventriculo-pulmonary orifice was attended with complete absence of the arterial duct; and the first case is especially interesting when placed beside the example cited by the latter author. The absence of any trace of an arterial duct at a period of a few years after birth, may not be deemed positive evidence that such communication has never existed, but the fact of its entire absence in two fœtuses dissected by different observers, is sufficient proof that the existence of the duct, or of any analogous communication, is not absolutely essential for the continuance of intra-uterine life; providing the supply of blood to the lungs be limited by a narrow condition of the pulmonary artery, and by a free communication between the right and left cavities of the heart.

Nich. Steno observed, in an otherwise monstrous fœtus of the female sex, that the *pulmonary artery* was much narrower than the aorta. The latter vessel, which was situated to the right, arose from both ventricles, the *septum* being imperfect. The auricles were in the usual fœtal condition. There was *no ductus arteriosus*. (*Acta Hafniensia*, p. 1. p. 200, *as quoted by Hein, Tab. II.*)

The following history has been given by Drs. Aran and H. Bennet (Lancet, 1844, vol. i. p. 501).—A girl, ætat. 20, who had been the subject of cyanosis of a very aggravated kind, died in the Hôtel Dieu, death being apparently mainly the result of disease of the hip, and erysipelas consequent upon caries of one of the ribs. Her stature was small. She presented no signs of puberty, and her understanding was imperfectly developed. She had no cough, except when hurried, nor dyspnœa; the feet were often œdematous. The superficial venous system was much developed; voluminous veins of a deep blue colour crossed the skin in every direction, and were united by a net-work of anastomosed veinulæ. Latterly, vertigo, and even loss of consciousness, frequently resulted from exertion. A short time previously to her death the cardiac impulsion was found to be smart and short, but considerably stronger and more extensive than natural: it was followed by a diastolic backstroke. Opposite the apex of heart, the two sounds were perceived, with an appearance of systolic murmur; but the first was a loud prolonged bellows murmur, with a very sensible purring tremor opposite the arterial orifices of the organ; and the second very short and loud. The bellows murmur was very sensible and superficial over the pulmonary artery, from its valves to the top of the sternum, along the left side of that bone. This murmur was limited superiorly by the clavicle, and on the sides by the right edge of the sternum, and by a perpendicular line proceeding from the external extremity of the left clavicle; no murmur in the vessels of the neck, nor upon the course of the aorta; pulse regular, 112, very small, and weak; resonance of the lungs normal, and the vesicular murmur pure in all the extent of the chest.

On dissection, the body was found to be generally œdematous; the heart was one-half larger than natural, right ventricle one-third more voluminous than the left. The walls of the right ventricle were half an inch in thickness; the *arterial orifice* was seated at the superior part of the ventricle, near the septum: it was contracted to the size of a goose-quill; its circumference was surrounded by cartilaginous patches, and almost obliterated by small yellowish and fibrinous vegetations, very little adherent to the edges of the orifice. The pulmonary valves were higher seated, an inch above the contraction, so that between the orifice of the ventricle and the pulmonary valves was a cavity, half an inch high, and one inch in width, covered by the lining membrane of the heart slightly thickened, and containing a polypous concretion; the pulmonary orifice was contracted to a quarter of an inch, and the arterial coats were thinner than natural. There was *no vestige of ductus arteriosus*. The *ventricular septum* was perforated at its upper part by a smooth and circular aperture half an inch in extent: aortic orifice and valves perfectly sound. Right auricle dilated, and slightly hypertrophied. The *septum of the auri-*

tion and hæmoptysis, and at times convulsions. During several months that he continued in the hospital the hæmorrhage from the lungs became more and more frequent, and in one of these attacks he died. The heart was found about three times as large as the patient's fist. The enlargement was principally in the right ventricle, behind which was the left ventricle like an appendix. The *foramen ovale* was closed. At the upper part of the *inter-ventricular septum* there was a semilunar opening of nine lines extent. In the right ventricle the orifice of the *pulmonary artery* formed a circular opening two lines in diameter with a cartilaginous margin; two lines within this opening there existed two folds resembling semilunar valves. The sinus of Valsalva formed a pouch eleven lines in diameter, filled with fibrinous clots, organised and adherent to the parietes, so that the canal was of the same diameter as its orifice; the length of the pouch was six lines. There was *no vestige of an arterial duct*.*

M. Gintrac relates† the case of an infant who had exhibited in a very marked degree the symptoms of morbus cæruleus, and which died in the second week after birth. The cerebral vessels were found extremely gorged with blood; the lungs were also very highly congested, and did not crepitate firmly on pressure. The heart was very large, and shaped somewhat like that of a turtle. The right ventricle was highly muscular; from this cavity the aorta as well as the *pulmonary artery* was found to arise. The latter was, however, nearly closed up; and *no trace of ductus arteriosus* was visible. The left ventricle was atrophied, and exhibited no appearance of arterial aperture or of mitral valve. In the *septum cordis* was a large round aperture which permitted a very free communication between the two ventricles.

In the preceding section a case related by Drs. John Crampton and Todd was cited, in which the contracted pulmonary artery arose from a supernumerary muscular cavity which communicated with the right ventricle. The following is a somewhat similar instance.

Mr. G. Holmsted attended a girl, ætat. 9 years, who, up to the age of 3½ years, was quite healthy, but then began to be affected with shortness of breath, lividity of the surface, and extreme chilliness. She continued in this state till about three months previous to her death, when, in consequence of a severe fright, she went into a kind of fit, which lasted about two hours. After this she became subject to occasional attacks of the kind, which came on with pain in the head and giddiness, a pallor of the face, with extreme difficulty of breathing, and increased palpitation. She died rather suddenly in an attack attended with pain in the head, and the evidences of thoracic obstruction. The heart was more than naturally large and muscular. The right auricle was found much distended, containing four or five ounces of blood. The *foramen ovale* was perfectly closed. The aorta was placed over the *septum of the ventricles*, and about the centre of the right ventricle there was a small opening into a corresponding cavity in the muscular structure of the heart, and from the upper part of this cavity the *pulmonary artery* went off. The left cavities were comparatively small. The *ductus arteriosus* was closed. The lungs were of a dark colour; the vessels of the head were a little fuller than usual.*

Except in the closed state of the foramen ovale, the condition of parts in the above case did not materially differ from that observed in Dr. Crampton's patient: it is, however, evident from the figure which accompanies Mr. Holmsted's narrative, that here the point of obstruction was formed at the narrow opening which led from the right ventricle to the supernumerary muscular cavity, and not at the entrance of the pulmonary

cles was perforated by a valvular slit. Great consolidation of the lungs by tubercles, of which a great many were softened; there were two little caverns at the summit of the left lung. Large tubercular masses were also found in the spleen.

The case alluded to at p. 371, where a diagram is given of the peculiar valvular apparatus with which the pulmonary artery was furnished (fig. *g*), was that of a young lady, a patient of Dr. Iliff's, who died at the age of 12 years, with the usual symptoms of malformation of the heart, which had appeared during her infancy, and gradually increased. The state of the heart, discovered after death, has been described by my friend, Mr. W. T. Iliff, jun. (Med. Gaz. vol. xxxvi. p. 1176). The organ was rather large, the right auricle strong and large, the *foramen ovale* open so as to admit a goose-quill, but valvular on the right side; the ventricles were about equal, communicating at their base by an opening in the *septum* sufficient to admit the little finger; *pulmonary artery* small and thin, with a conical valve truncated above to form an opening, which, being thickly beset with vegetations, would scarcely admit a crow-quill. The cord of the *ductus arteriosus* was complete. There were also vegetations on the aortic and mitral valves. The heart is preserved in the museum at Guy's Hospital (Preparation 1379[3]). I observe that portions of the inner surfaces of the tricuspid valve are also coated with adherent clots. The left auricle and ventricle are of small capacity. An inspection of the preparation, or of the diagram which I have given, will at once shew that the peculiar form of the funnel-shaped valve, and the arrangement around its opening of a little tuft of elastic vegetations which close together over the aperture, must have completely prevented even the slightest reflux of blood into the ventricle during the diastole of that cavity.

* Gazette Méd. de Paris, Feb. 11th, 1843.

† Archives Générales, as quoted in Med. Chir. Review, Jan. 1839.

* London Medical Repository, vol. xvii. p. 455.

artery, as was the case in Dr. Crampton's instance. The position, in Mr. Holmsted's case, of the supernumerary cavity at a considerable distance below the spot at which the pulmonary artery usually arises, must also be regarded as a proof that this additional ventricle was not formed by a mere dilatation of the infundibular portion of the right ventricle in consequence of an obstruction at the pulmonary orifice.

The following cases present several remarkable varieties of this kind of malformation.

Mr. J. Marshall gives the following account* of a man who had been cyanosed from birth, and had always suffered from symptoms of suffocation upon the slightest exertion:—The surface was cold; the pulse small and frequent, but regular. A few months before his death, he became dropsical, his breathing grew extremely oppressed, and he was incapable of lying down. Four gallons and a half of effusion were removed from the abdomen, with considerable temporary relief; but re-accumulation of the fluid occurred, and the patient died, at the age of 23 years. The abdomen was found to contain five gallons of fluid. The right auricle of the heart was large; the *foramen ovale* perfectly closed. In the *ventricular septum* there existed an aperture about an inch in diameter. The semilunar valves of the *pulmonary artery* were shrivelled, and appeared useless, but were not ossified. About an inch above the valves the artery was contracted so as to form a stricture, hardened by ossific deposit, and about the diameter of a goose-quill. The coats of the artery above the stricture were very thin, and formed a sort of sac. The ventricles were of equal strength. The pulmonary veins and left auricle were very much diminished in size; the left ventricle also was small. The aorta arose naturally, and its valves were healthy. The lungs were small and black from the blood they contained, but were not diseased.†

The following case is interesting, from the care with which the physical signs were observed:—

Dr. C. F. Gravina relates‡ the case of a boy, ætat. 9 years, who had been cyanosed from his birth, and suffered from a violent and continual palpitation of heart, and short rapid respiration. On the least exertion these symptoms were severely aggravated. The respiration was heard distinctly all over the chest; but in the precordial region two abnormal cardiac sounds were recognised: one of them resembled the purring of a cat, and was heard during the systole of the heart; the other, which appeared to accompany the contraction of the auricles, was so clear and sonorous that it was heard over the whole of the right side of the chest; over the left side, however, it was scarcely heard. While the boy was in a state of repose his pulse beat from 78 to 84 in the minute; but, on the least excitement, rose to 100 or 105. He died suddenly, in consequence of an attack of convulsions and dyspnœa. The lungs were found of a violet colour, regular in their form, and crepitating on pressure. The right ventricle was nearly twice as large as the left; but its walls were of the ordinary thickness. The left ventricle was smaller than common. The *ventricular septum* was deficient below the point where the aorta arose, leaving a circular communication between the two ventricles. This opening was largest on that side which corresponded with the right ventricle, but was somewhat contracted on that of the left. The aortic orifice was much dilated, as were also the semilunar valves, which appeared proportioned to the increased dimensions of the vessel. The *pulmonary artery* was very much contracted, but its walls were unaltered. The *foramen ovale* was closed.

Dr. Pulteney attended a young gentleman who could not walk across the room without turning almost black in the face and hands, and becoming faint and almost breathless. He sunk under an attack of dysentery, at the age of 13 years and 9 months. The lungs were found to be remarkably small and collapsed, and some parts of them were so flaccid as to suggest the idea of their having been incapable of performing their functions. The heart was of the natural size. The orifice of the *pulmonary artery* was much smaller and more firm than is usual, but there was no fault in its valves. The ventricles communicated by a deficiency in the upper part of the *septum* capable of admitting the end of a finger. The ventricles and right auricle appear to have been of ordinary size; but the left auricle was very small.*

In this case the imperfect state of the ventricular septum appears to have been due to mere rigidity of the structures at the base of the pulmonary artery, without any imperfection in the vessel itself. It might be judged from this, and one or two other examples in which the pulmonary ostium was not found extremely contracted, that the closure of the foramen ovale has been due to the comparatively slight amount of obstruction existing in the vessel. In se-

* Medical Gazette, vol. vi. p. 886.

† I have alluded to this case at the commencement of this paper, and given a copy of Mr. Marshall's sketch of malformed artery (fig. *e*, p. 370.)

‡ Schmidt's Jahrbucher, July 1839, and Edinburgh Medical and Surgical Journal, vol. xxxv.

* Medical Transactions of the College of Physicians, vol. iii. p. 334.

veral cases, however, extreme contraction of the artery has been found in conjunction with an impervious state of the auricular septum. The following instance may be taken as a fair example of this condition. It is certainly possible that the narrowness of the orifice may have been gradually increasing up to the time of the individual's death; but the history of the symptoms renders it probable that the vessel had been greatly obstructed from the earliest period:—

The second case recorded by Dr. Pulteney* is that of a lad whose complexion had always been dark, or inclining to black; he was liable to fits, upon the accession of which he suffered from oppression at his heart, became weak or faint, grew dusky in his colour, and at last almost black, fell down, and seemed insensible. He commonly soon came out of the fit with sobbing and yawning, and a sense of fatigue. He could escape the fit by instantly lying down on the carpet on his left side, and remaining immoveably fixed in that posture for about ten minutes. He died at the age of 13 years. The orifice of the *pulmonary artery* was so small that it could barely give passage to a small probe. The *ventricular septum* was perforated so as to allow the thumb to pass. No other cardiac lesion is mentioned either in this or in the preceding case; and, as the instances were recorded at a time when patency of the foramen ovale was regarded as one of the most frequent causes of the blue disease, it is quite improbable that Dr. Pulteney could have overlooked such a deficiency had it existed.

Besides the specimen presented by Dr. Iliff, and already referred to, there are two very interesting examples of this kind of malformation in the collection at Guy's Hospital.

The preparation marked 1379[50] is taken from a lad, æt. 16, who was of rather robust appearance, and who had since his birth been much troubled with palpitation of the heart and great dyspnœa upon the slightest exertion. He had also suffered from frequent attacks of pain in the left side. He entered the hospital on account of an indolent ulcer on the leg, depending on feeble circulation. Both lower extremities were rather œdematous. There was usually lividity of the lips, but it was only on increased exertion that his face became decidedly blue. The pulse was regular, but slow and feeble. He was subsequently attacked with fever, and died after profuse hæmoptysis, from which he had never before suffered. A small quantity of bloody serum was found in the cavity of the right pleura; the substance of the lung was rather œdematous, and contained miliary tubercles. The left lung was enormously engorged with blood, friable, and containing, at its apex, a tubercular excavation, apparently of old standing. The bronchial mucous membrane was highly vascular; the pericardium contained about ℥iij. of sanguinolent serum.* The *pulmonary artery* at its commencement was about half its usual size.† The right auricle was enormously dilated, the tricuspid orifice was wide, and its valve doubtless admitted of free regurgitation. The right ventricle was exceedingly large, and bore traces of hypertrophy. The aorta arose from the right ventricle, posteriorly to the pulmonary artery. The *ventricular septum* was deficient above, leaving a communication an inch in diameter between the ventricles. The left cavities were small, and their parietes were much attenuated. The aorta was very large, and its valves were somewhat thickened. The *foramen ovale* was closed. There was a considerable deposit of fat upon the surface of the heart.

The specimen numbered 1382[92] is a heart, which appears to have belonged to a person 15 or 16 years of age; the pulmonary orifice would barely admit a common pencil; the upper portion of the artery is of fair size. The aorta arises above an aperture in the *ventricular septum*, and communicates rather more freely with the right than with the left ventricle. The walls of the left ventricle are thinner than those of the right; the right ventricle is much stronger and more capacious than the left. The right auricle is dilated, the left much contracted. The *foramen ovale* is closed. The preparation does not present any trace of an *arterial duct*, and, as the aorta becomes suddenly smaller at the spot where the arterial ligament is usually attached, there has not improbably existed some irregular communication between one of the vessels of its arch and the pulmonary artery. The aortic, mitral, and tricuspid valves, present numerous vegetations. The upper intercostal branches of the aorta are rather larger than natural.

Several other examples of this species of malformation have been recorded. Of these the following are the principal.

Mr. Abernethy ‡ mentions the case of a child, æt. 2 years, who died with symptoms which indicated an impediment to the transmission of the blood through the lungs. The aorta arose from the right ventricle, which was of unusual size and thickness. The orifice of the *pulmonary artery* was small, and the remainder of the vessel one-

* Op. citat. vol. vi. p. 299.

* Report by Mr. Dade, MED. GAZETTE, vol. xi. p. 159.

† In the preparation it scarcely appears to be capable of admitting a slender little finger.

‡ Surgical and Physiological Essays; Part Second.

third less than its usual size. The *ventricular septum* was imperfect. The left cavities were considerably smaller than the right.

In the heart of a cyanosed boy, æt. 6 years, M. Olivry* found contraction of the *pulmonary artery* associated with an imperfect state of the *ventricular septum*. The *foramen ovale* and *arterial duct* were closed. A similar condition of parts was observed by Meyer†, in a girl who died in her 7th year.

Dr. Farre describes‡ a heart (preserved in Sir A. Cooper's collection), which was taken from a boy in whom, at the time of birth, nothing unusual was observed; but after a few months cyanosis began to appear, which was heightened by passions of the mind, and by cold. Before he was three years old he had lost the use of his lower extremities; but this paralysis subsided in a few weeks under medical treatment. At the earliest period at which he was able to express his feelings, he complained of frequent nausea and severe headache. These symptoms were brought on or increased by the causes above mentioned, and were established at the age of five years. At the age of nine years and five months he was found to have lost the use of his left thumb; and, in two days afterwards, there was paralysis of the arm and leg on the same side, with partial convulsions of short duration. He expired in a few days, with the symptoms of an acute head affection. Upon dissection, an abscess was found in the right hemisphere of the brain, containing about an ounce and a half of a thick, dark-coloured, and extremely unhealthy pus. The ventricles of the heart communicated at the root of the aorta. The semilunar valves of the *pulmonary artery* were contracted into a small circle. The *arterial duct* was closed. The state of the auricular septum is not mentioned, but patency of the *foramen ovale* could scarcely have been overlooked.

Dr. Watson§ makes slight allusion to the case of a lad, æt. 17, in whom the pulmonary orifice would not admit a goose-quill, the *ventricular septum* being imperfect.

A similar instance of malformation in a girl 21 years of age, is recorded by Professor Dunglison, in the Philadelphia Medical Examiner for May 1845. Of this patient's early history nothing was known; but during the last year of her life she was unable to perform any work, or take exercise of any kind, as upon the least exertion or excitement she was attacked with palpitation and dyspnœa, accompanied with some blueness of the skin,—all of which gradually disappeared on her remaining at perfect rest. She was also subject to attacks of the same kind after dinner: these paroxysms during the latter part of her life came on every day, continuing for two or three hours, even though she remained perfectly quiet, being apparently excited by the mere stimulus of food. She had also a constant and strong rasping bruit, accompanying the first sound of the heart, and masking it partially. She suffered much from headache, which, a few days before her death, became greatly increased, accompanied with high febrile excitement, followed by delirium, coma, and death. At the time of her death the patient was menstruating profusely. On examination, the *septum ventriculorum* was found deficient at the orifice of the aorta, giving that vessel an origin from both ventricles; the *pulmonary artery* was contracted, so as barely to admit the little finger; both lungs were studded throughout with crude and miliary tubercles, none of which were softened; the left lateral ventricle of the brain was filled with pus, the lining membrane being inflamed and thickened, and in the posterior lobe of the same hemisphere was found a cyst the size of a robin's egg, also filled with pus, but having no connection with the ventricle. The substance of the brain presented numerous red points, but was not altered in consistence.

A large proportion of the above cases afford ample additional confirmation to the fact which I have already stated, that contraction of the pulmonary artery, which has, at a very early period of intra-uterine life, produced sufficient obstruction to prevent the complete development of the ventricular septum, may still fail to determine the persistence of the ductus arteriosus and foramen of Botal. But, although the inter-ventricular communication was large and free in several of the examples just cited, I am not acquainted with any instance of this class where the septum was in so imperfect a state as to leave the ventricular portion of the heart in the condition of a single cavity, while in not less than six of the instances where the auricular septum remained incomplete, the partition of the ventricles was found in a merely rudimentary state.

In the whole of the remaining instances of congenital arctation of the pulmonary artery with which I am acquainted, the ventricular septum was completely formed.

There appears to be no reason to believe that, in any of the class of cases at present under consideration, closure of the foramen ovale has occurred previously to birth. In the few instances where that remarkable aberration from the natural condition has been observed, there has existed a state of parts altogether different from that noticed in the above examples.

This question may possibly admit of dispute, but it seems to be in the last degree

* Quoted by M. Gintrac, op. cit. p. 177.
† Meckel, Archiv. B. i. H. 2, Tab. n, 42, p. 284.
‡ Op. cit. p. 24.
§ Medical Lectures, vol. ii. p. 247.

improbable that occlusion of the communication between the auricles should occur at a sufficiently early period in fœtal existence to become the means of preventing the full development of the ventricular septum.

VIII. *Contraction of the right arterial ostium, with patency of the foramen ovale, the ventricular septum complete, and the arterial duct obliterated.*

It is evident that, in the cases where this species of malformation occurs, the inflammatory or other morbid condition which has produced the arctation of the pulmonary orifice has become established at a period when the ventricular septum has obtained its full development. The obstruction to the pulmonary circulation determines the permanency of the foramen ovale, but the arterial duct becomes naturally closed. It is difficult to explain the difference in this latter respect between the cases now under consideration, and those of Class III.*—in which arctation of the pulmonary orifice is attended with a perfect condition of the ventricular septum, but with a pervious state doth of the foramen of Botal, and the ductus arteriosus,—otherwise than by the conjecture that, in cases of the eighth class, the impediment in the artery has originally been slighter in than those of the third; or that, in the latter instances, the arterial lesion has been established for some time previously to birth; while, in the former, disease has been set up in the vessels almost immediately after the commencement of respiration, when the arterial duct has begun to close, the foramen ovale still remaining open.†

Whatever may be the true explanation of this point, it is however certain that many causes of obstruction to the circulation which are capable of determining the patency of the septal opening, prove quite insufficient to prevent the closure of the arterial canal: hence the frequency of imperfection of the auricular septum, and the rarity of permanence of the duct. In some of the cases of this class which I shall have to quote, there appears to be reason to believe that the foramen ovale had become closed, but that the progressive increase of obstruction at the pulmonary orifice had given rise to so great distension of the right auricle as to occasion perforation of the membrane of the ovalian fossa.

* Page 374.

† Bernt of Vienna (quoted by Mr. A. Taylor) states, that if a child respire only for a few seconds, the duct becomes contracted at one of its extremities, commonly at the aortal end. I have not satisfied myself of the correctness of this observation, but there appear to be strong grounds for believing that the process of closure usually commences later in the foramen than in the arterial duct.

The two following cases are rendered interesting by the close analogy which they bear to other instances previously detailed. The first is also remarkable from the fact that the lesion of the arterial orifice was obviously attributable to the presence of a morbid deposit, and did not appear to be attended with faulty development of the valvular apparatus.

Dr. Edward Hallowell* examined the body of a female child, æt. 6 months, who had from birth been more or less affected with difficulty of respiration, and latterly much subject to cough, during the paroxysms of which the face became suffused, and the nails and ends of the fingers cyanosed. On inspection, the body was found to be well formed, with considerable embonpoint, and no œdema; the fingers were curved inwards, and of a purplish colour. The lungs were quite healthy, of a light pink or rosy hue, not engorged in the slightest degree, and perfectly healthy throughout. The mucous membrane of the trachea and bronchial tubes was slightly injected; the pleuræ free from effusion; pericardium healthy. The heart measured from apex to root about 2 inches; its transverse measurement was about 2½ inches. The right ventricle was enormously hypertrophied, there being scarcely any cavity; its walls were about 5 lines in thickness, the septum 6½ or 7 lines: those of the left ventricle were about 2 lines: the cavity of this ventricle was of natural size. The diameter of the aorta nearly double that of the *pulmonary artery*, the orifice of which was almost entirely obliterated by a warty excrescence, arising from the middle semilunar valve upon its under surface, and extending to the others, to which it adhered. The external surface of this vegetation was lobulated, having a warty appearance, resembling a small raspberry, though less regular in its conformation, presenting, when cut into, an almost cartilaginous hardness, and adhering very strongly to the surface of the valve. The obstruction to the artery was so great that a silver probe passed with some difficulty: the coats of the artery appeared to be thinner than natural. The right auricle was greatly dilated, its capacity being double that of the left; the greatest thickness of its parietes, one line, that of the left, half a line. The *foramen ovale* was patulous, its opening being about 3 lines in diameter. The tricuspid, mitral, and aortic valves were healthy. The liver was greatly enlarged and congested, occupying one-third of the abdominal cavity. The other organs were not examined.

* American Journal of Medical Sciences, vol. xxii. p. 365.

M. Lallemand relates* the case of a woman who, from her earliest infancy, was liable to become in some measure cyanosed upon unusual exertion. Her respiration was habitually oppressed. At the age of 47 years she ceased to menstruate, and began to suffer from palpitations attended with acute pain in the precordial region, and her lips and general surface became of a livid blue colour. She also became subject to attacks of profuse epistaxis. At the age of about 57 years she was attacked with hemiplegia of the left side of the body, the paralysed limbs being occasionally convulsed: bleeding gave some relief, but she died a short time after the paralytic attack. The heart was found enormously enlarged; the right auricle was greatly dilated, and contained seven ounces of blood. The *auricles communicated* by an opening about 4 lines in diameter. The right auriculo-ventricular orifice was contracted. The right ventricle was not more than large enough to contain a pigeon's egg, its parietes being from 11 to 16 lines in thickness. The *ventriculo-pulmonary orifice* presented a septum which was convex towards the artery and pierced in its centre with a perfectly circular orifice 2½ lines in diameter. Above this the artery presented nothing material. The left auricle was much larger than natural; the left ventricle was also of very large size, and its walls hypertrophic. The *arterial canal* was entirely obliterated. In the anterior part of the right hemisphere of the basin was found an encysted purulent collection.

It will be remarked, in the above instances, how completely the condition of the right cavities of the heart corresponded with that which was noticed in a set of cases observed by Dr. W. Hunter and others (p. 280), where obliteration of the pulmonary artery was attended with an open state of the foramen ovale and arterial duct, the ventricular septum being complete; as also with that noticed by M. Bapt. Schuler, in a case (Class III. p. 374), where arctation of the pulmonary orifice, (also unaccompanied by perforation of the septum,) had in like manner determined the permanence of the canal and duct. The instances at present under consideration, in which the foramen was open and the duct closed, complete the series. In both of these cases the main current of blood, failing to gain a free passage through the right ventricle and pulmonary artery, was diverted into the left cavities through the open foramen ovale; hence, the right ventricle, being deprived of its due quantity of blood, became greatly diminished in capacity; the occurrence of this contraction being doubtless considerably aided, in M. Lallemand's case, by the narrowed condition of the right auriculo-ventricular orifice. It is probable that, in each of these individuals, the aggravation of the symptoms, and ultimately the fatal event, were owing to gradually increasing contraction in the right ventricle and pulmonary orifice, and, possibly, also to diminution in the size of the foramen ovale.

It is to be regretted that in the following case no distinct idea is given of the size of the right ventricle. As, however, it is merely stated that the heart was "*somewhat* enlarged," and that the walls of the right ventricle were greatly hypertrophied, it is probable that its cavity was, at least, not very greatly dilated.

Dr. Robert Spitta lately communicated to the Medico-Chirurgical Society* the case of a lady, ætat. 40, who was of diminutive stature and pigeon-breasted, but without any positive deformity. She had cyanosis from birth, indicated by blueness of the cheeks, lips, and tongue. The heart's action was said to have been regular when at rest, but sudden exertion produced palpitation and dyspnœa. She suffered very much from cold. In March 1845, she was suddenly seized (while at rest) with dyspnœa, and partial syncope, accompanied by convulsions, and afterwards with intense pain referred to the epigastrium, loins, and hypochondria. This condition lasted twenty-four hours, when she died. The patient had previously suffered from serious attacks of a similar nature occurring at long intervals. In November 1843, she had also an anasarca of the abdomen and lower extremities, from which she completely recovered, and never afterwards became dropsical. She had never suffered from rheumatism. The heart was found to be heavy and firm, and somewhat enlarged. The hypertrophy was chiefly on the right side, the right ventricle being as thick as the left, and the right auricle three times as thick as the left auricle. The *foramen ovale* was patulous, the opening about 4 lines in diameter, and valvular. The auriculo-ventricular valves were thickened, but movable: the aortic valves healthy. Above the semilunar valves of the *pulmonary artery* an adventitious membrane was stretched completely across: it was a line in thickness, and perforated in its centre by a mere slit with margins of a red colour, and fringed with fibrine of the blood, (vegetations?) The three semilunar valves were thrown up, as they are naturally during the systole of the heart, and fixed in that position by the adhesion of (what would have been) their free borders to the adventitious membrane.

* Recherches Anatom.-Pathol sur l'Encephale, t. ii. p. 7.

* Medical Gazette, vol. xxxvii. p. 365.

The following case is singular, from the existence of a remarkable malformation of the right ventricle which has been noticed in connection with several kinds of congenital lesion of the organ, but which has not, so far as I am aware, been observed in any other instance of narrowing of the pulmonary orifice.

M. Obet narrates* the case of a female child who began to suffer from the symptoms of blue disease a few days after birth. She was also subject to occasional prolonged attacks of bilious vomiting. She died at the age of 5 years from aggravation of the cardiac disturbance. The right cavities of the heart were found distended with blood and fibrinous concretions. There was very considerable thinning of the walls of the right ventricle, especially at its apex, where they were not more than a quarter of a line in thickness. An unnatural enlargement of the tricuspid valves (musculi pectinati?) caused the right ventricle to be partitioned into several cavities. The left cavities of the heart were narrowed, the *foramen ovale* was open, and about 4 lines in diameter; both arterial apertures were narrow, and the width of the *pulmonary* orifice was less than that of the aorta.

In this case, as in most of those which have still to be cited, the obstruction to the pulmonary circulation had produced its ill effects principally by occasioning impediment to the emptying of the right ventricle, there being, in all probability, in the first place, neither sufficient contraction of the pulmonary orifice, nor a sufficiently free communication between the auricles to occasion diversion of the main current of blood through the auricular septum into the left cavities.

One of the earliest recorded instances of contraction of the pulmonary orifice is that related by Morgagni. A young woman, who had always been sickly from the time of her birth, whose breathing was oppressed, and whose skin was of a livid colour, died at the age of 17 years. The heart was found to be small, and rounded at its basis; the right auricle was more fleshy than the left, and twice as large; the *foramen ovale* would admit the little finger. The tricuspid valve was malformed, two of its curtains being unusually small. The valves of the *pulmonary artery* were in a natural state at their base, but their upper edges were cartilaginous, and even partially ossified, and so united together as to leave a passage for the blood not larger than a lentile. This opening was furnished with membranous and fleshy excrescences, so placed as to answer the purpose of valves, by allowing the exit of blood, from the ventricle and preventing its return.

In the following instances it is by no means clear that obliteration of the foramen ovale had not been effected subsequently to birth; on the contrary, it appears to be highly probable that in both cases the membrane of the fenestra ovalis suffered perforation in consequence of the gradually increasing obstruction at the entrance of the right ventriculo-arterial orifice.

Dr. Craigie has given the following narrative* :—

A countryman, æt. 19, had, from infancy, been always subject to shortness of breath, which was very urgent in cold damp weather; the lips, hands, and nails, had always been of a purple or livid colour. From his earliest recollection he had always been subject to palpitation, aggravated to an extreme degree by exertion. During the last two years there had been occasional swelling of the feet and ankles. Notwithstanding these complaints, he continued to work at agricultural labour of a light nature until about five weeks before his death, when he was suddenly attacked with rather copious hæmorrhage from the lungs while sitting quietly by the fire. In a week after this he came under Dr. Craigie's care. The hæmorrhage had recurred several times during the week. The face, lips, tongue, interior of the mouth, and nails, presented deep purple and leaden tints. The anterior part of the chest was very prominent, the sternum arched, and the shoulders much elevated. Respiration was 28 in a minute, performed by the accessory muscles with much labour. There were physical signs of extensive bronchitis, with scanty secretion. Extensive dulness over the cardiac region. The cardiac beats were from 92 to 96 in the minute, performed with rather strong impulse, and a rough rasping or sawing murmur, considerably prolonged, accompanied the first sound; this was heard most distinctly on the right side of the sternum, immediately between the third and fourth ribs. The more urgent symptoms were greatly relieved by perfect rest and the limited use of anodynes and anti-spasmodics; but, five days before his death, hæmoptysis again occurred, but soon ceased, the heart's action becoming strong and labouring, with a very small pulse at the wrist. The livicity increased; the patient suffered much from coldness of the surface and hurry of respiration, which progressively increased until his death. The pericardium was found to contain ℥ij. of serum. The heart, which weighed eighteen ounces, was much enlarged,

* Rev. Méd. t. vi. p. 175.

* In a valuable and elaborate paper published in the Edinburgh Medical and Surgical Journal, vol. lx. p. 268.

especially its right auricle. The membrane of the *fenestra ovalis* was thin and reticulated, and presented many minute holes, and, at its lower limbus, an elliptical hole, one-third of an inch in diameter, communicating directly with the left auricle. The right ventricle was large in capacity, and its walls were much hypertrophied. The origin of the *pulmonary artery* was provided with a firm opaque membrane, concave towards the ventricle, convex towards the artery, and so elevated as to form a truncated cone, with a small aperture in its centre and apex, not more than large enough to admit a common probe. This aperture was surrounded by a fringe of warty prominences. The upper part of this cone presented three membranous slips, indicating the position of the attachment of each valve. The left auricle and ventricle were of the natural size, and otherwise healthy. The state of the left lung only is mentioned; portions of its tissue were solid, firm, and unusually dark-coloured, and it contained several dilatations of the bronchial tubes, one of which was as large as a filbert. One of the kidneys was atrophied, and its secreting portion almost lost; the other was large, but in some spots presented traces of the granular change.

M. Cherrier* attended a man ætat. 34 years, who had been free from any signs of heart affection until the age of 14 years, when he fell into a deep ditch full of water in the winter time. For some days after the accident he suffered from a low fever, and, during a protracted convalescence, blueness of the lips and extremities of the fingers manifested itself; and, from that time up to his death, the cyanosis continued to become aggravated, growing more and more intense during the last two years of his life. The discolouration of the entire body was extreme. During the latter period he suffered from four severe attacks of pneumonia. For the cure of these, bleeding was employed; but this produced such great weakness that it had to be discontinued. At the age of 28 years he married, and had two children, which died of scrofulous affections at the age of 3 years. He suffered from extreme debility, palpitations, coldness of the entire surface, and difficulty of breathing. He became affected with a severe pleurisy of the left side, which was attended with great aggravation of the dyspnœa. This was followed by two attacks of apoplexy; the last of which was followed by hemiplegia, and proved fatal. The left pleura was found to be filled with sanguinolent serum, compressing the lung, and evidently the product of acute inflammation. The heart was enormously large, its vessels and cavities, and more especially the ventricles, being engorged with blood. The right cavities were singularly large; the auricles exteriorly did not present any very remarkable appearance, but they communicated by an orifice more than an inch in diameter. The walls of the ventricles were diminished in thickness; the interventricular septum was complete; the free edges of the *pulmonary sigmoids* were ossified, rendering the orifice so narrow as scarcely to admit a crow-quil. There was great congestion of the nervous centres and abdominal viscera.

* Quoted by M. Gintrac, op. cit. p. 189.

There can be little doubt that, in the whole of the above-cited instances of this class, the narrowing of the pulmonary orifice dated from a period antecedent to, or very shortly after, birth,—although, as generally occurs in every form of congenital contraction, the narrowing appears to have continued to increase as life advanced:—still, in all instances of this class, it will be necessary to ascertain whether the obstructive disease of the pulmonary valves has not been set up at a considerably later period of life, either occurring in coincidence with accidental non-occlusion of the foramen ovale, or becoming in itself the cause of perforation of the interauricular septum, as the occurrence of such a train of lesions is not altogether improbable.

The narrator of the following case believed that the perforation of the auricular septum was due in his patient to accidental causes; but this appears to be questionable, as the precise condition of the structures surrounding the opening are not stated with sufficient accuracy to enable us to judge whether it was due to imperfect development or to ulceration:—

Cajetanus Tacconus relates* the history of a beggar-girl, about 15 years old, who walked with great difficulty, and was remarkably cyanosed. She suffered from constant palpitation of the heart, and extreme weakness in all her limbs; was able to swallow only very thin or liquid aliment, and even that not without much difficulty. Her complaints were much increased during winter; she had as yet no appearance of the catamenia; her pulse was quiet and weak. She attributed this disorder to a sudden and excessive fright which she experienced, the consequence of a fall from a great height, at the age of 5 years. She remained under the author's observation for three years; and during this period he could never discover that her nostrils, thorax, or abdomen had the least degree of motion in respiration. At the end of this time, during a very severe season, she began to complain of pain in her left side, and to discharge by

* De Bononiensi Scientiarum et Artium Instituto atque Acad. Commentar. t. vi.; Bonon 1783, and Med. Jour., vol. vi. p. 407.

the mouth a black grumous blood. She died on the four-and-twentieth day after the accession of these symptoms. It was found, on inspection, that the contents of the abdomen encroached so much upon the thorax as to render the capacity of the latter cavity extremely small. The lungs were hard, dry, and contracted; the left lobe was of a dark livid colour, and adhered to the pleura at the part where the patient had complained of pain. About three ounces of black grumous blood were found on the surface of the diaphragm and in the trachea. The shape of the heart was cubical rather than conical; the left ventricle, in its shape and structure, appeared as the right ventricle usually does in a natural state, and *vice versâ*. The valves of the *pulmonary artery* were adherent to each other in such a manner that water could be pressed into the artery only by a small opening; and this the author supposed to have been made by a probe with which he examined the state of the valves. The pulmonary vein (possibly the veins united in a single trunk) was empty and contracted; the *canalis arteriosus* was closed, but the *foramen ovale* was found open, and larger than it usually is in the fœtus. The remainder of the dissection afforded nothing preternatural.

IX.—*Contraction of the pulmonary orifice, the ventricular and auricular septa being complete, and the arterial duct obliterated.*

In certain cases of this description it may also be extremely doubtful whether the abnormal condition of the artery is congenital or of later date, the duration and character of the symptoms being the only means by which a decision can be formed; but, in the examples which I have selected as illustrative of this species of malformation, the appearances discovered after death appear to have rendered the congenital origin of the disease a matter of almost absolute certainty. In the following case by Dr. Blackmore, where the appearances discovered were of the most rare and singular character, it is probable that the foramen ovale had become closed previously to birth, and that a ductus arteriosus had never been developed, as the arrangement of the parts was such that the presence of those communications during the fœtal existence of the child was rendered wholly unnecessary:—Here the narrowness of the pulmonary artery does not appear to have been the primary lesion.

A female infant began to suffer from the ordinary symptoms of blue disease at the age of 2 months. All her complaints became aggravated after her inoculation at the age of 16 months. Assimilation was imperfectly performed. She never walked alone, nor talked intelligibly. The body was extremely puny, and the chest chicken-breasted; the pulse at the wrist was small, very feeble, but regular. Death occurred at the age of 3½ years, after a flow of black blood from the mouth and nose. The heart was found to be so large as to hide and compress the lungs: the right auricle and superior cava were greatly distended with blood; the right ventricle was greatly hypertrophied; the left ventricle was extremely small in comparison with the right. Where the left auricle should have been, only small muscular vestiges were found, without cavity. On attempting to lay open what at first sight appeared to be the tip of the left auricle, the knife entered a large single cavity, between which and the left ventricle no communication existed, except through a small fissure, the natural ventricular opening being occluded by a firm adherent fleshy membrane. From the posterior superior part of the right ventricle arose the aorta, much dilated; immediately below the aorta a small *pulmonary artery* had its origin. The left ventricle held a small coagulum of black blood, which must have entered it by the fissure above described, and which was its sole outlet. Its substance was healthy; no *ductus arteriosus* remained. The right auricle proper received the trunk of the superior cava, around the orifice of which was attached a fine delicate membrane. Two small pulmonary veins entered the inferior part of the left auricle, which was nearly obliterated, and one large vein entered its superior part. The auricular septum presented the *fossa ovalis* in its natural state, without communication with the right auricle. The left auricle had, however, a direct communication with the left ventricle by means of a small fissure through which the blood returning to the lungs must have passed, and thence into the right side of the heart. The lungs were small, particularly the left, and dense, like flesh; but there was no tubercular deposit or confirmed induration.*

In the remaining instances of this class which have been observed, the lesions were of a far more simple character than in the above example:—

Dr. Elliotson† refers to the case of a very young woman who died in St. Bartholomew's Hospital. A bellows sound was heard in the precordial region, and the patient suffered from venous congestion and dropsy. The walls of the right ventricle appeared to have grown up around the mouth of the pulmonary artery, so that the opening from

* Edinburgh Med. and Sur. Jour., vol. xxxiii. p. 268.

† The recent Improvements in the Art of Distinguishing the various Diseases of the Heart, p. 21, pl. 1, fig. ii.

the ventricle was no larger than the circumference of a goose-quill; and a short canal of this size had to be traversed before it reached the mouth of the *pulmonary artery*, which was of natural size. Dr. Elliotson believed this malformation to be congenital, as two little supplementary right ventricles existed here: one leading from the pulmonary artery, and the second from the first. The heart is in the Museum of the College of Physicians.

Dr. Craigie details* the case of a railroad labourer, ætat. 44, who became a patient of Dr. Graham, with symptoms which were at first mistaken for those of fever. His history was indistinct. About two weeks previously, after a debauch of ten days' duration, he was seized with severe cough, hoarseness, headache, and thirst, and gradually lost flesh and strength. When admitted to the Infirmary his intellect seemed rather confused; he complained of frontal headache, and denied that he had any other local uneasiness; was very hoarse, and spoke very indistinctly, apparently from a very hurried state of his respiration. He had frequent cough, without expectoration; pulse 120; face rather livid. After his admission, the respiration continued very frequent—about 60 in the minute. He was delirious and restless at night, and died on the third day. Both lungs were found to contain tubercles, and were much consolidated: the apex of the left was occupied by a large tubercular cavity. The heart weighed thirteen ounces. The right auricle was much dilated, and decidedly thicker and firmer than usual. The right ventricle was much hypertrophied, its walls being nearly as thick as those of the left usually are, and it cavity was at the same time smaller [than natural?]. The *pulmonary semilunar valves* were so united as to form a ring, which could only admit the point of the little finger. The artery above was much dilated, and its walls attenuated. The left cavities appeared healthy. The aortic semilunar valves were somewhat thickened, but were adequate. The aorta was also dilated at its arch, but not to the same extent as the pulmonary artery.

Death seems to have been produced in this individual by the sudden aggravation of his pulmonary disease, rather than in consequence of the cardiac obstruction, which does not appear to have been excessive. I have met with several instances of heart disease in which the cavity immediately posterior to the seat of obstruction appeared, when first examined, to be in a state of concentric hypertrophy, but this condition was always, in reality, merely apparent, the cavity becoming expanded so soon as the cadaveric rigidity of the heart had ceased. This is liable to occur in cases where death is rather suddenly induced by accidental causes which do not immediately influence the heart.

The following case affords an excellent illustration of the effects of extreme obstruction to the circulation through the right cavities of the heart, where the embarrassment of the over-distended auricle and ventricle is not relieved either by an open state of the ventricular septum or by patency of the foramen ovale.

M. Fallot has recorded the case of a woman, ætat. 63, who, after the cessation of the menstrual discharge at the age of 47, became subject to dyspnœa. The heart beat tumultuously, but the pulse was regular, although hard and vibrating. Relief appeared to be afforded by bleeding, which was frequently practised. Three months previously to her death she was seized with violent pains in the region of the heart, and with frequent attacks of vertigo. Her respiration became more embarrassed, and she was unable to stand. Two or three weeks before her death she became delirious. She expired in great agony. The heart was found to weigh 14½ ounces; by its excessive development it had pushed the mediastinum into the right cavity of the thorax. The right auricle and ventricle, and the two venæ cavæ, were much enlarged; and, in consequence of the dilatation of the cavities on this side, the tricuspid valve did not nearly close the auriculo-ventricular opening. The walls of the right ventricle were softened, and much thinned, but were covered by a thick lamina of coagulum, composed of different coloured strata, and partially enveloping the columnæ carneæ. In place of the semilunar valves of the *pulmonary artery*, a thick membrane, attached to the circumference of the orifice of the artery, was found; its concavity, which was directed towards the ventricle, was pierced at its centre with an opening about the size of a goose-quill. The free and attached borders were thicker than the rest of this membrane. The left cavities of the heart were of the natural size, and were perfectly healthy. The tissues of the lungs were much compressed by the enlarged heart*; the liver and entire system of abdominal veins were found gorged with blood†.

* Op. cit. p. 271.

* It is probable that in this as well as in other cases where it has been stated that the structures of the lungs have become altered by the pressure of a dilated heart, the pulmonary lesions have not in reality been due to this cause, but have resulted either from imperfect expansion, congestion, pneumonic consolidation of the lungs, pleuritic effusions, or other similar conditions.

† As quoted in the London Medical and Surgical Journal, vol. v. p. 61.

I have now endeavoured to trace out, by means of a classified series of fifty-five detailed cases, the several forms in which congenital narrowing of the pulmonary orifice is wont to occur; detailing the various modifications of the lesion from its most complicated form, in which the arterial impediment, becoming established at a period when the heart is still in the condition of a single auricle and ventricle, prevents the development of the cardiac septa, and determines the permanence of the arterial duct, until we arrive at the simplest condition in which the malformation can possibly occur; *i. e.* as a defect which is confined to the pulmonary ostium, and which has been produced at so late a period as to have no influence whatever in preventing the other portions of the heart and its appendages from attaining their full and normal development. I should have been content with illustrating each species of the lesion by merely one or two examples, had it not been for the singular fact, that nearly every instance of this kind of malformation has been found to present certain marked peculiarities which in a striking degree distinguish it from all the other recorded instances to which it is allied; in fact, it appears that scarcely any two cases of this kind perfectly agree: the history of each appears to develope some new feature in cardiac pathology, and to afford some additional insight into the inexhaustible resources by which nature is prepared to enable the central organ of the circulation to adapt itself to those vicissitudes which are so liable to affect it during the progress of its development.

I shall reserve the discussion of many important points connected with the history of the above lesions until I arrive at the consideration of the morbus cæruleus, and the diagnosis, prognosis, and treatment of diseases originating in the pulmonary artery.

Absence of the sygmoid valves.

In many of the cases of narrowing of the pulmonary ostium which have been already cited, the valvular apparatus at the origin of the vessel presented scarcely the faintest resemblance to the natural arrangement of the sygmoid curtains, the development of the valves being especially incomplete in those cases where the imperfect state of the ventricular septum, and other extremely defective conditions of the heart and its vessels, proved that the arterial lesion had occurred during the earlier periods of intra-uterine life. In the larger proportion of these instances, however, there existed some form or other of imperfect valvular apparatus, which must have proved more or less efficient in guarding the vessel against reflux; and, in one only—that observed by Dr. Crampton, (p. 457)—was the vessel entirely destitute of a valvular apparatus: a similar defect was also observed in the following case, which has been recorded by Dr. Favell.*

The subject of this extraordinary malformation was a boy, ætat. 8 years, who died of cerebral irritation. During his illness his pulse was always slow, not exceeding 70 in the minute: the boy was well developed. On placing the ear below the left nipple there was a loud souffle, and it was stated that, if the boy was much excited, or had taken active exercise, his countenance became blue. On examination after death, the heart was found to be of normal size; but, on opening the right auricle, the *foramen ovale* was seen completely unclosed, and capable of allowing the passage of the little finger. There was no communication between the right auricle and the corresponding ventricle, so that the whole of the blood must have passed through the foramen ovale. In the left ventricle, immediately behind the mitral valve, there was a large aperture through the *septum* which opened into the commencement of the *pulmonary artery*: this vessel had no valves. The right ventricle would scarcely contain a small walnut.

This case is extremely interesting when compared with the example by Dr. Blackmore (cited at p. 749). Here we observe a singular adaptation of parts by which the immediate ill effects of the primary lesion,—complete occlusion of the *right* auriculo-ventricular orifice,—were averted: in Dr. Blackmore's case was displayed a still more extraordinary alteration in the course of the circulation consequent upon non-development or early occlusion of the *left* auriculo-ventricular opening. The faulty condition of the pulmonary artery in either case, although apparently secondary to the other cardiac lesions, was evidently the principal cause of the lividity of surface, and symptoms of cardiac obstruction, from which the patients suffered. It will be noticed that, both in this case and in that related by Dr. Crampton, the valveless pulmonary artery communicated directly with a small muscular cavity which in reality merely formed an appendix to the ventricle from which the vessel received its supply of blood: it is probable that this arrangement of parts in some degree compensated for the absence of a valvular apparatus, by preventing the refluent blood from being immediately thrown back into the ventricle from which it had been propelled. It is to be regretted that it has not been stated whether the bruit which was heard in both of these cases was synchronous with the diastole or the systole of the heart. There can be little

* Provincial Med. and Surg. Journal, vol. iii. p. 440.

doubt that in cases of this description rupture or inflammation of the valvular apparatus, occurring at a very early period of fœtal life, has been followed by its absorption. In another portion of this memoir I shall have to shew that, even in adult life, the valvular apparatus of the pulmonary artery may be almost entirely destroyed and removed by disease.

The pulmonary artery furnished with two semilunar valves.

Several cases of this description have been recorded, and I have already cited one by Dr. Houston, and another by M. Duret, in which the valvular defect was attended with a contracted state of the artery. In the following instances this malformation was apparently not accompanied by congenital narrowing of the vessel. It is, however, evident that, in an example of the kind adduced by Sandifort*, there must have originally existed some serious cause of obstruction at one or other of the cardiac outlets.

The heart of a 7 months male fœtus was found to be almost round. The left ventricle, which was very small, communicated with the right by a foramen in the *septum ventriculare*. The aorta communicated with both ventricles, but more freely with the right. The *pulmonary artery* was of the usual size, but was only furnished with two valves. The *arterial canal* was narrow, resembling a little branch of the pulmonary artery. There was only one left pulmonary vein.

The following case is rendered of great interest, by the fact that, in the aorta as well as in the pulmonary artery, two sygmoid curtains only had been developed.

Dr. Taylor attended a bricklayer of intemperate habits, ætat. 38 years, who died of pneumonia, pleurisy, and pericarditis, and in whom the physical signs of hypertrophy of the heart and regurgitant disease of the aortic valves were very apparent. On inspection, the heart was found to present the following lesions. The pericardium bore traces of inflammation, and contained two ounces of turbid serum. The aortic valves admitted of regurgitation; the orifice of the aorta was about half closed by numerous large vegetations adherent to the sygmoid valves; these vegetations were partly soft and of recent formation, and partly firm and calcareous; there were only two sygmoid valves in the aorta. The mitral was slightly thickened; its orifice admitted three fingers. The walls of the right ventricle were a little thickened. There were only two sygmoid valves in the *pulmonary artery*: it was not ascertained whether these valves were efficient, but they presented no disease whatever, neither thickening nor any other change.* Dr. Taylor also refers to an example of this malformation related by Briquet.

* Observat. Anat. Path. lib. iii. c. i.

Dr. Graves details the case of a man, ætat. 66 years, who died with hydro-pericardium and pneumonia, in whose heart the following appearances were discovered. The *pulmonary artery* contained a fibrinous clot, which presented the usual division produced by the branches of that vessel. There were only two valves, and they were both coated with a recent deposition of lymph, which was, in some situations, almost a quarter of an inch thick. A small part of this lymph was accidentally removed while examining the valves, and the latter were seen much thickened and opaque; in this respect contrasting in a very remarkable manner with the valves of the aorta, which were quite free from disease.†

From the duration of the lives of several of those who have been the subjects of this kind of malformation, it is evident that the valvular defect is not necessarily attended with regurgitation, so long as the tissues of the curtains remain in a sound condition, but parts thus malformed appear to be extremely liable to undergo various morbid changes, the occurrence of which usually determines the fatal issue of such cases. This is exceedingly well illustrated in the following case by Mr. Paget:—

A girl, ætat. 20, of irregular habits, and the subject of renal disease, died apparently from the effects of an extensive deposition of coagula upon the valves and in the branches of the *pulmonary artery*. Mr. Paget found that this vessel was furnished with only two sygmoid curtains, which were thickened and opaque, and had on their free borders and adjacent surfaces large growths of brownish-yellow, soft, fibrine-like substance, which firmly adhered to them, and which had in their interior small grains of earthy substance‡.

In commenting upon this case, Mr. Paget directs attention to the fact, which he believes has hitherto passed unnoticed, that in the majority of cases in which only two valves have been found in the aorta or pulmonary artery, those valves have been often extremely diseased. This fact, Mr. Paget adds, is very important; for it affords evidence (which is confirmed by several other observations) that parts which, from defective or erroneous development, are wrongly shaped, are often at the same time imperfect in their tissue, and are therefore very liable to disease

* Lancet, Dec. 4th, 1841, p. 314.

† A System of Clinical Medicine: Dublin, 1843, p. 905.

‡ On Obstruction of the Branches of the Pulmonary Artery: Medico-Chirurgical Review, vol. xxvii. p. 182 (1844).

(p. 188). Previously to the appearance of the above observation, it occurred to me to remark that, where absence of the septum of two of the aortic sygmoids occurs as a congenital lesion, manifest traces of a more recent disease of the valves are not unfrequently also present, in the form of irregular thickening of the ill-shaped curtains, and deposits of small fibrinous concretions upon portions of their surfaces; as, independently of the great liability of the parts so malformed to suffer from vicissitudes in the circulation, they are also very apt to be implicated in the derangements of the heart and its appendages which frequently coexist in these cases*.

Absence and congenital closure of the arterial duct.

Independently of those cases in which the office of a ductus arteriosus is found to be performed by a distinct vessel passing from the right ventricle to the aorta, and of those in which the place of the duct is supplied by a communication between the pulmonary artery and one of the large vessels of the arch, there are several kinds of cardiac malformation in which there is every reason to believe that closure of the duct exists as a congenital lesion. Although, in the majority of these cases, no vestige of the duct is discoverable subsequently to birth, it does not appear probable that, in any instance, this canal has been originally wanting; unless we presuppose the existence of some primary defect in the branchial vessels. It seems to be more feasible that the communication in question, having been rendered unnecessary by the establishment of certain abnormal conditions in other portions of the cardiac apparatus, has either become closed at the period when the arterial root and branchial vessels assumed the condition of the primitive, systemic, and pulmonary arteries, or has undergone contraction and absorption at a somewhat more advanced stage of fœtal existence.

Absence of the arterial duct has been observed in coincidence with the following conditions of cardiac malformation:—where the ventricular portion of the heart is single, giving origin to a single arterial trunk;—where a single artery originates from ventricles which are partially divided by a septum;—where the pulmonary artery is narrow. Absence of the duct has also been observed (by Dr. Blackmore) in the case, already cited, of obliteration of the mitral orifice with narrowing of the pulmonary artery, and has been remarked in a singular case of transposition of the aorta and pulmonary artery which will be detailed in a subsequent part of this memoir. The two latter instances appear to be solitary examples of this kind of lusus; but, in the three former kinds of malformation, it has been more frequently observed. Allusion has been already made to the fact that, in those instances where the ventricular portion of the heart is single, giving origin to a single arterial trunk or to a distinct aorta and pulmonary artery arising by separate orifices, or in which the ventricles, communicating by an opening in the septum, send off an artery which afterwards separates into pulmonary and systemic branches, the ductus arteriosus is absent,—this peculiar arrangement by parts rendering its presence unnecessary. I shall quote the following examples in illustration of this point; as instances of these kinds of malformation are of by no means frequent occurrence.

In a male fœtus, of eight months, F. R. Meckel* found the heart to be nearly equal in length and breadth; its apex was divided into two tubercles. The aorta, which was one-half wider than natural, arose from the middle of the base over the septum, there giving off a very narrow *pulmonary artery* as a branch which passed behind the trachea towards the vertebral column. There was *no canalis arteriosus*. The *septum of the ventricles* was perforated by an opening of the measure of one line vertically, and four lines in width†.

In the following case the malformation was in some degree analogous to that observed by Ant. de Pozzi, Tiedemann, Chemineau, and others (in cases cited at pp. 276—278). But the heart, although essentially single, in reality presented four cavities.

In 1842, a female child, ætat. 4 months, and of whom it was impossible to obtain any precise information, was deposited in the Hôpital des Enfans Trouvés, of Paris. The child was seized with dyspnœa several times in the course of the day, the attacks being accompanied with a bluish tinging of the whole surface of the body; it was accordingly sent to the infirmary. Here, on the ensuing day, it presented general cyanosis, and the tongue, anus, genitals, and adjacent parts, were of a blackish-violet colour. Asphyxia supervened five or six times in the twenty-four hours. The heart beat 136 times per minute, and a marked *bruit de soufflet* replaced the normal sound in the heart during its diastole. About ten days after this, the child died of dyspnœa: nearly all the anterior half of the thorax was found occupied by the pericardium, and, on laying open this, it was seen that the heart consisted of only one

* Guy's Hospital Reports, vol. vii. p. 408 (Oct. 1842).

* Reil's Archiv. Bd. 9, p. 437, quoted by Hein.

† This case almost precisely resembles one related by M. F. Tiedemann, and cited at the commencement of this paper (p. 277). I had not met with this instance when I cited that by M. Tiedemann.

ventricle and one auricle, the deep sulcus between which was filled up with a process of the left lobe of the liver (with, of course, a portion of the diaphragm). The auricle, which was much larger than the ventricle, was nearly spherical, and its sides, which were about one-eighth of an inch in thickness, were furnished internally with strong columnæ carneæ (musculi pectinati?). At its posterior part the two venæ cavæ opened into it by a common mouth, or a sort of sinus, apparently a rudiment of a right auricle. The pulmonary veins terminated in their ordinary manner. The ventricle was of a conical shape; its walls were much thicker on its left than on its right side. Three openings existed in its base. The auriculo-ventricular closed by a true mitral valve, the aortic opening; and on the right side of the latter an opening into a cylindrical sac which seemed to represent the right ventricle, but had no communication with the auricle. The *ductus arteriosus* was wanting. There was no malformation in any of the viscera except the heart, nor in any part of the arterial system*.

Here the circulation must have pursued exactly the same route as in the case by Dr. Favell, cited at the commencement of this chapter: but, in the present instance, the malformation appears to have been more extreme, especially that of the auricular portion of the heart.

M. Hein (op. citat. p. 29) appears to have been the first author who noticed the coincidence of absence of the arterial duct with great narrowing of the pulmonary artery; citing the cases by Otto, Knox, Meckel, and Steno, in illustration of this fact. To these instances I have added the examples by MM. Lexis, Aran, Huss, Gintrac, and Dr. Blackmore†, which taken together are sufficient evidence of the fact that narrowing of the pulmonary orifice is frequently attended with absence of the arterial duct. It is difficult to account for the occurrence of this coincidence otherwise than by concluding that early obliteration and absorption of the duct has been followed by an imperfect development of the artery; the canal of which has, up to the period of birth, remained merely of sufficient size to transmit the slender current of blood which circulates through the lungs previously to the commencement of respiration.*

The question of the *possibility of closure of the arterial duct occurring previously to birth* is one of extreme interest and importance, especially in a medico-legal point of view. That the passage may become closed during the earlier stages of intra-uterine life is rendered certain by the facts already adduced; whether occlusion of the canal and conversion of its tissues into an impervious cord can occur at a later period of fœtal existence is a more doubtful question, but one which I believe may, not improbably, be ere long answered in the affirmative.

A case has recently occurred at Ayr, in which there appears to be strong reason to believe that closure of the duct had occurred previously to birth. The medical facts of this case, so far as they have been afforded, are as follow. The body of a female child was discovered buried in a waste ground enveloped in a woollen bag. The body was found to be in a state of putrefaction, with desquamation of the cuticle. It weighed five pounds, and was twenty inches in length. Its mouth and nostrils were stuffed with flax. The umbilicus was in the centre of the body, the cord cut close to the abdomen, and left without ligature. The scalp was covered with hair, and the nails were full-grown; a large portion of the brain, which had been reduced to a pulpy state, had escaped through the openings of the skull. There was an extensive ecchymosis all over the fore part of the neck, and an effusion of blood on the exterior of the trachea. The heart and lungs weighed one ounce; the lungs were collapsed: the right was considerably decomposed, and sunk when put in water. The left was of a red colour, and firm in texture, and floated on the surface when immersed in a vessel filled with water; but on pressure there was no crepitation. The right side of the heart was filled with coagulated blood, the *foramen ovale* partly open, and the *ductus arteriosus* impervious. The liver was large, and of a leaden hue; the *ductus venosus* almost obliterated, and the meconium was found in abundance in the lower bowels.

Strong circumstantial evidence was adduced to prove that this infant came into the world alive shortly before 6 o'clock A.M, and that its body was deposited in the

* Gaz. des Hôpitaux, Supp. Dec. 1842, and Lancet, 1843.

† While citing examples of narrowing of the pulmonary artery attended with absence of the ductus arteriosus, I omitted an example related by M. Deguise, and cited by M. Ollivier, Dict. de Méd., art. Vaisseaux Pulmonaires, of a woman, ætat. 20, who suffered from cyanosis, debility, palpitation, and dyspnœa; a bruit de souffle was prolonged in the course of the *pulmonary artery*. On inspection, this vessel was found to be extremely narrowed. The ventricles and auricles respectively communicated with each other, and the *arterial canal* was absent. The precise meaning of the latter expression is rather doubtful.

* Otto alludes to other instances observed by Stenson, Bartholini, Kreysig, and Chemineau, in which the duct was entirely absent, and also makes slight reference to the existence of the same defect in a monster, with abdominal cleft and hernia cerebri, but I am not acquainted with the details of these cases, unless the instance by Chemineau be one of non-development of the ventricular septum, to which I have alluded at the commencement of this paper.

spot where it was subsequently discovered between 10 and 11 o'clock, A.M. on the same day. This train of evidence, if confirmed, would lead to the conclusion, either that the duct became closed almost immediately after birth (for it cannot be believed that this infant survived its birth more than four hours and a half, to say nothing of the probability that it did not respire even for so many minutes), or that the child had come into the world with the duct nearly or quite closed. The facts of this case, however, were not proved in a sufficiently demonstrative manner to render it an example upon which it is possible to argue with any degree of certainty; still, it is one, which unquestionably very decidedly suggests the probability that closure of the duct in the ordinary manner may occur previously to birth, and one which strongly shows the necessity for further examination of this most important physiological question. No fact can be more clearly ascertained than that the foramen ovale may become closed previously to birth; and it appears to me that, as the question at present rests, no medical witness is justified in declaring that the existence of a contracted or closed state of the arterial duct is a certain evidence that the infant has respired*.

We have, at present, no absolutely demonstrative knowledge of the causes which naturally produce the closure of the arterial duct: we are, therefore, not justified in declaring that those causes may not, in certain instances, be brought into operation previously to the birth of the child; many instances of cardiac malformation countenance this idea, and there are numerous facts which, to a certain degree, suggest, although they do not absolutely confirm, an opinion that the commencement of the process of contraction in the ductus venosus, foramen ovale, and arterial duct, may possibly be among the causes which determine the expulsion of the fœtus from the uterus at the ninth month.

Low division of the pulmonary artery.

The pulmonary artery has, not very unfrequently, been found to divide into its two main branches at a very short distance above its origin. In examining the body of a young girl who died of a passive aneurism of the left cavities of the heart, M. Cassan found that the pulmonary trunk did not, in reality, exist at all, but that the vessel divided just above its origin into its two branches. These two branches were of the ordinary width.*

In the case by Mr. Bloxham, already cited, of a child, ætat. 3 years, the pulmonary artery was extremely narrow, and bifurcated immediately on its exit, so that the trunk was not, in any part of its circumference, more than a line in length. Slighter degrees of this kind of malformation are not unfrequently noticed.

MALFORMATION BY EXCESS OF THE PULMONARY ARTERY.

The pulmonary artery arising from the heart by a double origin.

The only instance of this kind with which I am acquainted is that related by Kerckring.† The subject of this malformation was an infant about 3 months old, in whom the heart was divided into three ventricles; *i. e.* the right was double, and from each of its two cavities there arose a pulmonary artery which united above into a common trunk. The heart had been carelessly removed from the body for another purpose, so that the distribution of the cavæ and other vessels which had been cut away was not observed.

Neither the above account, nor the representation of the heart (which is exactly copied from Kercking's plate in the subjoined cut), render it perfectly clear that the vessel which took its origin from the two ventricles was, in reality, the pulmonary artery. More than one case has been recorded in which the aorta arose by a double origin;‡ and, upon examination of the



B, Two right ventricles of the heart.
C, Left ventricle.
D, Pulmonary artery given off from both right ventricles.

* It does not appear to have been stated in evidence in the above case whether the pulmonary orifice and other portions of the heart and its appendages were in a natural condition. In any instance where there exists narrowing of the vessel or any other serious malformation of the heart, or in which those who examined the body have neglected to observe the previous condition of each cavity and outlet of that organ, the fact of obliteration of the arterial duct certainly cannot be received as a proof that the infant has been born alive.

* Archiv. Générales, P. S. t. xiii. p. 82.
† Spicilegium Anatomicum, Obs. 49, p. 129-40.
‡ Dr. M. E. White, of Carlow, has described the heart of a double-headed fœtus in which there were three ventricles, but here a single vessel was placed anteriorly, and gave off a single pulmonary trunk; while, from the two posterior cavities, the systemic vessels arose. (Dublin

figure, it will be observed that the long single vessel formed by the union of the two ventricular branches has rather the appearance of an aorta than of a pulmonary artery.

Unusual number of valves.

The instances in which the pulmonary artery is found to be furnished with four sygmoid valves are by no means extremely rare.* Dr. Todd (Cyclop. of Anatomy and Physiology) speaks of the occurrence of five valves in this vessel; Malcarne (quoted by Dr. Graves) mentions a case where the aorta divided soon after its origin, and was furnished with five valves, but I have not been able to meet with the details of any example in which a similar arrangement occurred in the pulmonary artery.

Dr. Theophilus Thompson has collected some interesting facts with relation to this species of irregularity in illustration of the following case†:—

The author was requested to visit a woman, æt. 38, suffering from great drowsiness, with lividity of the complexion, a purple and swollen state of the lips, distension of the external jugular veins, and anasarca. The pulse was rapid and feeble; the impulse of the heart rather weak; the first sound shorter, and more flapping, the second less distinct, than natural; both sounds were, however, unattended with roughness, and there was no evidence of any obstacle to the circulation. The debility gradually increased, her legs became more and more œdematous, and at length erysipelatous and gangrenous, when she died. This patient, although never robust, had enjoyed an average share of health until attacked with Asiatic cholera during the prevalence of that disease in this country, after which her strength was permanently impaired, and she occasionally complained of palpitation of the heart on going up stairs. Two years before her death she suffered from an attack of fever. From this time she exhibited a livid complexion, and a peculiar drowsy apathetic appearance, which induced visitors to suppose her idiotic. On inspection, there was found some effusion into the peritoneum, pleura, and pericardium, and serous infiltration, with redness of the bronchial tubes. The heart was larger than natural, and exhibited a circumscribed dilatation at the part of the right ventricle more immediately connected with the right ventricle. This ventricle was found to be formed of two cavities, separated from each other by an imperfect septum. This septum was composed, not of a uniform fleshy wall, but of decussating and hypertrophied columnæ carneæ, some of which, separating from each other near the base of the ventricle, left an aperture of communication about an inch long and half an inch broad. The arterial chamber of the right ventricle was rather less spacious than that adjoining the auricle, which corresponded in size and appearance to the right ventricle in its natural condition. The connecting orifice was partially covered by one of the divisions of the tricuspid valve*. The walls of the left auricle, and of both the ventricles, were of natural thickness; but the right auricle was twice as thick as the left. The *pulmonary artery* was furnished with four valves, which were found, on admeasurement, equal in size. Each of the valves was well-developed, furnished with a corpus sesamoideum, and about nine-tenths of an inch in diameter. As each of the valves was of natural size, an additional valve was rendered necessary by the preternatural width of the pulmonary artery, the circumference of which exceeded that of the aorta by nearly an inch. The author justly observes, that this specimen furnishes an exception to the rule laid down by Meckel.

Medical Press, vol. i. p. 212.) Dr. Chichester has detailed (London's Magazine of Nat. History, vol. i. p. 135, quoted by Dr. Paget) the appearances observed in the dissection of a monstrous sheep, with one head, one heart, and one stomach, but with the rest of the body generally double: from each ventricle sprang an aorta: of the two vessels one turned to the right, and the other to the left. The one which turned to the left went down in the natural direction, while that which turned to the right crossed the right vertebral column, and passed down on the outer side of it. A transverse canal of somewhat small calibre passed down from the beginning of the turn of the arch on one side to the same part on the other side, thus forming a communication between the two aortas.

* Dr. Graves refers (in his Clinical Medicine, p. 905) to the specimens in the College of Surgeons of Ireland, which exhibit the aorta with four valves.

† Med. Chir. Trans. vol. xxv. p. 247.

* A malformation of the right ventricle in some measure similar to that observed in Dr. Thompson's case, but unaccompanied with an analogous state of the valves, has been described by Dr. Fletcher. The man from whom the specimen was taken was about 68 years of age, an itinerant collector of rags, who, after a walk of five or six miles, was found dead in his bed. It was stated that he had latterly been rather asthmatic. The tricuspid valve was healthy; across the cavity of the right ventricle was a muscular partition, dividing it into two unequal parts, the larger next the auricle, the smaller communicating with the pulmonary artery. The partition seemed made up of carneæ columnæ, and was perforated by an opening capable of admitting the little finger, the edges being semi-cartilaginous; an opening large enough to admit a goose-quill communicated with the left ventricle, and the larger cavity of the right ventricle. The foramen ovale was capable of being opened by a probe. The left auricle and mitral valve were healthy; in the left ventricle the opening from the right ventricle was seen before (below ?) the origin of the aortic valves, two of which had ossific deposits in their substance. The aorta was slightly dilated. The veins of the arachnoid were congested, and there was effusion of fibrinous serum between this membrane and the pia mater.

A case by M. Obet, in which a very similar condition of the ventricle was observed in connection with arctation of the pulmonary orifice, has been already detailed.

"Omnes in eo conveniunt valvulæ numero imminutas mole augeri, numero auctas mole minui."

It will be found, however, that the first, third, and fourth of the following examples, corroborate the statement of Meckel, and correspond with two cases observed by Petsche and Bizot*, in one of which the additional valve was found singularly small, in the other quite rudimentary. The first in some measure corresponds with an instance mentioned by Morgagni, in which one of the valves was larger than the rest. In the fifth instance the condition of the artery closely resembled that described by Dr. Thompson, the whole of the valves being regular and symmetrical in form.

In the Museum at Guy's Hospital there are four preparations (numbered 1435^{25} 1413^{25-30}) in which various modifications of this condition of the artery are remarkably well displayed.

In one of these, three of the curtains are well formed, but the vessel is also furnished with an additional sygmoid pouch, which is capable of holding a pea.

In a second there are four valves, but the septum between two of these is longer than that which separates either of the others, the free edges of the curtains having apparently been stretched downwards.

In the third specimen there is a very minute imperfectly formed additional valve, formed by a little curtain supported above by slender isolated cords; these cords attach the upper margin of the valve, almost at its central part, to the arterial walls.

The fourth preparation displays a still more minute extra valve, which is not only attached laterally to the artery by tendinous cords, but also presents a thin membranous bridle, which extends upwards from that portion of the free edge at which the corpus arantii is usually situated, and is united with the arterial wall above. This is the only specimen which I have seen in the human subject where a sygmoid curtain was attached to the artery by a tendinous cord occupying the situation of the corpus arantii, but such an arrangement of parts is observed to be permanent in the upper row of valves defending the aorta of the shark; and there is every reason to believe that, in the human heart, the primitive condition of a sygmoid valve is that of a membranous curtain attached to the vessel at its upper part by isolated tendinous cords. I have stated the grounds on which this conclusion is formed in a previous essay†, and I have no doubt that the corpora arantii are the vestiges of the attachments of the central chordæ tendineæ of the valves.

The same collection also contains another specimen (marked 1413) which presents four perfectly symmetrical valves, which are in every respect healthy and natural in structure, except that here, as in most of the other specimens, some of the lateral attachments of the curtains are formed by isolated cords; a condition of parts which I believe to be in every instance congenital, and not the result of atrophy of the tissues of the valves.

* Haller, Collect. Anatom. Dis. vol. vi. p. 774;—Mém. de la Société d'Observation, vol. i., quoted by Dr. Thompson.

† Guy's Hospital Reports, vol. vii. p. 414, and plate 1.

Origin of extra branches from the pulmonary artery.

A few instances have been noticed in which one or other of the subclavian arteries took its origin from the pulmonary. Breschet* mentions a case in which the left subclavian arose from this vessel; and J. F. Meckel† has described the origin of the right subclavian from the division of the pulmonary artery in a calf. Instances in which the descending aorta, left subclavian, &c., apparently arose from the pulmonary artery, and in which the latter vessel communicated with the innominata or left subclavian, belong more properly to another division of this subject.

The lungs supplied by additional branches from the systemic circulation.

In the course of the foregoing observations, various instances have been alluded to in which obliteration or contraction of the pulmonary artery was in some measure compensated by the existence of branches passing to this vessel from the subclavians, or arch of the aorta, or by the establishment of a permanently dilated condition of the aortic bronchial arteries. In addition to these, Otto refers to several cases in which, besides the usual pulmonary artery, the lungs were supplied by accessory arterial branches; for example, an instance where a large branch from the aorta, in the region of the sixth dorsal vertebra, passed to the lobes of the right lung‡. Another case, where a single twig passed to the under lobe of the left lung§; and one in which very small pulmonary arteries passed equally to both lungs from the pericardial arteries‖.

A very remarkable instance of this kind has been related by M. A. Maugars, of Angers.¶ The subject of this malformation was a child, æt. 7 years, in whom a considerable artery, which arose from the

* Quoted by Hasse.

† Tabul. Anat. Path. Fas. ii. p. 2, cited by Otto.

‡ Huber, in Anat. Helvet. vol. viii. p. 85.

§ J. F. Meckel, in his Archiv. f. d. Physiol. vol. vi. p. 454.

‖ Jacobson, in Meckel, vol. ii. p. 154.

¶ Sidellot, Recueil Period. vol. xiii.; and in Corvisart, Journ. de Méd. An. x. Pluv.

superior anterior and right side of the abdominal aorta, and ascended between the aorta and œsophagus, entered the chest through the œsophageal opening of the diaphragm, having given off, at a short distance from its origin, the right sub-diaphragmatic artery. Immediately above the diaphragm, and above the œsophagus, it divided into two branches. Each of these entered the posterior inferior and internal part of either lung, and was distributed throughout the inferior lobes. The trunk of the artery, and its principal divisions, gave off no other branch than the sub-diaphragmatic, and were not accompanied by any corresponding vein. The trunk and right branch of the pulmonary artery itself were of the ordinary dimensions, but the left branch was much smaller than usual, not equalling the right by one-half. This defect was evidently compensated by the accessory branch from below. On injecting the pulmonary arteries, it was found that they inosculated freely with the branch derived from the abdominal trunk. The pulmonary veins were distributed in the usual manner. The lungs were sound, but were rather larger than usual. The heart was also rather enlarged.

This last case appears to afford a type of the whole of the instances in which the lungs are supplied with accessory vessels from the aorta or its branches, an arrangement which seems to have been invariably determined by the existence of some considerable impediment to the circulation through the pulmonary artery during the earlier periods of cardiac development. There are strong reasons for believing that, in many instances of contraction and obliteration of the pulmonary ostium, these lesions have been partially compensated by the distribution of additional arterial branches from the systemic vessels to the lungs, an arrangement which has been overlooked in dissection. It would add greatly to our knowledge of the abnormal anatomy of the blood-vessel system if the plan of injecting the arteries and veins, and then carefully dissecting the parts *in situ*, were adopted in every case where there was reason to believe that death had resulted from congenital malformation of the heart.

Permanence of the arterial duct.

In a previous essay* I have endeavoured to trace out the principal causes which occasionally determine the non-closure of the arterial canal subsequently to birth; and have also attempted to afford some elucidation of the means by which the natural obliteration of this vessel is effected in the healthy infant: it now remains to analyse and classify the great variety of instances of congenital malformation of the heart and its large vessels in which permanence of the arterial duct constitutes the leading feature in the malformation; and it is certainly extremely interesting to observe how many different forms of congenital obstruction are attended with this condition: and to remark the singular diversity of lesions, the ill effects of which are, to a greater or less degree, mitigated by its establishment.

The following are the principal conditions under which non-occlusion of the arterial canal has been found to occur:—(1.) Where the duct occupies an unusual position; (2.) Where the orifice of the pulmonary artery is obliterated or congenitally narrowed; (3.) Where the aorta arises from the right ventricle and the pulmonary artery from the left; (4.) Where the orifice of the aorta is closed; (5.) Where the upper portion of the descending thoracic aorta is contracted or obliterated; (6.) Where the foramen ovale becomes closed previously to birth.

It has been very justly considered that the existence of any permanent cause of embarrassment to the circulation, similar to those which are present in most of the above-mentioned kinds of malformation, is sufficient to determine the non-closure of the arterial canal subsequently to birth; but it is also probable that various other circumstances, such as the existence of some severe, but temporary, cause of obstruction in any portion of the vascular circle of the child either before or after its birth, a diseased condition of the tissues of the canal, or a failure in the operation of those means by which the natural closure of the duct is usually effected, may determine its persistence, as a direct communication between the pulmonary and systemic trunks, throughout the entire life of the individual. To complete the series of instances where non-occlusion of the duct has been observed, we must add those in which it is associated with (7) imperfection of the ventricular septum, and with (8) an unclosed state of the foramen ovale; together with those (9) in which there is not discoverable any other appearance of malformation or defect in the heart or its appendages.

I have already adduced cases in illustration of several of these kinds of congenital aberration: the following interesting examples will assist to complete the series.

I. *Cases in which the arterial canal occupies an unusual situation.*

Failure of the process by which the duct becomes materially closed is not by any means invariably determined in cases where this vessel is irregularly distributed, but it is

* On Permanence of the Ductus Arteriosus; and Constriction of the Thoracic Aorta; and on the means by which the Duct becomes naturally closed, Medical Gazette, vol. xxxvi. p. 187.

very remarkable that, in by far the larger number of instances where any deviation from its usual course has been observed, the canal has remained in a pervious condition. The following example is, I believe, unique.

The arterial duct arising from the right ventricle.

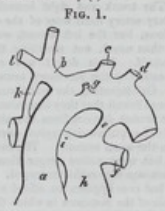
Wrisberg* refers to the case of a girl, æt. 7 years, in whom the *ductus arteriosus*, which was as large as it usually is soon after birth, arose from the right ventricle close to the orifice of the pulmonary artery. In this manner the aorta received blood from both ventricles.

Communication between the pulmonary and subclavian arteries, associated with other congenital defects in the heart or its appendages, independently of contraction of the pulmonary orifice.

I have already cited (pp. 283, 374, 452) instances in which an obstructed condition of the arterial outlet of the heart was in some measure compensated by the existence of an abnormal communication between the pulmonary trunk and the left subclavian artery, adopting the view of Dr. Paget, that, in these cases, the irregularly disposed arterial canal is not in reality the ductus arteriosus, but the remains of a communication which, during the earlier period of fœtal life, naturally exists between those portions of the branchial apparatus which afterwards form the right and left pulmonary and subclavian arteries. The following remarkable case has been cited by Dr. Paget, and appears to afford unquestionable evidence of the correctness of his view.

The pulmonary artery communicating with the aorta in the usual position of the canalis arteriosus; a vessel at the same time uniting the left branch of the pulmonary with the innominata artery. M. G. Breschet describes† the case of a male infant which lived 6 weeks, and during the brief period of its existence suffered from difficulty of breathing, attacks of vomiting and convulsion, and an habitual drowsiness, with coldness of the extreme parts of the body. On examination, the heart, which was of ordinary size, was found to be situated in the median line of the thorax, its point being directed to the left. It had but one auricle and one ventricle, which communicated by so large an opening that the organ might be considered to consist only of a single cavity. The aorta (*a*) arose in front and to the right, and gave off from the upper part of its arch (which passed to the left) the brachio-cephalic (*b*) and left carotid and subclavian arteries (*c*, *d*), and at its under part received (*e*) a large vessel (the arterial duct), which M. Breschet describes as arising from the left branch (*f*) of the pulmonary artery. A small branch (*g*) was also distributed from the anterior part of the arch to the thymus gland. Further behind the aorta, and to its left, arose (*h*) the pulmonary artery. Between the right branch (*i*) of this vessel and the brachio-cephalic trunk there existed a communicating vessel (*k*), which was larger and narrower than the arterial duct, and which was inserted just at the root of the right subclavian artery (*l*).

FIG. 1.



I am not acquainted with any other instance in which two direct communications existed between the pulmonary artery and systemic vessels in the form of a ductus arteriosus and a branchial canal. M. Breschet's case also forms a remarkable exception to the rule which I believe generally obtains—that, where there is total deficiency of the ventricular septum of the heart, the arterial duct is absent.

A single branchial canal uniting the pulmonary with the left subclavian artery.

One of the earliest recorded instances of the existence of a branchial duct, is that mentioned by John Christian Reinmann.* In the body of a beggar, who had died of suffocative catarrh, this author found an arterial canal ascending from the pulmonary artery and uniting with the left subclavian.

Dr. James Jackson has detailed the history of a female child, ætat. 5 years, whose death was principally due to the effects of an acute gastro-enteritic attack of five days' duration. She lay quiet during nearly the whole of her last illness, and was disposed to stupor. The countenance "was not very morbid," and was of a good colour; the respiration was most uncommonly accelerated, and was peculiarly irregular; the action of the heart was rapid, troubled, and confused; the palpitation of the heart, and

* In Gottingische Anzeigen, 1778, Num. 30; and Sandifort, Obs. Anatom. Pathol. t. iii. c. i. p. 39.

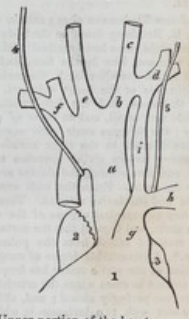
† Mémoire sur l'Ectopie du Cœur, p. 9.

* Nova Acta Acad. Nat. Cur. T. i. Obs. 74, p. 302.

shortness and rapidity of respiration, had long and often been noticed by those about the child. These symptoms had been increased by exercise. The child was always unwilling to go upstairs; she always went up slowly, and often begged to be carried up. She had not, however, been subject to such paroxysms of dyspnœa as seemed to those about her to constitute disease. It was not remembered that her countenance or extremities had ever been livid, or even otherwise than fair and florid. Her death was rather sudden. The mucous membrane of the entire alimentary canal was found to present marked traces of intense inflammation; the lungs were natural; the pericardium contained more fluid than is usual; the heart was uncommonly large; the auricles were very greatly distended; the *pulmonary artery* was much larger and more prominent than the aorta: it formed a small arch, and divided into two branches immediately as it began to descend, after having made its curvature. These branches went to the lungs as usual; but the left, which was the larger of the two, distributed *a small branch to the left subclavian*. The aorta arose almost perpendicularly from the heart, passing to the right of the arch of the pulmonary artery, and, after ascending rather more than two inches, it divided into two branches, which immediately subdivided into the carotids and subclavians.* The *foramen ovale* had not closed, and there was an *opening between the ventricles* large enough to admit the passage of a finger.†

Otto Bernhard has described the case of an infant which died very shortly after birth, in which the viscera of the chest and abdomen were generally normal, and the body of the heart was naturally formed; but the aorta (*a*) presented a singular aberration: it first ascended perpendicularly to the region of the fourth or third dorsal vertebra, and then turned to the right; so that, above the right bronchus, it formed its arch, which was directed to the right; then, curving behind the right bronchus, it descended on the right side of the spine. The truncus anonymus (*b*), which was the first vessel given off on the left side of the arch, separated into the left carotid (*c*) and subclavian (*d*). The right carotid (*e*) and subclavian (*f*) arose by separate orifices from the right portion of the arch. The *pulmonary artery* (*g*) was naturally formed, and divided into its two branches, of which the left (*h*) passed directly to the left lung, and the right (not represented in Bernhard's figure) entered the right lung, after having curved behind the ascending aorta, and in front of the right bronchus and descending aorta. The *ductus arteriosus* (*branchial canal*) (*i*) arose from the pulmonary artery rather from its left branch: it was still pervious, and longer than common, and entered the left subclavian artery (*d*) at the distance of a few lines beyond the separation of that vessel from the innominata trunk.*

FIG. 2.



1, Upper portion of the heart.
2, 3, Auricles.
4, 5, Right and left vagi nerves.

These, with the instances by MM. Obet and Holst, already quoted, are the only cases with which I am acquainted where an abnormal communication existed between the pulmonary and subclavian or innominata arteries; but cases have been related in which the vestige of a communication of this kind was found uniting the vessels in the form of an impervious cord.†

* The obscure wording of this description, and the absence of any allusion to the descending portion of the aorta, leaves the true nature of the malformation in the systemic trunk a matter of considerable doubt.

† London Med. and Phys. Jour. vol. xxxiv. pp. 100-2.

* De Arteriarum e Corde Prodeuntium Aberrationibus, p. 14. Berolini, 1813.

† In his splendid work on the Surgical Anatomy of the Human Body, Mr. Quain has given representations of a preparation in Guy's Museum, where the closed remains of a *branchial duct* passes upwards to a pouch in the aorta, from which the left subclavian artery takes its origin (p. 20, figs. 8 and 9). In a case related by Fiorati and Sandifort (and figured by Quain, pl. 7, figs. 1 and 1 b), besides other remarkable abnormal arrangements in the arch of the aorta, an *arterial ligament* (*a*) was found attached to a small pouch (*b*) from the descending arch of the aorta. The left subclavian artery (*c*) arose from this pouch in close approximation to the remains of the canal. The remarkable arrangement of the pneumogastric and recurrent nerve (*d d*) around the remains of this obliterated canal, as compared with the distribution of the left vagus

II.—*The cases in which permanence of the arterial duct attends closure or contraction of the pulmonary orifice* have been discussed in previous sections (pp. 280 and 372), and those in which—

III.—*Transposition of the aorta and pulmonary artery is attended with a similar condition*, will be noticed in a subsequent chapter.

IV.—*Patency of the arterial duct associated with closure of the orifice of the aorta.*

Professor Tiedemann cites a case in which E. L. R. Romberg found in the body of an exposed child, who had breathed with difficulty, who was blue in the face, and died on the fourth day after birth, the cavities of the right side of the heart enlarged, their walls thickened, and the left auricle and ventricle very small, and *no trace of aortic orifice*; the *foramen ovale* wide open, and its valve directed to the right auricle; the *pulmonary artery* giving branches to the lungs, and then sending the *ductus arteriosus* to the aorta. From the arch arose the carotid and subclavian arteries. There was therefore a congenital closure of the origin of the aorta, and beyond this the aorta must have received blood from the pulmonary artery.* A remarkable degree of congenital arctation of the aortic orifice has frequently been noticed in cases where the arterial duct has become perfectly closed; and, although I have met with two instances in which an open state of the canal was attended with a very diseased condition of the aortic sygmoids, I am not acquainted with any example where non-closure of the duct could be satisfactorily traced to this cause; but the possibility of the occurrence of such a result can scarcely be denied.

V. *Patency of the duct, associated with contraction or obliteration of the aorta distal to the left subclavian artery.*

In a previous essay* I have given notes of most of the recorded instances in which a more or less free communication has existed through the arterial canal where the aorta is (1) *constricted or closed immediately above the orifice of the duct*; (2) *where it is constricted opposite to the opening of the duct, and* (3) *where the artery is contracted both above and below the aortic end of the canal.* In none of these cases with which I am acquainted has the communication between the pulmonary and systemic vessels been more than two or three lines in diameter; and, in the majority of examples, the duct has been found of very inconsiderable size: but there is another class of instances, closely allied to these, in which the aorta having become greatly contracted or obliterated distal to the left subclavian artery, the arterial canal forms so wide a communication between the pulmonary and systemic trunks, that the descending aorta appears to be partially or wholly distributed by the pulmonary artery. The three following cases afford remarkably good illustrations of different modifications of this species of malformation.

Contraction of the aorta distal to the left subclavian artery. The arterial duct patent in such a manner as to give the descending thoracic aorta an appearance of being distributed equally by the arch of the aorta and by the pulmonary artery. Dr. Farre relates† the case of a male infant, who, during the first fortnight of his life, appeared like other children; but, after that period, breathed very quickly and began to waste. The skin was always *extremely pale*, and the extremities cold. Œdematous swelling of the legs, and sometimes of the face, took place. The functions of the stomach and bowels were imperfectly performed. He died suddenly, without convulsion, at the age of 8 months. On examination, Sir A. Cooper (in whose collection the heart was preserved), found that the chest was prominent and rounded; the pericardium and each of the pleural cavities contained serum. The heart was large and square; the *foramen ovale* was dilated; the *pulmonary artery* was larger than the aorta, and *arose from both ventricles*, but chiefly

in Bernhard's case, where the canal remained pervious, deserves to be noticed as a point of great physiological interest.

FIG. 3.



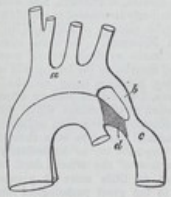
* As quoted from F. Tiedemann's "Von der Verengung und Schliessung der Pulsadern in Krankheiten," in Edinburgh Med. and Surg. Journal for July 1846, p. 149.

* MEDICAL GAZETTE, vol. xxxvi. p. 188.
† Op. citat. p. 15.

from the right. It is described that the ascending aorta (*a*) branched into the arteria innominata, left carotid, and left subclavian arteries, and then dwindled into a small vessel (*b*), which terminated in the descending aorta (*c*); the latter arose as a branch of the pulmonary artery in the situation of the *ductus arteriosus* (*d*). Having received the branch from the ascending aorta, it gradually dilated to its proper size.

The subjoined diagram from Dr. Farr's plate (fig. 10) will give a fair idea of the nature of the malformation. In this, as well as in the two following diagrams, I have defined, by a darker shade, that portion of the vessel which appears to me to represent the pervious arterial duct.

FIG. 4.



Great contraction of the aorta above and below the origin of the left subclavian artery; a wide communication by the arterial duct between the pulmonary artery and the descending aorta, giving the latter vessel an appearance of being distributed by the former. Mr. F. B. Barret, of Stone, Staffordshire, describes the condition of a female infant, which appears to have been born dead. It was the subject of a large ventral hernia, and presented several other remarkable deviations in the form and position of its viscera. The heart lay partly in the chest and partly in the abdomen, and could be seen projecting below the sternum under the abdominal integuments. The cartilaginous margins of the false ribs on the right side were hollowed out, forming a considerable concavity for its reception. Mr. Barret describes the condition of the great vessels as follows:—"The pulmonary artery was very large: it not only divided into its right and left branches, but proceeded upwards, forming a large arch; it then descended, occupied the usual situation of the [descending] aorta, and gave off the arteries to the trunk and lower extremities according to the usual distribution of the aorta. The ductus arteriosus arose from the upper part of this anomalous arch; and from the very centre of the ductus arteriosus was given off the left subclavian artery. The [ascending] aorta proceeded almost in a straight direction upwards, and, shortly after its junction with the ductus arteriosus, it divided into two branches—the arteria innominata, and the left carotid; and these were all its branches."* The condition of the ventricular septum of the heart is not mentioned.

After examining Mr. Barrett's very beautiful sketch, from which the subjoined outline is copied, I am inclined to adopt a somewhat different view with regard to the arrangement of the vessels from that advanced by the author. It appears to me that the narrow canal (*a*) is not the arterial duct giving origin to the left subclavian artery (*b*), but the descending portion of the arch of the aorta, much contracted. That portion (*c*) of the pulmonary artery which lies between the origins of the right and left pulmonary branches (*dd*) and the upper part of the descending arch of the aorta (*a*) is evidently the *canalis arteriosus*, the great width of which renders the pulmonary artery (*c*) perfectly continuous with the vertebral portion (*f*) of the descending thoracic aorta.

FIG. 5†.



Obliteration of the aorta distal to the left subclavian; the arterial duct patent; the pulmonary artery forming a continuous trunk with the descending thoracic aorta. The following case was communicated to Dr. Farr by Sir Astley Cooper.‡ A male infant, whose mother had previously given birth to children the subjects of cardiac malformation, died in convulsions 2 days

* Lancet, 1844-5, vol. i. p. 349.
† In the above cut (fig. 5) the letter *(a)* has been accidentally attached to the left subclavian artery instead of the narrowed portion of aorta from which that vessel springs.
‡ Op. cit. p. 13.

subsequently to its birth, having suffered from dyspnœa and cyanosis. The heart was found to be larger and thicker than natural. The *ventricles communicated* by an opening in the septum. The *pulmonary artery* arose from the right ventricle, and formed the aorta descendens, besides giving off its usual branches to the lungs. The ascending aorta arose naturally, but, passing upwards, terminated in forming the innominata, left carotid, and left subclavian arteries, and a very small branch, which was pervious only in a part of its course, to the descending aorta. The air-cells of the lungs did not appear to be completely filled with air.

Upon examining the subjoined diagram (which is taken from Dr. Farre's plate), the small partially-closed vessel (*a*) in which the ascending aorta appears to terminate, resembles, at the first glance, the remains of a ductus arteriosus; but it is in reality the trunk of the aorta, rendered impervious immediately distal to the left subclavian artery, while the continuation of the canal is formed by the wide arterial duct (*c*) which unites the pulmonary artery (*d*) with that portion (*e*) of the descending aorta which is situated immediately below the point of obliteration.

FIG. 6.



The first and third of these cases are good examples of a rare form of congenital malformation, namely, *the origin of the pulmonary artery from both ventricles.*

Other examples of this remarkable condition of permanence of the arterial canal consequent upon obliteration or contraction of the upper part of the descending aortic arch have been recorded.

Steidelle* has related a case where the aorta and *pulmonary artery* arising as usual, the aorta was entirely distributed to the head and upper extremities; while the pulmonary artery, after giving off two branches to the lungs, continued as the descending aorta without any communication with the ascending aorta. The explanations of the above cases will apply to this, which was evidently also an instance of obliteration of the aorta between the origin of the left subclavian artery and the orifice of the arterial duct.

Otto found the left subclavian artery and descending aorta given off at the separation of the pulmonary branches in a child with phrenic rupture, deficiency of a finger, &c. (Breslau Museum, No. 8025.) In this case the obliteration or contraction of the aorta must have occurred between the origins of the left carotid and subclavian arteries. I have more than once seen the aorta remarkably contracted at this spot. A narrowing of this kind is well displayed in a specimen placed by Mr. W. Muriel in the Museum at Guy's Hospital, and represented in the Guy's Hospital Reports, (Vol. vii. p. 453.) A similar contraction will also be observed in the heart represented by Mr. Barrett (fig. 5 of this chapter). Otto also found the left carotid, subclavian, and descending aorta, similarly distributed in one of the hearts of a double monster of a sheep (No. 8020, Breslau Museum). Here the obstruction in the main systemic trunk must have occurred anterior to the left carotid artery.

In my previous essay on the ductus arteriosus, I have attempted to afford some explanation of the causes which determine the occurrence of arctation and obliteration of the arch of the aorta at a short distance above, opposite to, or immediately below, the insertion of the arterial duct, and shall therefore do little more at present than allude to the great pathological interest of this question*. I think it cannot be doubted

* Quoted by Hein, Paget, and Otto, from Sammlung, Chirur. Beobacht. v. ii. p. 114, 1778.

* I cannot, however, allow this opportunity to pass without replying to certain objections which have been raised by the author of a very able review of Professor Tiedemann's work on Obliteration and Closure of the Arteries in Diseases, in the Edinburgh Medical and Surgical Journal for January 1846, p. 154, against my conjecture that contraction and obliteration of the aorta may be produced by the loop formed by the pneumogastric and left recurrent laryngeal nerves which encloses the arch of the aorta, at the parts where arrangement which I am also disposed to consider as mainly instrumental in producing the natural closure of the arterial duct. In allusion to this suggestion, the reviewer observes—"But this seems inadequate to account for the occurrence, if we remember that the recurrent nerve winds round the aorta in every case, whereas this accident occurs in a very small proportion of the human species; and if we further remember that the indentation of compression is deepest in the convex part of the aorta, whereas the recurrent nerve in its enclosure of the arch touches only the concave part of the artery." In answer to the first of these objections I must state that I never attempted to maintain that the *natural* arrangement of the nervous loop around the aorta is liable to produce constriction of this vessel, although it is probable that the arrangement in question is intended to effect the obliteration of the arterial duct. In every case where obliteration of the upper part of the aorta has occurred, that vessel has been found greatly distorted in shape, and more or less changed in position; and it can be only in cases where the nervous loop compresses the aorta in an *unusual* manner (as in Romer's case, where the nerve was found within the indentation of the constricted vessel) that this singular lesion is produced. I must venture to state that the reviewer's second objection, with regard to the position at which the indentation is deepest, is decidedly erroneous. There are, undoubtedly, instances in which the indentation is principally marked at the convex part of the aorta, but there are also others where the constriction affects nearly equally the entire calibre of the vessel; this is, of course, the case in every instance of complete obliteration of the artery at this spot, and the appearance is well marked in many instances of contraction, as shown in the subjoined cut *(a)*, which is accurately copied from the plate attached to Mr. Muriel's case of contraction of the aorta near the insertion of the arterial ligament *(b)*. But there is still a third

that, in nearly the whole of the above instances, the obliteration or contraction of the aorta, and the consequent alterations in the course of the circulation, must have been established during the period of intra-uterine life.

VI.—*Non-obliteration of the arterial duct in cases where the foramen ovale is closed previously to birth.*

In the few cases where the occurrence of obliteration of the foramen ovale previously to birth has been satisfactorily proved, the children have not, I believe, survived longer than from twenty to thirty hours. Still, in such instances it appears to have been evident, from the peculiar condition of the

FIG. 7.



class of instances, in which the constriction is *wholly confined to the spot at which the recurrent nerve winds around the concavity of the aorta.* This is remarkably well displayed in a specimen preserved in Guy's Museum, and numbered 1450 ⁷⁰. It may be argued that the nervous loop is *immediately* in contact with the aorta only at its concave part,—but the nerve so closely surrounds at least two-thirds of the circumference of the vessel as to render it clear that slight alteration in the relative position of the parts would readily bring the nervous loop into too intimate contact with either of those portions of the cylinder at which permanent constriction is liable to occur. I have, however, never ventured to advance my view of this subject otherwise than as an opinion, which, although founded upon a considerable number of remarkably corroborative anatomical facts, is as yet by no means absolutely proved. Still, I believe that its probability is not in any way invalidated by the above objections. I must again draw attention to the remarkable arrangement of the nervous loop around the obliterated branchial canal, represented in Fig. 3 of this chapter, as corroborative of my view respecting the influence of the recurrent nerve in closing the arterial duct.

cavities of the heart, that the open state in which the arterial duct remained was essentially necessary for the due maintenance of the circulation, not only before but subsequently to the commencement of respiration. This was unquestionably the case in a most remarkable specimen of the kind which I have been kindly permitted to examine by my friend Mr. Ebenezer Smith; here the closure, *ante partum*, of the foramen was associated with an extreme degree of contraction, almost amounting to obliteration, of the left cavities of the heart, a condition which rendered the existence of a free communication through the ductus arteriosus absolutely indispensable.

Vieussens relates* the case of a male child which, at its birth, appeared well nourished, and of good shape, but its respiration was always difficult, its voice hoarse and feeble, and the entire surface of its body constantly of a leaden colour. Its extremities were always cold. It survived its birth only about thirty hours, every means which was adopted to facilitate the circulation having proved ineffectual. The lungs were found to be remarkably swollen and congested, but not inflamed; the pulmonary blood-vessels were much dilated, and the pleural cavities contained a large serous effusion. The right ventricle of the heart was much enlarged, and the *pulmonary artery* extraordinarily dilated. There was no *foramen ovale*. "Thus," Vieussens remarks, "the blood had not passed with freedom from the right cavities of the heart into the left, and the left ventricle was unable to propel it to the surface and extreme parts in sufficient quantity, or with sufficient force, to maintain the natural warmth and colour of the body." No direct mention is made of the state of the *arterial duct* and left cavities of the heart in this case, but it appears probable, from the above description, that the former was pervious, and that the latter were deficient both in capacity and muscular power. In a case of this kind alluded to by Dr. Williams†, there existed an unusual communication between the vena cava and pulmonary veins.

Cases of this description are of the highest importance in a medico-legal point of view, as they fully disprove the opinion maintained by many anatomists, that obliteration of the foramen ovale must be received as a certain evidence that respiration has been established. It is assuredly impossible to deny that in the heart of a child which has died within the uterus, and has been expelled in a putrid condition, the foramen ovale may be found completely and permanently closed.

* Traité de la Structure du Cœur, Chap. viii. p. 33.

† Cyclopædia of Practical Medicine, vol. iii. p. 67.

In such cases as these, it would, however, probably be always possible to determine, by an examination of the heart and its appendages, that the closure of the foramen had occurred at some period or other antecedent to birth.

The above cases complete the series of instances in which non-obliteration of the arterial duct is found to exist in conjunction with some obvious cause of mechanical obstruction to the circulation either in the pulmonary or systemic vessels. A variety of examples in which an open state of the duct was observed independently of any evident cause of impediment to the circulation, will be adduced in the next section.

The cases of permanence of the canalis arteriosus which now claim our attention are less complete, and are therefore, in some degree, less important in a pathological point of view, than those which were discussed in the preceding section* :—they certainly tend to substantiate the negative statement that the duct may remain unclosed independently of any very distinct cause of obstruction to the circulation ;—still, most of these cases appear to be defective in several leading particulars, and it is highly probable that in the larger proportion of their number, if not in all, the non-closure of the vessel has been due to the existence of a serious impediment in some part of the vascular circle, which impediment may, in some instances, have ceased to act after a permanent communication between the pulmonary and systemic trunks has been established ; while in others it has continued in operation throughout the life of the individual, although its existence has subsequently escaped the scrutiny of the morbid anatomist. This conjecture is rendered probable by the fact, that, in some of the following examples, the patients appeared to be altogether free from the usual symptoms of the blue disease, while in the others the intense lividity of surface, and severe thoracic oppression from which the individuals suffered, rendered it evident that they were the subjects of some kind of permanent cardiac obstruction.

Permanence of the duct associated with an incomplete state of the ventricular septum.

In this class of instances it appears to be almost a matter of certainty that the cardiac and arterial defects must have been due to the existence both before and after birth of some serious impediment to the circulation, which either ceased to operate as age advanced, or remained undetected after death, by those who observed the cases : the scantiness of the following histories renders the latter idea by no means improbable.

Richerand* relates the case of a man who presented himself at one of the Parisian hospitals to be operated on for stone in the bladder. His complexion was remarkably livid, his respiration laborious, and his pulse irregular. He could not utter two words in succession without taking breath, was obliged to sleep in a sitting posture, and was particularly remarkable for his indolence. This indolence, joined to the great simplicity of his nature, was such that he had never been able to maintain himself. A small bleeding from the arm relieved his pains, but the difficulty of breathing increased, and he died from suffocation.

The heart was found to be full of blood. The right auricle was principally dilated ; the ventricles were nearly equal in the thickness and capacity of their walls. The *ventricular septum* was perforated by an aperture about half an inch in extent, and directed obliquely from below upwards, from before backwards, and from right to left, so that not only the direction of the opening, but likewise a kind of valve, formed in the right ventricle by a fleshy column so placed as to prevent the return of the blood into the right ventricle, clearly showed that the blood flowed from the left into the right ventricle, and thence into the *pulmonary artery*. This vessel was aneurismally distended up to its division. The *ductus arteriosus* was an inch in length, and large enough to admit a goose-quill, allowing a free passage of the blood from the pulmonary artery into the aorta. The *foramen ovale* was closed.

The long existence of symptoms in the above case renders it evident that the cyanosis and other evidences of delay to the circulation were not due, as frequently happens in cases of congenital heart disease, to the recent superaddition either of pulmonary obstruction or of endocardial or valvular lesions. It is to be regretted that the condition of the lungs and of the ascending aorta was not observed, as there had in all probability always existed some severe cause of impediment in both. There must have been a greater amount of impediment on the left side than on the right, but the small diameter of the arterial duct renders it probable that the aorta was not completely obliterated.

* *Erratum.*—In the last section, note to page 966, last line of first column, and first line of second column, *for* "the loop formed by the pneumogastric and left recurrent laryngeal nerves which encloses the arch of the aorta, at the parts where arrangement which," &c., *read*, "the loop formed by the pneumogastric and left recurrent laryngeal nerves which encloses the arch of the aorta at the parts where the vessel is liable to become constricted, an arrangement which," &c.

* Nouveaux Elémens de Phys., Paris, 1811, t. i. p. 295.

Sandifort speaks of a case in which the *arterial duct* was present, but not of so large a size as it naturally is, and the aorta arose equally from both ventricles.*

In the case (cited by M. Louis) of a man, ætat. 40, who had been the subject of a certain degree of cyanosis, the *pulmonary artery* was found to be dilated, and the *arterial duct* was sufficiently wide to admit a large goose-quill. The ventricles communicated by a large opening in the *septum*.

The preparation numbered 1382[16] in Guy's Museum presents a very remarkable modification of this description of congenital fault. It is the heart of a child between 3 or 4 years of age, who was under the care of my friend Dr. Lloyd suffering from hooping-cough. Dr. L. was informed that the child had always been liable to occasional lividity of the face and nails ; but, during his attendance, he had never perceived any appearance of cyanosis, until on one occasion, when he found the little patient in a violent paroxysm, with its face extremely blue and congested : the child was placed in a bath, and there expired. The right auricle is of large size, the right ventricle is also large, and its walls are rather hypertrophic. The right auricle presents an abnormal oval aperture, which is situated just above the left margin of the right auriculo-ventricular opening, partially separating the anterior tricuspid curtain from its upper attachment. This aperture extends into the right ventricle, and is continued through the upper and back part of the *ventricular septum* into the left ventricle, where it opens by an oval orifice, about three lines in diameter, in the tendinous spot, which is situated just below the aortic valves. In this manner there exists an abnormal communication between the right auricle and the right and left ventricles. The *pulmonary artery* is wide, its left branch (the right has not been preserved) remarkably so. The pulmonary valves are healthy. The *arterial duct* is open, but is barely capable of admitting a common probe. The *foramen ovale* is perfectly closed. The left auricle is rather contracted. The left ventricle is of fair size, but not apparently either hypertrophied or dilated. The aortic valves are quite healthy ; the ascending aorta is rather dilated ; portions of its lining membrane are somewhat diseased. As mentioned in the catalogue, *the descending aorta becomes remarkably small immediately distal to the left subclavian artery*, but without presenting any appearance of stricture, or the slightest trace of morbid alteration in its tissues.

It may perhaps be considered that in some of the above cases the permanence of the arterial duct was owing to the closure of the foramen ovale previously to birth, but it appears to be highly improbable that so unusual a change as the too early occlusion of the foramen could be effected in a case where obstruction to the circulation had already prevented the completion of the ventricular septum.

Patency of the duct associated with an unclosed state of the foramen ovale.

It appears that in most of the cases where the fœtal condition of the heart has become permanent, the individuals have suffered very severely from all the worst symptoms of cardiac obstruction. The details of the cases unfortunately do not furnish us with any distinct clue either to the causes which determined the non-closure of the passages, or to those which have subsequently given rise to the symptoms of morbus cæruleus. And it is, of course, unnecessary to attempt an hypothetical explanation of a point which appears to admit of being demonstrately elucidated by the careful examination of future cases of this description.*

M. Jurine observed the case of a child which suffered from the usual symptoms of blue disease, and died at the age of 6 months. The left auricle was very capacious ; the *foramen ovale* was still pervious, as was also the *arterial canal*, which was, however, sensibly diminished in calibre.†

Dr. Farre and Mr. English relate the case of a female child which lived 18 days. Nothing remarkable was observed at her birth, but she cried faintly, and seemed rather weak. Her skin and eyes were somewhat yellow. There was an occasional threatening of suffocation when she sucked ; unusual quietness and perpetual drowsiness were remarked during the first week ; and in the course of the second week she had fits of crying, during which the respiration became affected in a remarkable manner. On the 14th night, the fit of crying was more severe than on any former occasion, and for several moments the breathing was suspended, the lips becoming black, when a strong convulsive effort, with a deep sigh, restored animation. On the 18th day, the child had a violent paroxysm, in which the breathing was frequently suspended for more than a minute ; the lips were black, and the eyes fixed. At 8 o'clock she seemed to be dying : the extremities were cold ; the coun-

* Observationes Anatomico-Pathologicæ, 1779.

* In instances of this kind it appears to be absolutely necessary to ascertain not only the precise condition of the cavities and orifices of the heart, and of its immediate appendages, but also of the pulmonary vessels, and of the lungs and thoracic cavities and organs generally, as well as of the entire track of the aorta, the large systemic vessels, and principal abdominal viscera.

† Mém. de la Soc. Royale de Med. t. x. p. 52, and quoted by Gintrac.

tenance cadaverous; the lips black; the breathing performed by short convulsive inspirations and long groaning expirations, with now and then a sigh. On putting her into a warm bath, it was surprising to see the improved state of the respiration, the expirations being assisted by gentle pressure on the abdomen and ribs. This treatment having been continued for half an hour, she was wrapped in warm flannel, and seemed to sleep for an hour, when the breathing gradually got worse, and all the former train of symptoms returned. The warm bath was again resorted to, with the same benefit as before; but, on taking the child out of the bath, she soon ceased to breathe if left to her own efforts. Mr. English therefore kept his hands constantly on the thorax and abdomen, and, by assisting the expirations, prolonged her life about two hours. For ten minutes together she sometimes lay without the least appearance of life, when a strong convulsive action of all the muscles of the thorax and abdomen, with a deep sigh and several catching sobs, renewed the circulation. Then the colour would return to her lips; she would stretch her limbs and open her eyes. The last struggle of this kind took place a quarter of an hour after he supposed she was dead. He never could feel the pulsations of the heart, although he frequently examined the region of that organ. He once observed that the pulsations of the radial arteries were synchronous. The heart was found to be of proper size and figure, its respective cavities being only proportioned to each other; but the valve of the *foramen ovale* was so very imperfect that a free communication existed between the auricles. The *ductus arteriosus* was open, and larger than natural. The *pulmonary artery* was proportionably larger, but its right and left branches were of their proper size. The liquor pericardii was increased in quantity; the other cavities of the chest were free from serum, and the appearance of the lungs was natural. The liver was overcharged with blood; its gall-bladder and ducts preternaturally contracted. (The skin and eyes continued to be very yellow up to the time of the child's death, and the fæces were deficient in bile).

It is most probable that in this case the symptoms were not by any means due to the open state of the foramen ovale and arterial duct, but were owing to some undiscovered impediment either in the pulmonary or systemic vessels which in the first place occasioned the non-closure of the fœtal canals, and subsequently gave rise to the extreme embarrassment to the cardiac movements.

Mr. J. Hume Spry details* the case of a female, ætat. 17 years, who had neve menstruated except once, a short period before her death. She had always from her birth been remarkably dark-coloured, and the discolouration rapidly increased as she advanced to riper years. The colour of her face was said to have been always blue, and her tongue was very blue. She had continually a very acute throbbing pain in her head, confined principally to the forehead and temples. She had always felt a great pain and sense of weight in her left side, attended with constant palpitations of the heart. Respiration was performed with difficulty, and she had sometimes a very slight cough. She was very indolent; and upon any unusual exertion she was in danger of fainting. Some years previously to her decease she had small-pox so severely as to be in immediate danger. The body was found to be greatly emaciated. The venous system was preternaturally loaded with blood; the pericardium adhered very firmly to the pleura costalis, but did not contain any fluid. The heart was remarkable for its great size and weight, and was distended with an enormous quantity of dark fluid blood; the *foramen ovale* was pervious, and nearly two inches in circumference; the *canalis arteriosus* was open, but its size is not mentioned. The lining of the heart was white, and appeared to be rather thickened. The author made a particular examination of the lungs, having understood that in cases where the lungs have been much obstructed the foramen ovale has been found pervious; but the lungs of this woman were evidently free from any disease. Mr. Spry considered that the appearance of the heart probably did not depend upon obstructive or other disease of those organs.

Dr. Burns has given the following narrative*:—

J. M. was to appearance, when born, a healthy and robust child. Symptoms of disease commenced at the approach of his third year, and increased until his death. He was at first liable to be seized, after any over-exertion, with spasmodic twitches in his muscles, accompanied with urgent cough and dyspnœa; and occasionally, during these paroxysms, the colour of his skin changed to a faint purple hue. Cessation from exertions usually proved sufficient to restore the functions to their natural state. He passed through the usual infantile diseases without material alteration in his symptoms, which remained with little change for forty or forty-two years. During the whole of this period he had been able to follow his usual employment, which was that of a painter; but latterly he acquired a tor-

* Memoirs of the Medical Society of London, Vol. vi. p. 17.

* Observations on some of the most frequent and important Diseases of the Heart, &c. p. 17.

pid and unhealthy appearance. The symptoms increased in urgency; the paroxysms recurred more frequently than formerly, and were excited by the most trivial causes; the countenance was permanently bloated and purplish; the oppression and anxiety in the chest became intolerable; general œdema supervened; the body was almost incessantly bedewed with a cold and clammy sweat; the appetite failed; the pulse became small, feeble, and hardly perceptible; the debility was extreme. The anxiety and depression rapidly increased, until death put an end to his sufferings. On examination, the thorax was found to contain about a pound of aqueous fluid, of a yellowish tinge. The lungs were every where sound, and free from adhesion. The pericardium contained about two ounces of darker fluid than that effused into the chest. The *ductus arteriosus* and *foramen ovale* were open; the former equalling in size a large crow-quill; the latter the circumference of the barrel of a goose-quill. The brain and abdominal viscera were perfectly healthy; but the veins of the testicles and urinary organs were varicose and turgid with blood.

Although no allusion is made to the form and capacity of the chest in either of these cases, it seems to be very evident that, in the three latter examples, the original and principal obstruction to the circulation did not exist in the pulmonary apparatus, but that the chief obstacle lay in some part of the systemic circulation. These cases are almost rendered valueless by the silence of their authors respecting the condition of the aorta, as there appears to be strong reason to believe that there existed some considerable impediment to the flow of blood through that vessel in at least three of the instances. The details of M. Jurine's case are too slight to furnish any clue to the detection of the origiual cause of obstruction.

The following is an abstract of the case of a person whose body was inspected by Mr. Molloy and myself at Guy's Hospital:—

A female, ætat. 32, died of phthisis in Oct. 1837. She was the mother of eight children, five of whom were still living and healthy. Her constitution had never been robust, but she had not been the subject of any severe illness until the preceding winter, when she had a pulmonary attack, which, however, left her in the following spring. Her phthisical symptoms were of three months' duration. She was not supposed to have been the subject of cyanosis, although her face was rather livid during the last fortnight of her life. On inspection, the lungs were found to present extensive traces of phthisical disease. The heart was small and pale; its right cavities were little distended. The *foramen ovale* was sufficiently wide to admit the little finger. It was partially guarded by a wide and thin valve, which besides its usual connections on the left side, was also attached to the walls of the right auricle above and below by four or five short and slender cords; it appeared to have allowed the passage of blood from the right to the left auricle. The *pulmonary artery* was not diseased, but the *arterial duct* was sufficiently open to permit a large-sized probe to be passed from the aorta into the pulmonary artery; but on the pulmonary side its orifice was guarded by a small nipple-like process, resembling in form the pointed vegetations sometimes noticed upon the aortic valves; the apex of this was fissured, allowing the passage of blood or of an instrument from the aorta, but apparently acting as a complete valve in the contrary direction. The aortic orifice of the duct was wide, and surrounded by linear specks of white deposit. The left auricle and bicuspid valve were healthy. The walls of the left ventricle were rather hypertrophic and were well contracted. The aorta was perhaps slightly dilated, but it was otherwise healthy. The liver, spleen, and kidneys, were enlarged, and considerably congested. The heart with its appendages is preserved in the museum at Guy's Hospital, numbered 1383[50].

In this case the condition of the heart was such as to convince me that, although the non-closure of the foramen ovale and arterial duct was possibly determined by the existence of some cause of obstruction to the circulation subsequently to birth, that cause had long since ceased to operate. The fact that blood could pass through the duct only from the aorta to the pulmonary artery, and not in a contrary direction, was sufficient proof that the permanence of the canal had not been maintained by that diseased condition of the lungs which subsequently proved fatal; and the entire absence of cardiac symptoms, together with the generally healthy appearance which the tissues of the heart and its appendages presented, were convincing evidences that the organ had not been compelled, throughout the life of this individual, to struggle against the resistance of any permanent impediment to the circulation.

Patency of the arterial duct unassociated with any other defect in the heart or its appendages.

The appearances presented by a preparation numbered 1383[50] in Guy's Museum appear to me to have been due to causes similar to those which probably determined the permanency of the duct in the case last cited; but, as the preparation merely consists of a portion of the aorta and pulmonary artery, and as no history of the case has been preserved, it is of course uncertain whether it properly belongs to this class at

all. In that specimen the *ductus arteriosus* is merely sufficiently open to admit a very small probe. The tissues of the pulmonary artery, and of the aorta, are entirely free from the slightest appearance of dilatation, or of any other change. The parts are apparently those of a child 8 or 10 years of age. I believe that, in this as well as in the preceding case, the simply open state of the arterial duct was probably due to partial failure in the process by which the canal is naturally closed. The true nature of such cases must, therefore, remain unexplained until the causes which produce the obliteration of the duct have been clearly demonstrated.

The following case, by Professor Huss, of Stockholm, would, at first sight, appear to be one in which patency of the duct existed independently of any other congenital defect in the heart or large vessels; but I am inclined to believe that the lesion was of a far more complicated nature.

The subject of this case, a youth 19 years of age, had generally enjoyed good health: he had, however, been affected with palpitations of the heart after exercise. His skin had never been blue; on the contrary, it was always white and transparent. Five months before his entrance into the hospital, he had been affected with severe articular rheumatism, followed by palpitations, difficulty of respiration, cough, epistaxis, permanent tinnitus aurium, and gradual prostration of strength. After his entrance, he continued to sink, and, at the end of six weeks, expired, without his skin ever having assumed the least blue colour. On examination, the left ventricle was found of an oval shape, enlarged so as to be able to hold a goose's egg; its parietes were an inch in thickness. The aortic valves were corrugated, and studded with osseous concretions; two of these valves had each a perforation as large as a pea. In one of the sinuses of Valsalva there was a small aneurism. The descending aorta was dilated, and formed at the commencement of its arch a sac as large as a pigeon's egg, without there being any lesion of its internal membrane. Two inches three lines from the orifice of the *pulmonary artery* there was a direct communication between its left branch and the aorta, two lines broad and six lines long, so that the ring finger could be passed through it. It terminated in the aorta, just below the origin of the subclavian and carotid arteries*.

This case bears so remarkably close a resemblance in its leading details to one which occurred within my own observation, some years since, at Guy's Hospital, in which patency of the arterial duct, and the subsequent formation of an aneurism of the descending portion of the aortic arch, were evidently due to a strictured condition of the aorta distal to the left subclavian artery, that I am disposed to attribute the occurrence of similar lesions in this case to a like cause of impediment to the systemic circulation, which may have escaped the notice of Professor Huss. At all events, a comparison of this instance with my own case, which is given in the subjoined note*, may not be altogether uninteresting.

The details of most of the above cases are, upon the whole, by no means satisfactory, as there is reason to believe that many of them are deficient in some of their most important particulars; and it is probable that, had we been furnished with the entire histories, several of these examples would have required a different classification from that which I have now given them. Still, none of these cases are deficient in pathological interest, and more than one of them must be received as convincing evidence of the fact, that patency of the arterial duct may be found in cases where there does not exist any discoverable cause of permanent obstruction to the circulation.

* Gazette Méd. de Paris, February 11, 1843; as quoted in the American Journal of Medical Sciences.

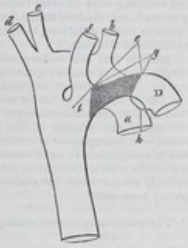
* The preparation numbered 1383*o in the collection at Guy's is taken from a man, æt. 21, who died of pneumonia, which was attended with some lividity of the face, but not with sufficient discolouration to lead to a suspicion that he was the subject of any cardiac disease. On inspection, the lungs were found to present extensive traces of recent pneumonia. The right ventricle of the heart was strong, and not particularly dilated; there was considerable thickening of the valves of the *pulmonary artery*, and small concretions adhered to their corpora Arantii. The trunk of the pulmonary artery was greatly dilated; its tissues were so much thickened that it no longer presented its usual structural appearance, but more closely resembled the systemic trunk: portions of its interior were raised by interstitial deposit, and, at one spot, its lining membrane was rendered irregular by vegetations. The pulmonary artery was adherent to the aorta at the spot where the arterial ligament usually exists, and there the two vessels communicated by a rather irregular circular opening, about two lines in diameter, (a far smaller aperture than that which existed in M. Hasse's case). Immediately above this opening, the aorta was remarkably contracted by a narrow and defined stricture. An aneurismal dilatation, about the size of a walnut, existed in the most prominent part of the descending arch of the aorta, nearly opposite to the opening of the duct. There were only two aortic valves: these were greatly thickened, and some irregular masses of vegetations adhered to their ventricular surfaces. It is a remarkable fact that the aorta of this man's brother, who died in the hospital some months afterwards, was found to be furnished with only two semilunar valves. The left ventricle was hypertrophied and dilated. An inspection of this interesting preparation will show that, although no distinct remains of an arterial canal exist, the communication between the arteries is not of an aneurismal kind. Some doubts may be entertained upon this point in M. Huss' case; but it appears, from the description, that there also the abnormal opening was distinct from the aneurismal pouch which existed in the descending portion of the arch of the aorta.

The left subclavian artery originating from the arterial duct.

A remarkable case in which the malformation was considered to exist, is related by J. T. Klinkosch*.

A woman, 32 years of age, who had been sterile for ten years, gave birth to her first child, a boy, which was living and well nourished, but it was the subject of great malformation of the head and face, and died in half an hour after its birth. It was found that the arch of the aorta, (*a*) arising from the left ventricle, passed to the right, and gave off three distinct branches, first the left carotid (*b*), then the right carotid (*c*), and afterwards the right subclavian (*d*). The *pulmonary artery* (D), arising in the usual manner, passed backwards and to the left, and then separated into its two main branches and the *canalis arteriosus* (*e*). This latter canal joined the descending aorta at the distance of four lines beneath the origin of the right subclavian artery; and, from the duct at about one-third of its length from the aorta, the left subclavian artery (*f*) took its origin.

FIG. 1.



I think that, upon inspection of the subjoined figure, which is a correct diagram from Klinkosch's plate, it will be perceived that this malformation differs in no very material degree from that already alluded to at page 963, note 3 (case by Fiorati and Sandifort), in which the left subclavian artery arises from a kind of pouch, situated at the commencement of the descending aorta, which pouch is joined below by the arterial or branchial canal. The portion of vessel (*g*) which is situated between the pulmonary branch (*h*) and the left subclavian artery (*f*) appears to me to represent the entire extent of the arterial duct, while the portion of the canal (*i*) from which the left subclavian arises seems to be merely prolonged from the aorta, and not to be in reality a portion of the arterial canal.

* In Dissertat. Med. Select. Pragensis, vol. i. Anat. Partus Capite Monstroso, p. 190, et Tab. x, figs. 4 and 5.

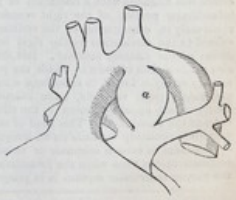
Aneurism of the arterial duct.

The only fully detailed instance of this remarkable lesion with which I am acquainted, is that related by M. Billard*.

In 1826, a male infant, two days old, was brought to the hospital of the Trouves. It was of middle size, its respiration was difficult, its face livid, and its cry stifled. The temperature of its body was natural, the pulse small, frequent, and easily compressed. The child remained two days in this state, and died on the third. On inspection, the stomach and intestinal canal were found remarkably congested, the liver was gorged with blood; both lungs were also engorged. The heart was more voluminous than is usual in infants at birth; the cavities on either side were nearly equally dilated, and were filled with dark blood. The arterial canal (*a*) presented the form of a large cherry-stone; its transverse diameter was about three and a half lines, and its circumference nine lines. Externally it had the appearance of communicating freely with the aorta, but this was not in reality the case, as its interior was filled with organised clots, disposed in layers such as are observed in the aneurismal tumors of adults, merely leaving in the centre a passage which would barely admit a crow-quill. The other organs presented no particular alteration.

M. Billard also makes slight reference to another case of aneurism of the duct which was related to him by M. Baron, and in which the symptoms were less marked than in the above instance.

FIG. 2.



* Traité des Maladies des Enfans Nouveaux-Nés et à la Mamelle, p. 567, Paris 1828, and Atlas, Planche viii. fig. 1.

It is probable that, in M. Billard's case, the symptoms were chiefly due to the dilated and weakened condition of the heart, consequent upon the great obstruction to the circulation which the disease in the arterial canal must have produced during intrauterine life. Still it cannot be denied, that the too early closure or permanent contraction of the arterial canal would be liable to produce very serious embarrassment to the lungs and right ventricle.

MALPOSITION OF THE PULMONARY ARTERY.

There are several species of congenital malformation of the heart in which the principal error is found to consist in an unusual mode of origin of one or both of the great arterial trunks. Thus (1), the pulmonary artery has been found to take its origin from a small supplementary right ventricle. (2), both aorta and pulmonary artery may arise in a similar manner from a kind of third ventricle. (3), the artery which supplies the lungs may arise as a branch from the aorta. (4), the aorta and pulmonary artery occasionally arise from the right ventricle; and (5), in other cases, they proceed from the left ventricle*. (6), the aorta may be situated over an opening in the *septum ventriculare*, while the pulmonary artery arises from the left ventricle. (7), the position of the ventricles, and of their corresponding vessels, is sometimes found to be transposed; and, lastly (8), the pulmonary artery may originate from the systemic ventricle, while the aorta arises from the pulmonary ventricle. To render the nature of these malformations sufficiently clear it will be necessary to consider each species briefly in detail.

(I.) *The pulmonary artery originating from an additional right ventricle.*

A description has already been given of cases in which contraction of the pulmonary orifice was attended with a dilatation of the infundibular portion of the right ventricle, apparently in consequence of the resistance offered to the circulation by the rigid condition of the diseased ostium†. But there also exists a class of cases in which the pulmonary artery is found to arise from a supplementary right ventricle, quite independently of any apparent lesion in the structures of the vessel itself. This kind of malformation probably depends on an irregular development of carneæ columnæ or musculi pectinati at the time when the formation of the proper ventricular septum is in progress (as illustrated in cases by Dr. T. Thompson and Dr. Fletcher, p. 833). An instance of this malformation, observed by Mr. Holmsted, has been cited at p. 700, as illustrative of the causes which may prevent the free entrance of blood to the pulmonary artery. The following is an example of a very similar malformation:—

Dr. Farre describes the preparation of a heart in the museum of St. Bartholomew's Hospital, in which the right and left ventricles are equal in muscular substance, and the valves natural. The aorta, which is of its natural size, arises over the *septum of the ventricles*, opening equally into both ventricles. Two apertures in the right ventricle communicate very freely with a very small third ventricle, from which the *pulmonary artery*, correctly formed, and of its usual size, arises. The *auricular septum* is perfect. From the appearance of the heart, Dr. Farre considered that the age of the subject exceeded 14 years.

(II.) *The pulmonary artery and aorta originating from a supernumerary ventricle.*

Mr. J. Bell (cited by Dr. Burns) has given an account of a heart in which "both the *pulmonary artery* and the aorta arose from a middle or third ventricle. The right and left ventricle were in every respect correctly formed, and received their blood from their auricles in the usual way, but delivered it into the third ventricle."

It is possible that this malformation may have depended upon some irregularity in the process of development of the ventricular septum, but it appears to be by no means improbable that the supernumerary cavity was in reality the remains of the *bulbus arteriosus*—an arrangement of parts which appears to compensate in some measure for a defect in the ventricular septum, but which is invariably absent where the ventricles are completely separated.

(III.) *The pulmonary artery arising from the aorta.*

Instances of several varieties of this species of congenital defect have been cited at the commencement of this paper*, but, as malposition of the artery which supplies the lungs is one of the most characteristic features of a very rare and interesting kind of cardiac malformation, I cannot omit the following remarkable example:—

Dr. Carson relates† the case of a child which was well developed at its birth, and which, for two or three days afterwards, appeared to be in perfect health, with a

* Allusion has already been made to those cases in which the pulmonary artery takes its origin from both ventricles.
† Cases by Dr. Crampton (p. 457), and Drs. Aran and H. Bennett (p. 699).

* Page 276, et seq.
† Edinburgh Medical and Surgical Journal, Vol. lxii. p. 134.

natural colour of the skin. On the third day it was unusually soporose, sighing frequently, and the respiration was more hurried than usual; it also ceased to take the breast, and Dr. C. remarked an unusual dark colour of the skin. On the fourth day it appeared to be in a profound sleep, breathing rather hurriedly, and frequently sighing deeply. There was no fever, nor any remarkable diminution of temperature. On applying the ear to the chest, the heart's sounds were found to be rather hurried, but their succession was regular: the whole body was of a dusky hue, resembling that of a dark-complexioned person. The child died on the fifth day. Slight effusion was found in the pericardium; there was one large artery given off from the heart, and from this the *pulmonary artery* sprung. The thus-united aorta and pulmonary artery was considered to be larger than the aorta of so young a child. It proceeded from a large ventricle, which appeared, at first sight, to constitute the whole of the ventricular portion of the heart. The systemo-pulmonary artery was separated from the ventricle by semilunar valve (valves?), and from a very large auricle by a tricuspid valve. Into this auricle opened, by three orifices, three pulmonary veins. The size of the two venæ cavæ led Dr. C. to think that one of these three veins might be systemic. The large auricle communicated, by a small opening, with another auricle about the size of a small horse-bean, into which entered the two venæ cavæ, each not above two lines in diameter. This small auricle communicated with a cavity of the size of a swan-shot in a fatty muscular mass upon the side of the large ventricle, constituting with it the whole ventricular mass, and evidently being the representative of the right ventricle. This and the right auricle were separated from each other by small cobweb-like membranes representing the auriculo-ventricular valve.

It is probable that the vessel which supplied the lungs in this case was not a true pulmonary artery, but was in reality the ductus arteriosus. The heart appears to have presented an extreme degree of that kind of malformation in which, obliteration of the ascending pulmonary artery occurring subsequently to the completion of the ventricular septum, the right ventricle, being no longer required to propel blood to the lungs, becomes extremely contracted, and the ductus arteriosus performs the office of the pulmonary artery*.

(IV.) *The aorta and pulmonary artery arising from the right ventricle.*

In the early part of this essay a description was given of those cases in which the pulmonary artery is found occupying its natural position, but is either obliterated or greatly contracted at its origin, and in which the aorta also arises from the right ventricle; in addition to these, there appears to be a class of cases in which, independently of any remarkable contraction of either vessel, the aorta and pulmonary artery originate from the right ventricle.

Dr. Paget* refers to a case of this kind, and quotes a similar example related in the Arch. Gén. de Médecine, T. xxiii. In a case which has been briefly related by Dr. F. Ramsbotham†, both the aorta and the *pulmonary artery* arose from the right ventricle, there being a communication between the ventricles by an *orifice in the septum*. There was no appearance of lividity in the child, except that the lips were usually pale, and the fingers occasionally appeared of a blueish-grey colour. The child died suddenly at 10 years of age. She was healthy until the age of 2 years, when exertion caused some shortness of breath. She was of a lively, placid disposition; the pulse was rapid and small; the pulsations in the carotids could be distinctly observed. She lay on her back, or on the left side, with her legs drawn up to the body, and the back bent to a semicircle. The appetite and general health were good. The sensation to the hand applied over the heart was that of a sawing, and to the ear that of the purring of a cat. Twelve months before her death she became much emaciated. The appetite was greater than natural; the skin dry; the heart laboured in its action; and the respiration was much hurried, and was attended with a short irritating cough, without expectoration. She was (latterly?) unable to lie down. At length the face and lower extremities became anasarcous. She expired suddenly, jumping up as if she had been frightened.

(V.) *The pulmonary artery and aorta arising from the left ventricle.*

There appear to be three cases of this description on record, but I have only been able to meet with the following example‡:—

M. Maréchal and M. Carré examined the body of an infant 3 months old which died with all the symptoms of the blue disease. When the child was six weeks old, its lower extremities and abdominal parietes became œdematous—a condition which twice

* See the case by Dr. W. Hunter, and other instances, cited at p. 280.

* Op. citat. p. 294.
† Med. and Physical Journal, Vol. lxvi. p. 548.
‡ In addition to M. Maréchal's case, Otto refers to an instance related by K. Hoffman, in the New York Medical and Physical Journal; and also to one by M. Mery, in the Mémoires de Paris, 1700; but I have in vain sought for the latter case in the Memoirs of the Academy of that period. Dr. Paget also makes brief allusion (op. citat. p. 294) to an instance of the kind.

yielded to the use of tincture of digitalis. It was found, on examination, that the peritoneum did not contain any fluid; the liver was larger than natural; the pleural and pericardial cavities contained a notable quantity of yellow serosity; the heart, especially its auricles, was, with its vessels, distended with dark blood. The aorta and *pulmonary* artery appeared to originate from the same cavity. The latter divided into two branches, which supplied the right and left lung; the *ductus arteriosus*, which arose from the left branch, was sufficiently open to admit an ordinary probe. The left ventricle presented at its base the orifice of the aorta, furnished with sygmoid valves; and, to its right, at the distance of three lines, that of the *pulmonary artery*, which was destitute of valves. Below the aorta, an auriculo-ventricular valve, divided into two portions, formed a canal capable of admitting the end of the fore finger, communicating with the left auricle, which formed a common cavity with the right auricle. Below the orifice of the pulmonary artery there was an opening about three lines in diameter, furnished with a kind of valve formed by a fold of endocardium: this opening communicated with the auricular cavity, and, at its upper part, was partially separated by a thick prominence from the orifice of another cavity of small size, which was the rudiment of the right ventricle.*

(VI.) *The aorta arising from both ventricles, the pulmonary artery from the left.*

The malformation described in the following case appears to form an intermediate link between that in which both arteries arise from the left ventricle, and that in which the aorta arises from the right ventricle and the pulmonary artery from the left:—

Dr. Wilmer Worthington relates the case of a female child which had from birth been liable to almost daily attacks of oppressed breathing, attended with blueness of the skin and nails. Some time before its death, which occurred at the age of 22 months, it began to emaciate, and had occasional cough and diarrhœa. On examination, the right auricle was found to be very small, and it exhibited no appearance of musculi pectinati. The pulmonary veins opened into this cavity, and the auricle appeared to be merely a dilatation of these vessels. There was no opening between this part of the heart and the right ventricle. The *foramen ovale* remained open, so that the blood from the lungs passed directly through this opening into the left auricle. This cavity was larger than natural, and presented the usual appearance of musculi pectinati usually found in the right auricle. The ostium venosum existed on the left side of the heart, with its usual valves. The ventricles were of the ordinary size and thickness. Their interior structure presented the usual appearance. The aorta arose from the right and left ventricle by a spreading mouth, which formed a communication with both these cavities, the *ventricular septum* being deficient immediately under the mouth of the aorta. The aortic valves were wanting. The *pulmonary artery* arose from the left ventricle, at a short distance from the aorta. No *ductus arteriosus* existed, this passage, as Dr. Worthington remarks, being unnecessary on account of the pulmonary artery and aorta proceeding from the same cavity.*

It is probable that the primary lesion, in this case, was obliteration of the right auriculo-ventricular opening. In several particulars this instance bears an interesting resemblance to one related by Dr. Favell, and cited in a previous chapter.† In that example, however, the pulmonary artery arose as usual from the right ventricle.

(VII.) *Complete transposition of the cavities and vessels of the heart.*

In many cases of complete or partial transposition of the thoracic and abdominal viscera, the pulmonary cavities of the heart, with the pulmonary artery and cavæ, are found to be situated on the left side of the thorax, while the systemic auricle and ventricle and the aorta are placed on the left side. In these cases the aorta and pulmonary artery arch to the right. The following examples will sufficiently illustrate this species of malformation:—

Meckel (filius) describes and figures‡ a specimen, preserved in his father's museum, in which the base of the heart is situated to the left, and its apex to the right. The cavæ were situated on the left side; the pulmonary auricle and ventricle were also to the left. The aortic ventricle was on the right side. The *pulmonary artery* was situated anteriorly to the aorta, which arched to the right, and descended on the right side of the dorsal vertebræ. The positions of all the other thoracic and abdominal viscera were reversed.

M. Parisot, of Nancy, relates§ the case of a man, ætat. 29, in whom the heart occupied the right side of the thorax, the aortic ventricle lying to the right and the pulmonary ventricle to the left side. All the divisions of the circulating system followed the same transposition. The aorta was placed to the right, and was crossed by the *pulmonary artery*, which passed from left to right, and the thoracic and abdominal aorta descended on the right side of the vertebral column. The other viscera of the chest and abdomen were also transposed*.

It does not appear that this kind of deviation from the natural position of the several parts of the circulatory apparatus has any tendency to shorten the lives of the individuals in whom it occurs†.

This peculiarity has been observed in the bodies of adults of all ages who do not appear to have suffered from any symptoms of thoracic obstruction. Dr. P. A. Jewett relates an instance of this kind‡ in which the individual lived to the age of 65 or 70 years. In fact, the transposed viscera are usually so well-formed, and so symmetrically placed, in these cases, that the peculiarity of their situation can scarcely be regarded as a defect. There also appear to be some instances of malformation in which the cavities of the heart occupy their natural situation, while the main arteries pass over to the right side of the chest instead of to the left.

The cases of the above class are altogether distinct from those in which the pulmonary artery arises from the systemic ventricle, and the aorta from the pulmonary ventricle. The different varieties of that remarkable species of malformation will form the subject of the ensuing chapter.

Transposition of the Aorta and Pulmonary Artery.

This remarkable and very serious kind of malformation appears to depend upon some error in the process which, at an early stage of intra-uterine life, causes the separation of the arterial bulb into two distinct vessels; the primitive systemic, and pulmonary trunks. That this erroneous division of the vessel occurs previously to the development of the ventricular septum is proved by the details of the following case.§

A child, born on the 21st of November, 1842, was deposited on the same day at the Foundling Hospital in Paris. Those who had the care of it remarked that it was often the subject of great distress of breathing, with a quick and dry cough, and especially that when it drank, a state threatening suffocation ensued. The child was large, and externally well formed; but it died in 11 days, apparently of umbilical phlebitis. The heart was found to be bag-shaped, or rather rounded at the place of the apex, and to consist of only one ventricular cavity. Viewed from the front, the *pulmonary artery* was quite hidden by the aorta, which originated from the right and anterior portion of the ventricle, and by the auricular appendices, for of those there were two which were well formed, although there was only one auricle. A line directed obliquely backwards, and from right to left along the base of the ventricle, would have crossed, first, the opening of the aorta; next, that of the pulmonary artery; and, lastly, the auriculo-ventricular opening. The spleen was large, but no other viscus or portion of the circulatory apparatus presented any anomaly. The most remarkable circumstance of this case was, that no cyanosis had been noticed during life.

A variety of appearances have been found in the several instances of this kind of malformation which have been recorded. In cases of transposition of the great arteries, some of those communications which exist, during fœtal life, between the pulmonary and the systemic heart and their vessels always remain permanent;—either the ventricular septum, the foramen of Botal, or the arterial duct, is always pervious in examples of this malformation; indeed, it would, at first sight, appear impossible that, in such cases, the circulation could be in any way maintained, even for a few minutes, unless *two* of these channels remained open—one to convey a portion of the venous blood to the left, which is here the pulmonary ventricle, the other to return the arterialized fluid to the right ventricle, or to the aorta, which here arises from that cavity. There are, however, cases of this description upon record in which only *one* abnormal communication, the arterial duct or foramen ovale, was observed to be present, and still the children lived for a considerable period. It is very probable that the histories of these cases are imperfect in some of their details, and that those who have recorded them failed to observe some abnormal communication which existed either between the cavities of the heart or some of the great vessels; as, for example, one or more of the pulmonary veins may have entered the venæ cavæ, or the bronchial arteries may have been much enlarged,

* Journal Gén. de Méd. t. iii. de la 2e série, p. 354, Dec. 1819, quoted by Cintrac, p. 173.

* American Journal of the Medical Sciences, vol. xxii. p. 131.
† Page 828.
‡ De Cordis Conditionibus Abnormibus, p. 4, Tab. 1, 1802.
§ Archiv. Gén. de Méd. June 1839, as quoted in Edinburgh Medical and Surgical Journal.

* For other cases of this transposition, see Haller, de C. H. Fabrica, T. ii. Lib. iv. § 4, p. 89; and Dr. Darling, in New York Journal of Medicine, p. 378.
† A case is mentioned in the New Orleans Medical Journal, and quoted in the Dublin Medical Press, Vol. 13, p. 202, of a male negro slave, ætat. 25, in whom the stomach, liver, and spleen, and the thoracic viscera, were found to be respectively transposed; the heart occupied the right side of the chest, but no allusion is made to the manner in which its large vessels were given off. There was extensive adhesion between the heart and pericardium, and death had been caused by rupture of the right auricle, but nothing is said with regard to the condition of the valves and cavities of the heart. It is probable that in this case the cardiac lesions occurred quite independently of the peculiar arrangement of the thoracic viscera.
‡ New York Journ. of Med. May 1844.
§ From the Archiv. Gen. de Med. Feb. 1843, as quoted in the Lancet for 1842-3, vol. ii. p. 224. I have already briefly alluded to this case, at p. 278.

and have thus produced an unusually free communication between the aorta and the lungs.

In some instances of this malformation the auricular and ventricular septa have been found imperfect; in others, the foramen ovale only is stated to have remained unclosed; in a third set the arterial duct and the auricular septum have continued permanently open; in a fourth, there does not appear to exist any abnormal communication except through the ductus arteriosus; and, lastly, the ventricular and auricular septa, and the arterial duct, may all remain pervious.

These facts will prove that, although transposition of the two primitive arteries is very liable to determine the establishment of a permanent communication through the ventricular and auricular septa, and the arterial duct, it does not by any means necessarily prevent the natural closure of either these parts. In the majority of these cases the cavities of the heart occupy very nearly their natural position, while the transposed arteries arch as usual to the left. In one instance (Gomage's), however, although the position of the ventricles was partially reversed, the aorta arose from what is usually the pulmonary ventricle, and arched to the right, while the pulmonary artery originated from the systemic ventricle. In another case (Walshe's), the ventricles were transposed, but the aorta appears to have arched to the left.

Dr. Walsh has observed that, in several cases of transposition, the large vessels of the arch have been given off in an irregular manner; in other instances, however, these arteries have been distributed in the usual way.

The following are the most remarkable recorded instances of the several varieties of this species of malformation.

(1.) *Transposition of the pulmonary artery and aorta; the septa of the auricles and ventricles imperfect; the ductus arteriosus closed.*

Mr. S. S. Stedman has narrated* the case of a female child which was born at the full period of gestation, but which was observed to be small at birth, although nothing unusual was then remarked in her appearance. She seemed to be in good health, and throve well for a fortnight, at the expiration of which time, without any apparent cause, she was seized with a violent fit of coughing, with convulsions and dyspnœa. The surface of the body became of a deep purple colour, and the lips, tongue, and roof of the mouth, became almost black. These symptoms continued for about six or eight minutes, and then gradually abated; the child, however, never regained its natural colour, but remained of a dull leaden hue until death. The child was always cold, drowsy, and torpid, and grew very slowly. About three weeks before death the wrists and legs began to be œdematous; and, for the last fortnight of her life, there was evident fluctuation of the abdomen from ascites. Death occurred, during a severe paroxysm, at the age of 7 months and 8 days. On examination, the abdomen was found to contain a considerable quantity of fluid. The liver was dark-coloured and much enlarged, and on its surface were flakes of false membrane, the results of inflammation. The right lung was much larger than the left, and appeared better inflated; the left was compressed by the greatly enlarged heart. The pericardium was thickened, and contained two or three ounces of fluid, which, together with several patches of false membrane on the surface of the heart, indicated the existence during life of intense pericarditis. The heart weighed 3½ oz. Its position was natural. The right ventricle extended considerably more towards the left side than is usual, and the aorta arose from its base instead of the pulmonary artery. The left ventricle was situated almost entirely on the posterior part of the heart, and gave origin to the *pulmonary artery*. The origin of the aorta was situated in front and to the right side of the pulmonary artery; its arch took the usual direction, and terminated on the left side. Its valves were of the usual number. The branches arising from the arch came off irregularly: the innominata and left carotid arose by a short common trunk, and the next branch was the left vertebral, and the third, the left subclavian. The *pulmonary artery* passed upwards, parallel to and in contact with the aorta, but behind and to its left side. Its orifice was not so wide as that of the aorta; its valves were natural. A little above its origin it was very much dilated; it divided, as usual, into its right and left branches, and the *ductus arteriosus*, which was completely closed, arose from the left branch, and was attached to the aorta in the usual situation. The right auricle was distended with blood, and its walls were remarkably thickened. The venæ cavæ opened into it as usual; the parietes of the right ventricle were remarkably thickened, and its cavity was large. The right auriculo-ventricular orifice was much dilated; it was furnished with a tricuspid valve as usual. The left auricle was comparatively small, and had opening into it the pulmonary veins, which also appeared smaller than natural. The walls of the left ventricle were rather thinner than those of the right. The left auriculo-ventricular orifice was about half the size of the right. The mitral valve was

* Lancet for 1841-2, vol. i. p. 645.

natural. The ventricles communicated by a large opening in the upper part of the *septum*. The *foramen ovale* was closed, with the exception of a small opening of the size of a crow-quill.

Mr. T. W. King has given the particulars of a remarkable instance of this species of malformation.* The subject of this case was a child 2 years and 9 months old, a patient of Mr. Sanders, of Gravesend. There had been blueness of the lips and nails, and some discolouration of the face and skin generally. The child was tall but emaciated, and it never walked. It died rather suddenly. The right auricle was found to be dilated and hypertrophic. The *fossa ovalis* was large and cribiform, having apertures capable of admitting a black-lead pencil. The tricuspid valve was well formed. The right ventricle was large and strong, and contained a solid clot fully as large as a pigeon's-egg, of irregular shape, and lamellar structure, but softer within than without. The right ventricle was stronger than the left. The arch of the aorta was natural in size and structure, and in the distribution of the coronary and brachio-cephalic vessels. Below the attachment of the *ductus arteriosus* (which was quite closed) the aorta was rather small. The left auricle was of moderate size, and appeared to have received only two veins†. There was an opening between the ventricles larger than a crow-quill. The *pulmonary artery* was very wide; its valves were considered to be efficient, but scanty: its main branches were both of large size, but the right was much larger than the left. The heart is preserved in Guy's Museum, and numbered 1392[48].

II. *Transposition of the pulmonary artery and aorta; absence of communication through the ventricular septum and arterial duct: the foramen ovale open.*

Providing the description of the following case be complete and accurate, it affords an instance of a most singular and interesting malformation; but there appears to be great reason to believe that the author either overlooked certain important anatomical details, or in some way misinterpreted the appearances which he observed.

Mr. William Gamage has recorded the history of a female child, which was born at the full time, apparently healthy, but remained of a purplish hue, particularly in the lips and gums, and at the extremities of the fingers and toes. For two months, it showed no further symptoms of deranged organisation except uncommon feebleness and an indisposition to motion. But, in a short time, it became subject to frequent paroxysms of violent struggling; in which respiration was hurried and difficult, the face became black, the whole skin was injected with dark blood, and death seemed inevitable, but the child would recover after a fit, and immediately fall asleep. The paroxysms increased in frequency; it moaned continually, but never cried; the languor increased; it slept, except it awoke to suffer. Every movement was weak, every faculty ill performed. The right arm was paralysed, the eyes had a glassy appearance, aphthæ covered the mouth, and spread extensively on the nates: there was also considerable and distressing cough. Death occurred 15 weeks subsequently to birth. The whole heart was found distended with black and fluid blood to twice its natural size. The situation of its cavities was completely changed. The right auricle and ventricle were superior (anterior?), and to the left side of the chest; the left cavities were towards the right side of the chest: the apex pointed to the right. The aorta descended on the right side of the spine; the vena cava was on the left. The arteria innominata distributed blood to the left, while the single carotid and subclavian were sent to the right. The position of the whole of the organs of the thorax and abdomen was in like manner reversed. The aorta was very small, and arose directly from what is commonly the right ventricle, and rode over the root of the pulmonary artery, which had its origin in the opposite ventricle. The veins were connected with the heart in the usual manner; the blood of the cavæ, therefore, was received into the same ventricle that gave origin to the aorta, and the pulmonary veins poured their blood into the ventricle from which the pulmonary artery had its origin. No communication existed between the aorta and pulmonary artery; the *ductus arteriosus* seemed never to have existed. The *foramen ovale* was open, and very much enlarged. Mr. Gamage considered that, through this aperture, the blood on each side must have mixed, that being the only way in which the body could have received one drop of arterial blood.*

It will be observed, that the malformation which is described to have existed in the above case, was very far more complicated than that which is noticed in the generality of instances of complete transposition of the thoracic viscera; for there, although the

* Monthly Journal of Med. Science, 1844, vol. iv. p. 32. The fact that the main arterial trunks were transposed appears from the title of the case, but it is not expressly stated in the anatomical description of the heart and its appendages.

† The contracted state of the opening in the ventricular septum renders it probable that the other two pulmonary veins communicated with one of the cavæ or with the right auricle.

* New England Journ. of Med. and Surgery, vol. iv. p. 344: Boston, 1815.

position of the cardiac cavities and of their respective arteries is reversed, the cavæ open, as usual, into the pulmonary side of the heart, and the pulmonary veins into the systemic side; but here, on the contrary, the aorta arose from that portion of the heart which was supplied from the cava, and the pulmonary artery was appended to that side of the organ which received its blood from the pulmonary veins. Thus far, Mr. Gamage's case corresponds with many of the other examples of transposition noticed in this chapter; but the most singular feature of the case is, that the only communication stated to have existed between the two sides of the heart was through the dilated foramen of Botal. Had the ductus arteriosus been pervious, or had the septum of the ventricles been incomplete, the case would have been perfectly clear; but, admitting the correctness of the anatomical description, it is difficult to perceive in what manner the circulation was carried on, as it can scarcely be conceived that a portion of the venous blood passed through the foramen ovale on its way to the pulmonary ventricle, and subsequently returned, in an arterialised state, through the same opening to reach the systemic ventricle. It is most probable that either one of the venæ cavæ opened into the systemic auricle (only one vena cava is mentioned in the detail of the case), or that some of the pulmonary veins opened into the systemic auricle, or into the veins which communicated with that auricle, or that there existed an unusually free anastomosis between the descending aorta and the pulmonary vessels.

It appears that another case somewhat similar to that related by Mr. Gamage has been recorded*, but I have only been able to meet with the following brief particulars, as cited by J. F. Meckel. The aorta arose from the pulmonary ventricle, and the *pulmonary artery* from the systemic ventricle; the *arterial duct* was altogether closed, but the *foramen ovale* was open to the diameter of from eight to nine lines. The child lived 2½ years.

II. *The primitive arteries transposed; the ductus arteriosus and foramen ovale pervious: the septum of the ventricles perfect.*

The following case was observed by M. Ducrest.† A female child, born at the Hospital of Maternité, never had its respiration perfectly established; it never uttered a single cry, and refused the breast as well as all liquids which were offered to it. The skin was of a rather deep blue colour, and, notwithstanding extreme weakness, and these unfavourable circumstances, it survived its birth 10 hours. On examination, the body was found to be fully developed, and the limbs well proportioned, and it presented in every respect the appearance of a child born at the full period. Each lung presented a double fissure which divided it into three lobes; the lungs floated, and appeared to be healthy. The aorta took its origin from the right ventricle, and gave off in the usual manner two cardiac coronary arteries. The trunk of the *pulmonary artery* arose from the left ventricle, and, after a short course to the left of the aorta, divided into three branches—two going to the lungs, the third representing the *canalis arteriosus*, uniting with the aorta towards the termination of its curvature. All these vessels were of their usual proportions. The ventricle from which the aorta originated, besides its position on the right side, exhibited all the usual characters which distinguish it from the left ventricle. The two venæ cavæ and the large cardiac veins terminated in the right auricle; the left auricle received the four pulmonary veins. The *foramen ovale* was furnished with a perfect valve which opened from the right to the left auricle. The *septum* between the ventricles was complete. No unusual appearance was met with in any other part of the body.

A case of this description occurred to Dr. Wollaston, of St. Edmundsbury, and has been imperfectly recorded by Dr. Baillie.* A child, about 2 months old, was observed to have a most unusually livid skin, and the surface of its body felt colder than that of a child properly formed; the respiration was stated to be natural. After death, the heart was found to be of normal size; the aorta arose from the right ventricle, and the *pulmonary artery* from the left. There was no communication between these vessels except through the small remains of the *ductus arteriosus*, which was just large enough to admit a crow-quill. The *foramen ovale* was a little more closed than in a child newly born.

Dr. Farre† describes the case of a male infant (the eleventh child of a female six of whose infants were still-born at the full period) which appeared at his birth to have been well nourished. As soon as he was born, he began to cough violently. He lived 5 months, during which period he suffered from cyanosis, epigastric palpitation, coldness, difficulty of breathing, and cough. At the age of two or three months he had several fits, which were relieved by

* Winstar, System of Anatomy, vol. i.: Pennsylvania, 1811.

† Archiv. Gén. de Méd., Sept. 1840; and Edinburgh Med. and Surg. Journ. vol. lv.

* Morbid Anatomy (Second Edition), p. 38, Engravings of Morbid Anat. Fas. i. p. 6, figs. 1 and 2.

† Op. cit. p. 29.

the warm bath. He received the infection of small-pox, and expired in a fit when the pustules began to appear. The aorta, with its ventricle, marked by its muscularity and figure, constituted the right portion of the heart; the *pulmonary artery* branched correctly; the origin of the *ductus arteriosus* was preserved, but, unfortunately, its connexion with the aorta was dissevered by the manner in which the latter was truncated. The duct was too small to admit the rounded extremity of a common probe; the right auriculo-ventricular valve was tricuspidal, and the left mitral. The *foramen ovale* was imperfectly closed, the valve being cribriform, but the foramina were very minute. The lungs had a natural appearance.

The preparation in Guy's Museum, numbered 1392*0, is the heart of a young child in which the aorta arises from the right ventricle, and the *pulmonary artery* from the left; the *arterial duct* is open, the *ventricular septum* is complete, and the *foramen ovale* appears to be rather freely pervious.

A fifth instance of this kind is described by Dr. Walsh.*

The subject of this case was a decrepit-looking male infant, ætat. 10 months, with extremely flaccid flesh, and slight œdema about the ankles. The skin was of a deep leaden colour, particularly the toes and the extremities of the fingers. The surface felt cool, and the infant appeared to suffer from chilliness: it was restless; its eyes were prominent and staring; the respiration was very frequent, and sometimes gasping; the pulse between 120 and 130. There was no abnormal murmur in the cardiac region, or in the course of the great vessels; the heart's action was tumultuous, and its impulse strong and widely diffused. The child died during a paroxysm (of dyspnœa?), death being in all probability hastened by a slight diarrhœa and pulmonary catarrh under which it suffered at the time. It was found that the aorta arose from the right ventricle, and, at its origin, slightly overlapped the *pulmonary artery* which sprung from the left ventricle. The aorta gave off the two coronary arteries in the usual way, and two subclavian and two carotid arteries arose from the upper border of the arch. The aortic and pulmonary sygmoids were well formed and healthy; the *ductus arteriosus* was pervious, and large enough to admit with ease a good-sized probe. The right auricle contained an enormous quantity of black grumous blood, and an adherent fibrinous mass was found close to its appendix. The walls of the left auricle were almost membranous: scarcely any muscular fibres were to be seen in them; its cavity appeared to be about one-third as large as that of the right: it received the four pulmonary veins. The *foramen of Botal* was perfectly open, of an oval form: it measured about ⅝ths by ⅜ths of an inch, and was capable of being considerably dilated. The ventricles were transposed, that on the right side being furnished with a mitral valve; that on the left with a tricuspid valve. The *septum ventriculare* was not perforated.

* Medico-Chirurgical Trans., New Series, vol vii. p. 1 (1842). Dr. Walsh's paper contains many interesting particulars relative to this species of cardiac malformation.

It will be observed that in this, as in many other forms of malformation of the heart not primarily depending upon obstruction to the circulation, the children continued to be perfectly well nourished up to the period of their birth, but that symptoms of cyanosis became very apparent immediately the processes which tend to close the arterial duct and foramen ovale subsequently to the commencement of respiration began to operate. In M. Ducrest's case, respiration was never fairly established. In Dr. Wollaston's case, death occurred when the arterial duct had become very narrow and the foramen ovale had begun to contract. In the heart described by Mr. Stedman, the duct was closed, and the foramen ovale was barely pervious. A nearly similar condition of parts was found in Dr. Farre's case. The death of Dr. Walsh's patient was in some measure due to acute pulmonary and intestinal irritation; but the immediate cause of dissolution most probably existed in the narrowed condition of the arterial duct. The cause of the cyanosis in these cases has been very clearly explained by Dr. Quain.

IV. *Transposition of the aorta and pulmonary artery, the auricular and ventricular septa complete, the arterial duct pervious.*

There appears to exist a class of cases of this description, but, as I have already stated, it is difficult to understand how the circulation can be maintained at all in a case of transposition of the vessels where there is only a *single* communication between the pulmonary and the systemic heart and their vessels. In these instances it must be concluded that some means of communication have escaped detection, unless we suppose that, in such cases as the following, life has been prolonged until the period at which the foramen ovale has become completely closed, when a sudden stop has been put to the circulation. This explanation is, however, not altogether satisfactory, especially as the details of the cases are by no means complete.

Mr. Langstaff mentions the case of a well-proportioned male infant which had cyanosis immediately after birth, and was

much colder than natural. For three weeks it had only a slight difficulty in breathing, except when it sucked; but the impediment to the respiration gradually increased until it amounted to dyspnœa, during which the skin became still darker, and the temperature lower. At the age of 10 weeks it died suddenly in one of those paroxysms. The pulse was remarkably languid and small, sometimes obscure, and the bowels were disordered. The right auricle was found to be so much distended as to be nearly equal in size to the rest of the heart. The right ventricle was natural, except that the aorta arose from it; the left ventricle was as thin in its parietes as the right, and had a smaller cavity; it gave origin to the *pulmonary artery*, which communicated freely with the aorta by the *ductus arteriosus*, the size of which was proportionate to that of the other vessels*.

I have thought it necessary to state this imperfect case, but it is doubtful whether it does actually represent a distinct species of malformation. A careful examination of the specimen might decide this question, which, it will be perceived, is one of considerable physiological interest.

V. *Transposition of the primitive arteries: the ventricular and auricular septa pervious and the arterial duct open.*

In cases where the aorta and pulmonary artery are transposed, and the ventricular septum is incomplete, it may, of course, be expected that the foramen ovale and the arterial duct will be found unclosed if the infant die within a few hours of birth: still, I am not aware that this has ever been observed in the human subject. In the heart of a lamb which had not lived longer than 2 days, J. F. Meckel† found the position of the pulmonary artery and aorta reversed, the aorta arising from the right, the *pulmonary artery* from the left ventricle. The *ductus arteriosus* was widely open; the branches of the pulmonary artery were small; the aorta arched to the left. The *foramen ovale* was open, and the *ventricular septum* was incomplete‡.

* Lond. Med. Rev. vol. iv. as cited by Farre p. 28.

† Tab. Anat. Path. Fas. 2, p. 1, and Tab. ix. fig. 1.

‡ Meckel also refers to a similar malformation which he found in a bicephalous calf (p. 2). Besides the above instances, several other cases of transposition of the aorta and pulmonary artery have been recorded. Otto gives the following references:—Tiedemann, in his G. R. and L. Ch. Treviranus Zeitschrift für Physiologie, vol. i. p. 1, tab. vii. E. D'Alton, D. de Cyanopathiæ Specie ex invicem permutata arteriæ pulmonalis atque aortæ origine. 4to. Bonnæ, 1824, with plates. Joseph Barkart, D. de Monstro Humano Notabili, Friburg, 8vo. 1825. Dugès, in Journ. Gén. de Méd. vol. ci. p. 88. Dr. Walshe also refers to a case by Martin in Müller's Archiv. H. iii. S. 22, 1839. The child lived ten weeks.

* Epist. xvii. § 13.

† Medical Observations and Inquiries, vol. vi. p. 306.—My attention was directed to this passage by a friend.

‡ Med. and Phys. Journal, vol. xiii. p. 431.

THE CAUSES OF PERMANENT ABNORMAL COMMUNICATIONS BETWEEN THE CAVITIES OF THE HEART.

Throughout the foregoing observations, it has been assumed, as a well ascertained fact, that, in all cases where the cavities of the heart remain single, or in which the ventricular septum is found incomplete subsequently to the birth of the child, these structural imperfections have depended upon the occurrence, at a very early period of intra-uterine life, of a certain amount of impediment to the pulmonary circulation, the result either of a structural defect in the pulmonary apparatus, or of a distinct cause of mechanical obstruction existing in some other part of the vascular system. It has also been taken for granted that, in a large proportion of cases where the foramen ovale and arterial duct remain permanently open, the persistence of these canals is due to the influence of similar impediment operating either before or very shortly after birth. This view has arisen from observation of the frequent association of imperfection of the auricular and ventricular septa, and patency of the ductus arteriosus, with contraction or obliteration of the right auriculo-ventricular orifice or pulmonary artery, with imperfect development of the lungs, or with narrowing or occlusion of the mitral aperture, or of some portion of the aortic arch.

It would now be difficult to ascertain the precise time at which the influence of vascular obstruction in producing non-closure of the fœtal passages of the heart first became known to anatomists, but it is certain that the credit of establishing this view cannot be assigned to any writer of the present day. Morgagni was undoubtedly well acquainted with the principle,* and, in the year 1783, Dr. William Hunter referred† to the existence of an abnormal communication between the ventricles, as an arrangement intended to make up for the defect in a pulmonary artery which was preternaturally too small to give passage to the whole blood of the right ventricle, without which compensation life could not go on. In 1805, Dr. Walker published some remarks on a case in which he very expressly attributed deficiency in the septum of the ventricles to a want of capacity in the pulmonary artery.‡

Subsequently to this, Dr. Burns explained the dependence of an open state of the foramen ovale upon dilatation of the left cavities,

or contraction of the right ventricle and pulmonary artery.* M. Hein has also confirmed this observation, assigning contraction of the pulmonary orifice as one of the causes of permanence of the foramen of Botal.†

More recently, this principle has received strong confirmation from various pathological writers:—among others, MM. Berard and Bouillaud have remarked upon the coincidence of patency of the cardiac septa with causes of obstacle to the emptying of the cavities; and a very clear exposition of the matter has been given by Dr. C. J. B. Williams, in the Cyclopædia of Practical Medicine.‡

Similar opinions have also been adopted and extended by Mr. T. W. King,§ and by Dr. Craigie.‖

The establishment of this view has gone far to correct the opinion, which is, even still, entertained in its fullest latitude by a few pathologists, that imperfection of the auricular and ventricular septa, and permanence of the arterial duct, are invariably to be regarded as injurious states, as the primary and essential conditions of the disease, and as the principal, if not the sole causes, upon which the symptoms of morbus cæruleus depend. It is doubtless true that,—in a certain small proportion of cases where the open state of the fœtal communications has depended upon *transitory* causes of obstruction occurring very early in life, and has become aggravated by subsequent disease after the impediments which produced the non-closure have ceased to be in operation,—patency of one of these openings must be regarded as a morbid condition; still it must be borne in mind that, in every instance, these communications have originally fulfilled a preservative office, affording the sole means by which nature has been enabled to prevent the circulation from being wholly arrested in consequence of some severe cause of obstruction.¶

It is to be remembered, however, that instances of this uncomplicated kind are of by no means frequent occurrence; every anatomist who has practically investigated the subject will agree with Dr. Craigie that "the [permanently] open state of the foramen ovale" [and also of the ventricular septum and arterial duct] "is rarely a primary and solitary lesion." In fact, these defects will generally be found to exist in association with some permanent obstruction to the circulation, either in the right or left heart, or in some portion of the pulmonary or systemic vessels.* A very simple illustration of this principle may be found in any case where the pulmonary artery is narrowed at its orifice, and either the ventricular septum, the ductus arteriosus, or the foramen ovale, is permanently open:—here the contraction of the artery is the disease,—the abnormal communication is the preservative adaptation, by means of which the circulation is rendered comparatively free, and the life of the individual is prolonged. I have already endeavoured to trace out the influence which progressive narrowing of these communications has in aggravating the symptoms of the disease; and, ultimately, in determining its fatal issue,† and it cannot be doubted that, were it possible to induce closure of the openings in the cardiac septa, and of the abnormal passage of the arterial duct in these cases, severe and ultimately fatal aggravation of the symptoms of obstruction would be produced in the majority of instances; while, in many, the consequence would of necessity be instantaneous death. It is, of course, quite evident that, in a large proportion of cases of ventricular and arterial communication, and in some where the auricular septum only is deficient, the circulation would be at once arrested by any cause which could obliterate the abnormal channel. It therefore can never be a matter of surprise that a patient should have lived for many years with comparatively mild symptoms of heart disease, although the subject of a singularly large communication of the kind in question; as it will be perceived that, had there not been an abnormal aperture between the cavities or great vessels of his heart, to compensate for some permanent obstructive lesion in another part of the circulatory apparatus he probably could not have continued to

* Op. citat. p. 19.

† Op. citat. (pub. 1816), p. 14.

‡ Dr. Williams observes, that "Openings in the septum of the ventricles, when combined with decided contraction of the arterial tubes, we cannot but view as in most instances the effects of the latter; for it is a simpler idea, and one more consistent with the laws of development, to suppose that an obstruction, which pathologically speaking, is of frequent occurrence, retains the current of blood in the course which it held in the early period of its formation, than to ascribe a perforated septum to some specific and inexplicable arrest of its natural developement. Art. Malformations of the Heart, vol. iii. p. 66. 1834.

§ London Med. Gazette, vol. xxvii. p. 751. Jan. 1841.

‖ Edinburgh Med. and Sur. Journal, vol. lx. p. 274.

¶ Dr. Craigie has illustrated this principle in the following just remark: "In opposition to what has been hitherto taught, the open state of the foramen ovale is in a large proportion of cases, the means of prolonging life." Op. citat. p. 274.

* It is frequently, if not generally, observable that, where free patency of the foramen ovale exists as the sole congenital defect, it is associated with marked dilatation of the pulmonary artery, shewing that impediment has existed either towards the extreme branches of that vessel, in the left side of the heart, or in the systemic circulation.

† See the previous chapter, p. 72.

exist at all; or that, had the communication been less free than it was, the symptoms would have been more aggravated, and the term of life far less prolonged. Hence, in the majority of these cases, we must not be satisfied with discovering a deficiency in one of the septa of the heart, and in attributing death to that imperfection, but it will be necessary to search further for the cause of obstruction to the circulation which has originally produced that defect, and in the subsequent aggravation of which the cause of death may generally be detected.

It has been loosely stated, that permanence of the foramen ovale has less tendency to destroy life than has any other species of congenital malformation of the heart;—but, although it is undeniable that, under unfavourable circumstances, every imperfection in the cardiac apparatus, however slight, has a tendency to turn the balance against the chance of recovery, it must not, we repeat, be usually considered that the ill consequences of this defect at all outweigh its preservative influence. Persons live longer, and are healthier with an open state of the foramen ovale, than with any other prominent form of cardiac malformation, because this deficiency, when it exists alone, usually depends upon slighter causes of obstruction than does either of the others to which the heart is liable; on the contrary, where the ventricular septum is deficient, or the arterial duct remains rather widely open, the symptoms are usually of a more prominent character, and the term of life is considerably shortened: this is, however, traceable to the fact that the defects in question are almost invariably due to the presence of some very considerable source of impediment either in the pulmonary or systemic circulation. It is, therefore, by the amount of obstruction, and not by the extent of the abnormal communication, that our opinion on the severity of these cases must be formed.

M. Louis denies the probability of the opinion that a perforated condition of the ventricular septum depends upon narrowing of the pulmonary artery, but he admits that such constriction of the vessel must, if it be congenital, prevent the foramen ovale from becoming closed, in consequence of the resistance which it affords to the passage of the blood from the ventricle*. This distinction is evidently not a just one, as the same cause which would prevent the closure of the foramen of Botal subsequently to the birth of the child, would, if occurring sufficiently early, equally tend to arrest the formation of the ventricular septum. M. Louis' opinion was probably due to the observation of cases in which there was reason to believe that perforation of the septum ventriculare occurred subsequently to birth. It is, however, impossible to believe that the exceedingly frequent coincidence of a distinct mechanical impediment to the circulation through the pulmonary artery, with congenital deficiency of the ventricular septum, can be regarded otherwise than in the relation of cause and effect.

I am not aware, from my own observation, that stricture of the aortic orifice, where that vessel is distributed from the left ventricle, has ever been found to occasion congenital deficiency of the ventricular septum,* but various forms of obstructive aortal disease, occurring at a later period of life, are observed to be attended with a tendency to perforation, or aneurismal yielding of the upper and fibrous portion of the septum, which is occasionally found to be extremely thin immediately below the base of the right aortic sygmoid valve,—a condition which has been described by Dr. Spitta, and which I have myself seen. Perforation of the ventricular septum may also occur at various periods of life from several causes, such as abscess, rupture consequent upon fatty degeneration, acute ulceration in rheumatism, &c.

In his recent work on Narrowing and Obliteration of the Arteries in Disease, Professor Tiedemann notices the frequent association of patency of the foramen ovale with arctation of the orifice of the pulmonary artery, whence Morgagni inferred that the narrowing of the vessel had taken place at birth; but the Professor contends that this could not be the case, otherwise the ductus arteriosus must be open also, which it is not. This reasoning is, however, erroneous; the ductus arteriosus has been found open in several instances of contraction of the pulmonary orifice†, and we have seen that, in cases of congenital obliteration of the ascending pulmonary trunk, permanence of the arterial canal is the most frequent compensating provision; on the other hand, a case of congenital narrowing of the pulmonary orifice has been recorded‡, in which the foramen ovale was obliterated, while the ductus arteriosus remained pervious. And, in a very large number of examples, where the congenital origin of arctation of the right ventriculo-arterial orifice was evidenced by its association with an incomplete condition of the ventricular septum, both the foramen of Botal and the arterial duct

* Mémoires ou Recherches Anatomico-Pathologiques, Paris, 1826.

* It has been argued that congenital imperfection of the septum may be determined by a narrowed state of the aortic orifice; but I am not acquainted with any of the cases upon which that conclusion is based.

† Records of six cases are cited at pp. 373-4-5, and 452.

‡ Dr. Houston's case, p. 374.

were found perfectly impervious.* Indeed, a case which I have cited, at the commencement of this paper (Dr. T. Chambers' case, p. 284), proves that the pulmonary orifice may become completely obliterated during intra-uterine life, without preventing the subsequent closure of the duct and foramen. I have already attempted to explain the reason why permanence of these two canals is not determined in all cases of congenital narrowing of the pulmonary orifice (pp. 372, 3). In some instances of contraction of this aperture, in which patency of the foramen ovale is the only other associated lesion, it may possibly not be very easy to decide with absolute certainty whether the morbid appearances are congenital, or whether, as Professor Tiedemann suggests, the foramen may not have been reopened in consequence of the impediment which the blood encountered in moving from the right side of the heart; still, the cases must be of rare occurrence in which a careful examination of the structures will not enable the anatomist to form a tolerably accurate opinion as to whether the lesions in question were of congenital origin or of more recent date. It may perhaps happen that, in some cases of great contraction of the pulmonary orifice, either the ventricular or the auricular septum may become thinned, and perforated by ulceration;† but I am not acquainted with any cases in which the appearances presented by the abnormal apertures rendered it at all certain that this event had occurred‡. It will generally be easy to distinguish an ulcerated opening in any portion of the cardiac apparatus from a congenital deficiency of the tissues of the part; and I believe that most persons who have been in the habit of examining malformed hearts, will concur in declaring, without the slightest hesitation, that the smooth and regularly formed circular openings through which the blood is diverted in cases of arctation of the pulmonary artery are indisputably the results of congenital defect. If the opinions of Professor Tiedemann are intended to apply to cases of this kind in general, they are assuredly not valid, and they cannot be regarded as in any degree subversive to the views on this point which had been long previously established.

The only other controvertible opinion on this subject which it is necessary to allude to here, is that of Mr. Thiebault, who has suggested that the blue disease may arise, in the first instance, from a plethoric state of the infant at birth, preventing the changes which should take place in the circulation at that period. It is true that the severity of the first symptoms of cyanosis may occasionally sustain a temporary relief from the employment of that practice which Mr. Thiebault recommended on the strength of his theory, viz. of allowing a certain quantity of blood to flow from the umbilical cord immediately after birth: still, it is of course impossible to believe that a cause of this kind, occurring at so late a period, could have any influence in producing an irregular disposition of the vessels of a malformed heart, or that the treatment in question could avail in modifying the confirmed structural defects which are observed in such cases.

If, however, it be merely argued that engorgement of the heart may become the cause of the first *symptoms* of cyanosis in cases where the organ is congenitally malformed, or that a state of extreme plethora may have a tendency to retard the closure of the foramen ovale and arterial duct, the plausibility of the opinion can scarcely be denied.

ON THE CAUSES OF CYANOSIS.

The malformations to which the heart is liable usually consist of lesions, which are not inconsistent with the prolongation of intra-uterine life*, but which are calculated to produce very serious embarrassment when respiration commences, and when the alterations naturally effected after birth in the circulatory apparatus begin to be established.

The paroxysms of suffocative dyspnœa, the lividity of the surface, and all the other distressing symptoms which constitute the leading features of cyanosis, were formerly

* For examples, see p. 699.

† It has been considered that in cases of extreme mitral contraction, the fossa ovalis occasionally becomes perforated in this manner — this may doubtless occur; but it is probable that, to say the least, in the majority of these instances, the open state of the foramen is in reality merely an accidental complication, the size of the congenitally patent communication becoming much increased by subsequent distension. This latter opinion has been clearly laid down by Dr. Farre, and it is certain that, in cases where the foramen ovale is perfectly closed, distension of the left auricle may proceed to an enormous degree without giving rise to perforation of the septum. In the Museum of St. George's Hospital there is a specimen of mitral contraction in which a most extraordinary dilatation of the left auricle has occurred, rendering the septum perfectly translucent, but without destroying the continuity of its fibrous tissue. Professor Tiedemann himself refers to a still more remarkable case of this kind in which the auricle was more than large enough to admit three fists within its dilated and thin-walled cavity.

‡ A heart is preserved in Guy's museum (numbered 1380), which presents a large perforation, by some considered to be the result of ulceration, in the upper part of the ventricular septum. Both the aortic and the pulmonary valves are considerably diseased, but there does not exist any remarkable degree of narrowing at the orifice of either vessel.

* There are some exceptions to this rule, but they do not appear to be of frequent occurrence.

attributed solely to the admixture of venous with arterial blood through the abnormal cardiac apertures which are usually discovered in these cases, and to the consequent diffusion of a dark and vitiated fluid through every part of the arterial system; but this opinion has been in great measure abandoned since the facts have been established that the symptoms in question may be present in cases where no abnormal communication whatever exists between the cavities of the heart, as well as in instances where it is utterly impossible that the smallest quantity of venous blood could have entered the arterial system: while, on the other hand, the symptoms of morbus cæruleus are not by any means necessary attendants either of patency of the cardiac septa or of permanence of the arterial duct.

The opinion at present adopted by many pathologists with regard to the cause of the symptoms of morbus cæruleus is, that they depend entirely upon delay to the passage of the blood through the lungs, resulting from the presence of a fixed impediment to the circulation.

Morgagni appears to have been the first writer who attributed the intense lividity of cyanosis to obstruction in the trunk of the pulmonary artery. Louis ascribed this symptom to some obstacle to the circulation of the blood through the veins; and MM. Bertin and Berard coincide in believing that the blue appearance of the surface in those affected with abnormal apertures in the cardiac septa depends on the stasis of the blood in the right cavities of the heart, and upon the consequent difficulty with which the venous blood circulates; and, though it be complicated almost always with the mixture of the two kinds of blood, still it is not produced by this mixture. The opinion that cyanosis is exclusively due to the circulation of venous blood through the arterial system, has been satisfactorily disproved by Dr. Stillé*, who adduces ample evidence in proof of the conclusions, (1) that cyanosis may exist without admixture of the blood; (2) that there is not always a proportion between cyanosis and the degree in which the blood is mixed; (3) that complete admixture of the blood may take place without cyanosis; and (4) that cyanosis depends upon congestion of the general venous system from obstruction in the right side of the heart or in the pulmonary artery, impeding the return of its blood to the lungs.†

The results of my own investigations are almost entirely confirmatory of Dr. Stillé's inferences. Cases of cyanosis will very rarely occur in which the morbid anatomist will fail to discover some organic cause which acts virtually as an impediment to the pulmonary circulation. Dr. Stillé has, perhaps, referred somewhat too exclusively to the right side of the heart and the pulmonary artery as the seats of the mechanical obstacle to the circulation in these cases, for it will occasionally, though rarely, be found that the physical impediment to the circulation exists in the pulmonary tissue, or is even external to the lungs, as in Dr. Marcet's well-known case*; and, in some few instances, the cause of obstruction is situated either in the left heart or in the aorta. Still, in every case of cyanosis, there will be found to exist some cause or other which tends essentially to prevent the free and complete circulation of the blood through the lungs, to retard its passage through the venous system, and, consequently, to render the process of its arterialization slow and incomplete.†

Dr. Stillé has also argued that obstruction to the pulmonary artery is never found without the concurrence of cyanosis. This is perfectly true as regards most of the cases of congenital narrowing of this vessel, but it does not hold good in all: for instance, where congenital imperfection of the pulmonary valves does not become seriously obstructive until late in life, the symptoms which it produces are not necessarily those of cyanosis‡; and I shall hereafter have to cite an instance in which extreme narrowing of the pulmonary orifice, the result of endocarditis occurring at the adult period, was not attended with the slightest appearance of lividity of the surface: in fact, it appears that, for the complete establishment of that generally dilated condition of the entire

* In a valuable paper on Cyanosis, in the Americal Journal of the Med. Sciences, vol. viii. p. 25: 1844.

† In 54 out of 62 cases of cyanosis analysed by Dr. Stillé, the pulmonary artery was either contracted, obstructed, or impervious. In the remaining 8, the conditions presented by this vessel were fully capable of producing great venous congestion.

* Edinburgh Med. and Sur. Jour. vol. i. p. 412.

† As a general rule, the heart's action is greatly accelerated in cases of cyanosis depending upon contraction of the pulmonary orifice, and upon various other kinds of cardiac malformation: an arrangement by which the smallness of the quantity of blood which passes through the lungs, and becomes oxygenised there, is, in some measure, compensated by the rapidity of its transit. Still, in the majority of cases, this adaptation is not sufficiently complete either to prevent great delay to the circulation through the right cavities of the heart, or to produce the oxygenisation of the entire volume of the circulating fluid so rapidly or so effectually as is usual.

‡ In the cases of a man, ætat. 44, related by Dr. Craigie, and of a woman, ætat. 63, detailed by Dr. Fallot (cited at pp. 749-50 of this paper), the pulmonary valves were found united into a thickened ring: in the one case, capable of admitting the end of the little finger; and in the other, merely allowing the passage of a goose-quill. There were evidences of very considerable impediment to the circulation in both of these cases, but there does not appear to have been any cyanosis in either.

venous system which attends cyanosis, the obstruction to the circulation must have been present either at or before birth, when the capillary vessels are naturally more capacious than they are in the adult, or it must become confirmed previously to the full development of the body, while the entire vascular system is pliant and dilatable, and is still capable of readily adapting itself to permanent changes in the circulation.

It is, of course, well known that various kinds of obstructive disease of the heart and lungs, occurring in adult life, are liable to produce extreme internal venous congestion and considerable lividity of the surface; but I am not acquainted with any instance in which an impediment of this kind, coming into operation subsequently to the age of twenty-five years, has produced that general and intense blueness of the entire surface which forms the characteristic feature of true cyanosis depending upon congenital malformation of the heart.

In extreme cases of original defect of the cardiac apparatus, such as those in which the ascending pulmonary trunk is obliterated or absent, the cyanosis appears to be due less to the circuitousness of the course by which the lungs are supplied with blood, than to the unnatural narrowness of the pulmonary vessels,* which are almost invariably far less capacious than in the ordinary condition; hence the pulmonary veins and left auricle are usually more or less contracted in these cases, while the lungs are either badly developed and imperfectly expanded, or present the evidences of chronic impediment in the dilated condition of their tubes.

"There are still a few pathologists who adhere to the old opinion that cyanosis mainly depends upon the circulation of carbonised blood through the arterial system, insisting upon the fact that, in the great majority of cases of morbus cæruleus, the septa of the heart are more or less deficient. As I have already stated, it is now established that cyanosis may exist quite independently of imperfection of the cardiac partitions or of admixture of the venous and arterial blood: still, I apprehend that M. Berard and Dr. Stillé have argued somewhat too exclusively in maintaining that admixture of the two currents has no influence whatever in producing cyanosis, as it appears by no means unreasonable to conclude that, in extreme cases of this kind, where the impediment to the pulmonary circulation is great, and where a large quantity of venous blood evidently passes into the aorta at every systole of the ventricles, the discolouration of the surface, and especially the lividity of the mucous membranes, which is so frequently observed in these cases, is, in part at least, due to the dark hue and impure condition of the arterial blood. Admitting this, it must be borne in mind that the principal reason why cyanosis is generally present in cases of extensive communication between the cavities of the heart will be found in the fact that a cause of obstruction which is capable of preventing the natural closure of the septa will rarely fail to occasion permanent and severe impediment to the circulation. Where an abnormal opening is discovered in the cardiac apparatus of one who has only lately become cyanosed, or where such an aperture presents traces of recent enlargement, it must not be at once concluded that the presence or augmentation of this communication has occasioned the cyanosis; but the first cause of the disease must be sought for, and this will generally be discovered in the form of some manifest impediment to the circulation which has determined the patency of the opening from birth, and which, having become recently aggravated, has produced the cyanosis at the same time that it has increased the size of the abnormal foramen.

Dr. Meigs adheres to the doctrine that persistence of the foramen ovale is the cause of cyanosis in infants. He observes, that, "as the occlusion of the foramen ovale is prevented by the torrent of blood flowing from the inferior vena cava, raising and keeping raised the interauricular valve, which is thin and floating, it occurred to him to place the cyanosed child on the *right* side, with the head and trunk somewhat raised, so that the interauricular septum should be maintained horizontal, and the blood contained in the left auricle should press with its whole weight on the closed valve. He has frequently seen the blue colour disappear at the very instant the infant was placed in this position, proving that the oxygenating blood only entered the arteries." Dr. Meigs adds, that he has thus saved the lives of fifty or sixty children in a hundred; whereas, as is well known, all the other means hitherto tried have failed.*

Successful as this application of Dr. Meigs's theory has evidently proved, it is certain that his explanation of the fact is by no means demonstrative. So far from patency of the foramen ovale being an essential concomitant of the blue disease, it is well known that, in a very considerable proportion of instances of cyanosis, the auricular septum is perfectly closed; and two cases

* Some of the cases in which the heart has been found situated below the diaphragm, prove that, where its canal is free, great deviation in the course of the pulmonary artery is not a necessary cause of dangerous impediment to the circulation, and is not the main cause of dyspnœa and cyanosis in extreme cases of malformation.

* Report of the Academy of Sciences, Paris, June 2nd, 1845, and Dublin Med. Press, vol. xiv. p. 18.

are upon record* in which cyanosis was distinctly attributable to closure *ante partum* of the foramen of Botal. Wherever this communication remains too long open in a child, there must exist some cause, either of obstruction to the circulation, or of over-distension of the heart, to prevent its closure; and it is to that cause, and not simply to the patency of the auricular septum, that the cyanosis is due; otherwise it is clear that every infant would remain cyanosed until the termination of the usual period at which the foramen becomes naturally closed, and every individual whose auricular septum remained imperfect would be the subject of morbus cæruleus,—neither of which circumstances are found to obtain. The position of the body recommended by Dr. Meigs is, however, well calculated to relieve those paroxysms from which the subjects of congenital heart disease suffer, as it places nearly the whole of the voluntary muscles in a state of relaxation, thereby rendering the circulation through the extreme vessels as free as possible, and (what is of still more importance) as it facilitates the supply of arterial blood to the lungs and to the brain.

Much unnecessary discussion has been expended upon the question of whether, in cases of septal deficiency, admixture of the venous with the arterial blood occurs constantly, or only as the result of occasional causes of impediment to the pulmonary or systemic circulation. In by far the larger proportion of instances of extensive congenital malformation of the heart, and certainly in all those cases where direct communication between the cavities or arteries exists as the result of a permanently obstructed state of any of the cardiac orifices or vessels, admixture of the two currents of blood is a matter of necessary occurrence—the sole means by which the circulation is maintained at all; and here the state of the parts shews at a glance in which direction the diverted current has been accustomed to pass. Thus, in cases of transposition of the aorta and pulmonary artery, where the ventricular septum and foramen ovale remain pervious, it is evident that blood must be continually passing directly from the right to the left ventricle, and from the left to the right auricle. In other instances, where the orifice of the pulmonary artery is closed, and the aorta arises from the right ventricle, it is apparent that the contents of the left cavities can only reach the aorta by passing from left to right through the aperture in the septum, which is always provided in these cases. So, also, in the majority of instances where the foramen ovale remains open, but protected by an efficient valve, it is clear that blood has traversed the aperture only from right to left. But in many cases of congenital malformation of the left cavities of the heart, it is evident that the current through the foramen has always been from left to right. In cases of patency of the ductus arteriosus, associated with contraction of the pulmonary orifice, the lungs, of course, receive some portion of their supply of blood through the duct from the aorta; but, where there exists a contracted or obliterated state of the aorta below the origin of the left subclavian, it not unfrequently happens that a considerable stream of blood is regularly conveyed by the duct from the pulmonary artery into the aorta. In the larger proportion of these cases it is impossible that the direction of the current should be permanently reversed; the foramen ovale is generally defended on one side by a more or less efficient valvular apparatus, and an analogous arrangement has occasionally been developed in patency of the ventricular septum and arterial duct*.

It has been argued by M. Cloquet and Dr. Willis, that when the right and left cavities of the heart are of equal and proportionate strength, no admixture of the arterial and venous blood will occur during their contractions, even although there may exist free communications between the vessels, or through the septa. A few cases have been observed which go far to substantiate the general correctness of this doctrine; but the instances of extensive malformation of the heart are so few in which the two sets of cavities are exactly proportioned to each other, or in which the whole of the cardiac outlets are perfectly free from obstruction, that the rule is by no means one that admits of being either extensively or frequently applied.

It is now allowed by the majority of pathologists that, in itself, patency of the foramen ovale (where the opening, although free, is guarded by an efficient valve) is by no means necessarily attended with cyanosis; and it is probable that, where this exists as the principal defect in the cardiac apparatus, the passage of blood through the aperture is ordinarily by no means large, and that the transit of a full stream from one auricle to the other may be merely an occasional occurrence for the purpose of relieving distension under circumstances of accidental engorgement or obstruction. Still, it is doubtful whether we can fully admit the opinion of Bichat and Louis, that, "in examples of septal deficiency, or at least in cases of open foramen ovale, no admixture of venous with arterial blood occurs except

* Cases by Vieussens and Mr. Ebenezer Smith, p. 967.

* See cases observed by Richerand, p. 1087, and by the author, p. 1090.

under circumstances of obstruction;" for, as we have already seen, these deficiencies are seldom, if ever, present where there is not also discoverable some cause of permanent impediment to the circulation, which probably at all times occasions a certain degree of commingle ment of the currents, although that mixture may not be sufficient to produce serious vitiation of the arterial blood. It is generally found that, when the subjects of the minor degrees of septal deficiency become effected either with pulmonary disease, or with any causes of delay to the systemic circulation, the dyspnœa and lividity of the countenance are greater, and the consummation of the fatal issue is usually more rapid than might have been expected from the extent of the recent pulmonary disease, or from the severity of the other superadded causes of obstruction, had these existed alone—facts which go far to corroborate the belief, that in cases of permanence of the septal openings there generally exists some fixed impediment to the circulation, although that impediment may not be sufficient to produce any visible ill consequences while the heart is tranquil, and the lungs remain free from congestion or other superadded lesion.

It is a demonstrable fact, that there may constantly occur considerable commixture of venous with arterial blood, and yet the individuals may be well nourished and active, and may arrive at maturity without ordinarily presenting sufficient blueness of the surface to attract the attention even of a medical man.

In other instances of this kind the patients may continue for many years to enjoy tolerable health, being only occasionally liable to more or less lividity of the surface, either with or without a certain amount of dyspnœa, occurring in consequence of extraordinary exertion, repletion, or transient causes of pulmonary obstruction. Here the intensity of the cyanosis can never be taken as an indication of the degree of abnormal communication which exists between the cavities of the heart. Louis has very justly remarked, that "the change of colour is never found to be in proportion to the freedom of the communication;"—for it is, of course, evident that, wherever obstruction of the outlets exists, the more freely the cavities communicate the less will the circulation be impeded.

In either of the above sets of cases the symptoms of morbus cæruleus may become permanently developed in their greatest intensity whenever additional and permanent obstruction occurs to the passage of the blood through the lungs, or immediately the muscular power of the heart becomes seriously impaired. Instances of considerable malformation of the heart occasionally occur in which cyanosis does not appear until the age of puberty, and others have been observed where the lividity of the surface, which had occasionally presented itself from birth, did not become permanent until a rather advanced period of life. In the former of this class of instances the increased impediment is probably due to a want of that development of the pulmonary apparatus which usually takes place at the approach of adult age; in the others it may be traced to additional narrowing or other consequences of acquired disease in the malformed structures, causes which are probably further aggravated by plethora, and by a certain amount of deterioration of the lungs.

Allusion has been already made to the influence of contraction of the foramen ovale and arterial duct in producing the first symptoms of morbus cæruleus in children who are the subjects of congenital cardiac defect; it does not usually appear that such defect necessarily interferes with the health of the infant so long as its system is freely supplied with placental blood; but, so soon as respiration and the organic changes which accompany the commencement of that process become established, the malformed heart fails to perform with facility functions for which its structure very imperfectly adapts it, and the evidences of severe obstruction are quickly developed: these are, in all probability, also aggravated by the increased bulk of the fluids, which is produced when the process of assimilation commences. The opinion advanced by M. Billard, that a perfectly oxygenated blood is not necessary to the new-born fœtus, taken in conjunction with the fact that the infant's body has usually a slightly livid appearance until the funis is secured and respiration is fully established, has been regarded as a sufficient explanation of the circumstance that several hours or days frequently elapse after birth before the symptoms of the blue disease present themselves in those children whose hearts are structurally imperfect. I am not, however, aware of any fact which proves that the blood supplied to the fœtus during intra-uterine life is less completely oxygenised than that which circulates through the arteries of the mother; and it is evident that the slight discolouration of the surface alluded to above is merely the transient result of the embarrassment and delay which the circulation necessarily sustains at the time when the infant is gasping in its first efforts to inspire.

ACUTE INFLAMMATION OF THE PULMONARY ARTERY.

Acute inflammation, arising spontaneously in an extensive tract of either of the great thoracic arteries, is a disease of by no means

very frequent occurrence: still, for many years past, acute aortitis has been recognised as a malady of perfectly defined character; and it appears to have fallen to the lot of most recent morbid anatomists to observe cases in which it has been evident that a more or less active form of inflammation of the pulmonary artery was one of the leading morbid appearances discovered after death. Still, even at the present time, some of those pathologists who have observed the larger proportion of the instances in question, deny that the morbid changes discovered in the artery have been, in reality, due to inflammation of its tissues, regarding them as the results of mere passive coagulation of blood within the vessel, either consequent upon impediment to the pulmonary circulation, or produced by a morbid tendency to spontaneous coagulation in the blood itself. Cases have, however, occasionally, though rarely, presented themselves, in which it was evidently a matter of absolute certainty that the lesions discovered in this artery were the results of acute and very active inflammation. In other instances, the leading features of the disease, as well as the morbid appearances presented by the artery, have not assumed so definite a character as to render the inflammatory origin of the lesions perfectly indisputable: still, even in these cases, the probability has been altogether in favour of the belief that the disease was essentially one of inflammation. Apart from these, there occurs a third class of highly important cases, in which the lesions bear a considerable resemblance to those displayed in the previous instances, but in which there is every reason to believe that the adherent coagula and other morbid appearances discovered in the vessel have resulted mainly from obstruction to the pulmonary circulation, attended, doubtless, with a certain amount of inflammation in the over-distended vessels. In a fourth set, it is extremely difficult to ascertain whether any inflammatory action has been at work in the formation of the fibrinous deposits found in the artery: here, if inflammation has existed, it has evidently been of a transient nature, and of a far less distinct type than in those cases alluded to under the first class.

I shall endeavour to present a brief description of each of these classes of cases in succession; commencing with those in which the symptoms during life, and the morbid appearances discovered on examination, were evidently due to acute inflammation of the pulmonary artery: alluding first, for the sake of arrangement, to those instances in which the *branches* of this vessel were the principal seats of disease; reserving those in which the *valves* were seriously affected for a subsequent section.

The comparative immunity from disease of the right cavities of the heart and pulmonary artery, as compared with the cavities on the left side and the aorta, has long formed a matter of speculation to pathologists: and many ingenious conjectures have been advanced in elucidation of this difficult question. Corvisart attributed this difference to the more decidedly fibrous organisation of the valves on the left side. Bertin regarded it as an effect of the more stimulating nature of the arterial blood. A similar view has been adopted by Dr. Furnivall, who conjectures that, as the "fully elaborated arterial blood is the natural stimulus to the heart's action," this fluid, "highly elaborated, and rich as it is in stimulant material, may be rendered by disease more stimulating to the parietes of the cavities and canals through which it circulates than the venous would be to the right ventricle." Dr. Hope believed that a concurrent, if not a principal cause, is to be found in the greater pressure or strain to which the left valves are subject, in consequence of the superior contractile energy of the left ventricle, and the greater weight of the arterial circulation. The correctness of this last opinion is strongly borne out by the fact, abundantly illustrated in the preceding chapters of this memoir, that, during intra-uterine life, while the right ventricle and its artery have a principal share in transmitting the blood through the systemic circulation, these parts are extremely liable to become the seats of disease and mechanical injury; while the left cavities and ascending aorta, to which, at this period, a much less active function pertains, comparatively rarely suffer from any serious lesion until after the direct communication between the right heart and one main systemic artery has been permanently cut off. It is probable, however, that here, as in the generality of nature's provident operations, the result is due not to a single cause, but to a combination of associated influences.

I am disposed to attribute the greater immunity from mechanical injury and disease of the right heart and pulmonary artery, in some measure to the comparatively yielding nature of their structures, the free reflux of which the tricuspid valve admits, whenever the ventricle is over-distended; and the great pliability and extensibility of the muscular, valvular, and arterial tissues,—circumstances which must all have an influence in rendering the parts less liable to mechanical injury upon occasions of obstruction and over-excitement. In a very large proportion of the cases of spontaneous mechanical lesion of the heart, the mischief is found to have occurred on the left side: the right cavities being more accustomed to obstructions, and being naturally more yielding, do not so readily suffer from undue distension. It is also highly probable that the cavities on this side of the heart are naturally more adapted to the reception of irritating fluids than are those on the left: as, in health, the former receive the highly carbonized blood from the veins, while the latter are accustomed to transmit only a perfectly depurated stream, so, in disease, a strongly azotised, or otherwise morbid fluid, may be conveyed with impunity by the right cavities, while it produces the most irritating effects upon the left. All are aware that pungent substances, which may be borne without injury on the mucous membrane of the interior of the lip, produce the most violent inflammation if applied to the conjunctiva of the eye; and yet it would be difficult to assign to this any other reason than that of necessity.

It has been already shewn that, in its structural characteristics, the pulmonary artery holds an intermediate position between the arteries and veins, possessing several of the characteristics of both classes of vessels; it is also observable that the acute diseases to which this artery is liable, present, in many respects, the combined characteristics of phlebitis and arteritis; indeed, upon the whole, the morbid conditions of the pulmonary artery appear to be more nearly allied to those of the venous than to those of the arterial system. The general pathological laws which regulate the deposition of coagula in these vessels, and the circumstances which produce decolouration of their tissues, appear, however, to be nearly the same in both instances.

I believe that the only perfectly unquestionable anatomical evidences of the recent existence of acute arteritis, or phlebitis, are the presence of layers of fibrine, or portions of coagula closely adhering to the lining of tracts of the diseased vessels, together with an infiltration of their subepithelial tissues with a deposit of translucent fibrine, (an appearance which is, however, very rarely indeed observed either in the veins or in the pulmonary artery;) and lastly, a certain degree of reddish discolouration of the interiors of the inflamed vessels.

Morbid anatomists are accustomed to meet with four kinds of coagula in the cavities of the heart, and in the larger arterial and venous trunks: viz. (1.) Loose masses of dark crassamentum, filling the cavity or tube, but not adherent to its parietes: these may either be found in association with an inflamed state of the portions of vascular tissue with which they are in contact, or may be simply the results of the ordinary coagulation which occurs within a few hours after death. (2.) Soft masses or cords of yellow fibrine, of laminated structure, enclosing in their interstices so large a quantity of serum, that very slight pressure with the fingers is sufficient to reduce them to less than half their original bulk; these are usually attached to layers of dark coagulum, and are evidently formed subsequently to death. (3.) Laminated masses and cords of flesh-coloured fibrine, tough, elastic, free from serum, and compressed into the smallest compass, evidently by a strong condensing force; in fact, these coagula closely resemble portions of fibrine which have been squeezed in a cloth until their serum has been removed. When occupying the ventricles, they are generally found interlaced between the cords of the mitral and tricuspid valves. It is almost impossible to avoid the conclusion that these coagula have formed previously to the complete extinction of life, and that their condensation has been produced by the muscular contraction of the living heart. (4.) Patches, tubes, or masses of more or less decolourized coagula, opaque and friable, and closely adherent to portions of the lining membrane of the diseased cavity or vessel. Wherever deposits of this kind are found in any portion of the vascular apparatus, the previous existence of an amount of inflammation sufficient to produce a complete alteration in the function of the lining membrane in those portions of the vessel to which the morbid deposit is attached, may be taken for granted. Each of these kinds of coagulum is liable to be found in the trunk and branches of the pulmonary artery, and a careful examination of their several distinctive characters will often prove the principal means of ascertaining the condition of the vessel previously to death.

It will here be necessary to offer a few remarks upon the red or purple discolourations so often met with in the pulmonary artery, as well as in other parts of the vascular apparatus; and which have been too exclusively attributed either to acute inflammation of the stained tissues, or to the post-mortem transudation of the colouring matter of the blood quite independently of any previous organic lesion. There are, doubtless, instances where reddening of the arterial and venous tissues occurs as a vital process; but the longer I have investigated the diseases of the blood-vessel system, the more I have become convinced that, in at least the larger proportion of cases, the florid stains which are frequently observed in the interiors of those vessels, whether arteritis or phlebitis has or has not existed, are the results of staining, or imbibition of the blood subsequently to death. It appears that the interiors of the large vessels are capable of receiving a red colour by vascular injection; but I feel assured that the discolourations in question are, in a vast majority of instances, post-mortem changes. Still it is important to observe that the pre-existence of inflam-

mation or softening in the tissues renders them particularly liable to become stained very shortly after death. In fatal cases of typhus, erysipelas, phlebitis, &c. I have occasionally found the heart and entire tract of the arterial system deeply stained, even before the body was totally devoid of heat: this evidently depended upon the low crasis of the blood, and its rapid disintegration, as well as upon the deficient cohesion of the vascular tissues.

In extremely close and sultry weather, and especially where the quantity of fluids in the bodies is large, as well as in certain cases of sudden death, poisoning, death by lightning, &c. it will frequently be found that transudation of the colouring matter of the blood through the vascular tissues commences very shortly after life becomes extinct quite independently of any previous structural fault in the blood-vessels themselves: but I think that, in the generality of cases, where, within two days after death, in cold weather, and under ordinary circumstances, the interiors of the large arteries and veins are found greatly discoloured, it may usually be concluded that, although this appearance may possibly not be associated with any inflammatory lesion, it has resulted either from a highly vitiated state of the circulatory fluid, from impaired vital cohesion of the vascular tissues, or in consequence of both of these causes combined. It is well known that the disease which has been termed erysipelatous arteritis usually proves so extremely rapid in its fatal progress, as not to admit of the gradual deposition of adherent lymph or coagula upon the interiors of the inflamed vessels—whence the only morbid appearances presented by these canals after death, is intense discolouration, with or without a certain degree of thickness and softening of their inner tunics; appearances which it is, at present, scarcely possible to distinguish from the effects of ordinary decomposition. The microscope may hereafter be successfully employed in discriminating between these cases; but we are now capable of forming our opinion respecting their true nature only by connecting the appearances presented in the vessels with the symptoms observed during life.

Acute inflammation of the pulmonary artery has been found to occur under the following circumstances. (1.) As a sequence of phlebitis. (2.) In cases of morbus Brightii; and in persons habituated to intemperance. (3.) As a result of exposure to cold and rheumatism; and (4.) as an attendant of certain forms of pneumonia.

The existence of noxious and irritative matters in the blood of patients suffering from rheumatism, gout, scarlatina, typhus, and renal anasarca, as well as from that worst species of phlebitis in which purulent matter gains access to the general circulation, is now very generally admitted; and the injurious influence of fluids thus vitiated, upon the tissue of the vessels which convey them, often becomes strikingly displayed in those diseases. I have, in a previous essay*, discussed at considerable length the influence of humoral changes in determining the occurrence of acute inflammation of the arterial system. I shall therefore, in this place, merely state my conviction that, in a vast majority of instances, the idiopathic forms of phlebitis, endocarditis, and arteritis, are mainly due to vitiated and irritating conditions of the circulating fluid.

Inflammation of the large arteries has been by no means unfrequently observed in association with *phlebitis*, and several cases have been recorded in which inflammation of a distant vein has been followed by the development of a similar morbid condition in the pulmonary vessels, associated with pneumonia, generally of the lobular kind, tending to the formation of circumscribed abscesses in the lungs. Some pathologists have been disposed to regard this train of morbid actions merely as the separate results of a diseased condition of the general system, producing a tendency to the coincident development in various parts of the body of certain destructive forms of inflammatory lesion. The truth of this view has certainly never been satisfactorily disproved: it is now, however, generally considered that, in cases of phlebitis, the arterial inflammation, and the suppurative disease of the lungs, liver, and other organs which usually accompanies it, are due to the entrance of purulent fluid into the blood—an accident which not only produces a most serious vitiation of the vital fluid, but is also believed to occasion an impediment to the circulation, by the lodgment of pus-globules in certain of the visceral capillaries, which are not of sufficient capacity to permit their transit. It is now satisfactorily proved that death may rapidly ensue where purulent matter is introduced into the general circulation, especially where the pus is derived from a foul or gangrenous surface: it has also been demonstrated that, in persons the subjects of phlebitis, who have died with symptoms closely resembling those presented by the animals into whose veins purulent matter was injected by M. Lebert, pus-globules could be detected in the circulating fluid. These facts, however, while they strongly favour the opinion that, here, the inflamed state of the vessels and internal organs results directly from the entrance into the circulation of a poisonous fluid from the diseased veins, do not by any means afford

* Guy's Hospital Reports, No. 13, "Observations on Acute Aortitis."

absolute confirmation to the ingenious hypothesis that the coincident occurrence of suppuration in the lungs or liver is due to the impaction of the absorbed pus-globules in the capillary vessels of those organs. Still, the idea is so feasible, and has received such strong confirmation from the experiments of the French pathologists, that we are by no means justified in treating it slightingly.

In some fatal cases of phlebitis the inflammation is found to have extended, not only to the heart and pulmonary artery, but also to nearly the entire tract of the principal systemic arteries. In other instances, the vessels intervening between the original seat of mischief and the inflamed pulmonary artery do not present any very decided morbid change; and it would appear that there is another set of cases in which the pulmonary veins become intensely affected, while the pulmonary artery remains exempt from inflammatory change*. In a fourth class of instances, inflammation of portions of the lungs and other organs may be discovered, the pulmonary artery, as well as the veins anterior to the primary seat of inflammation, having the appearance of being entirely free from all traces of acute disease.

Acute inflammation of the pulmonary artery may supervene in cases of *phlebitis* occurring under various circumstances. As, for instance, (1.) where inflammation of a portion of the venous system occurs spontaneously as the result of some grave constitutional defect; (2.) in cases of phlebitis of the uterine veins supervening upon parturition; and (3.) after injuries and surgical operations. I shall quote briefly the principal recorded instances of the disease, as developed under each of these conditions.

It appears that in females who have long been the subjects of derangement of the catamenial function, and in whom that diseased state of the fluids which characterises the worst form of chlorosis has consequently been produced, there occasionally arises a tendency to spontaneous inflammation of the uterine veins, which usually extends merely to the communicating vessels of the pelvis and to the crural vein, producing a variety of phlegmasia dolens. The disease rarely advances beyond this point, but the following case, by M. Lediberder*, shows that it is occasionally attended with very extensive lesions in various portions of the vascular system, and that acute inflammation of the pulmonary artery may occur at some period of its progress.†

A young woman, æt. 21, of a strong constitution, who had always lived under circumstances favourable to health, and who began to menstruate at the age of 17 years, entered La Pitié, on the 6th of April, 1837, having suffered for two years from the symptoms of gastralgia and chlorosis, which had gradually increased in intensity during the last three months. On the day of her admission she was attacked with vertigo, delirium, excitement, and headache, unattended with any lesion of movement or acceleration of pulse. The cerebral symptoms gained their height in the course of twenty-four hours, remained stationary for about four days, and then diminished gradually, but had not completely disappeared by the 20th, when an attack of hemiplegia, affecting both sensation and motion on the left side, supervened. The paralysis of sensation lasted four days, that of motion six days. On the 5th of May, five days after the disappearance of the last symptom, she was suddenly seized with excruciating pain in the right lower extremity, with swelling, and tension, and dilatation of the superficial veins of the limb, in which arterial pulsation could not be felt. Besides these local symptoms, the countenance appeared sunken, the body generally was cold, and the pulse small. In the course of twenty days these new symptoms had passed off, but on the 20th of June another formidable train of symptoms suddenly made their appearance. After two days she began to suffer from pain in the left hip: in the evening, at about half-past eight, she complained of an acute pain in the loins, and went to bed. Her face was pale, and expressive of suffering. She now complained of a feeling of suffocation; she breathed high, and with great difficulty; the heart's action was very rapid and violent. She shortly fell into a state of syncope, which lasted three minutes: during this faint, there occurred a very abundant alvine evacuation. She came to herself spontaneously, and for a few moments felt relieved: the palpitation was calmed, but

* Dr. R Lee (Med. Chir. Trans. vol. xix. p. 44), and Mr. Adams (Dublin Journal, vol. xviii. p. 142), each relate a case in which phlebitis—in one instance of the uterine veins of a female recently delivered, and in the other of the basilic vein, which had been opened for the relief of puerperal convulsions—was followed by inflammation of the pulmonary veins. It is unfortunate that, in neither of these cases, is the condition of the pulmonary artery at all alluded to: but it is highly improbable that these very accurate observers could have overlooked inflammation of this vessel had it actually existed.

* Quoted by M. C. Baron, Archives Générales de Médecine, 3d Series, t. ii. p. 18.

† It appears that, in M. Lediberder's case, the state of the uterine veins, and even of the internal iliac on the right side, was not investigated; but there are strong grounds for believing that, in the majority of instances of this species of phlebitis occurring in amenorrhœal subjects, the mischief originates in that set of vessels. It is possible, however, that, in this instance, the inflammation commenced either in the crural vein, or near the bifurcation of the cava. An accident which is of by no means very rare occurrence in various forms of cachectic disease.

the dyspnœa continued. In a short time the difficulty of breathing increased; she complained of pains in the abdomen; the body grew pale, and was covered with a cold sweat; then the agitation and suffering became extreme. Sensation ceased in the extremities, she screamed, wept, and exclaimed that she was dying. The breathing became more and more difficult, until at last the patient merely gasped. At about ten o'clock she expired without agony or convulsions. On examination, there was found a very considerable softening of the left cerebral hemisphere. The heart did not present any alteration, with the exception of a marked thickening of a portion of the left auricle just above the mitral orifice. There was a large quantity of soft dark coagulum in each of the auricles; the ventricles contained less blood. The lower part of the left internal jugular vein contained a yellow fibrinous clot, of firm consistence, and adherent to the lining membrane. The coats of this vein were more than twice their natural thickness, and the surrounding cellular tissue was indurated to the extent of a line. The lower part of the vena cava contained a clot which adhered posteriorly to about one-fifth of the circumference of the vessel. This coagulum was prolonged into the veins of the lower extremity, and it was found to be more and more adherent the lower it was traced. It adhered to the entire circumference of the crural vein: it passed but a short distance into the left hypogastric and iliac veins: it was prolonged into the crural vein and its collateral vessels, and extended into the upper part of the veins of the leg. Above, the clot was rather firm, and of a dark-grey colour; lower down, it became of a more sanguine colour, was less firm, and presented here and there small cavities. Near the crural arch, it was so adherent to the vessel as not to admit of separation except by a careful dissection; the walls of the vessel were somewhat thickened, and its investing cellular tissue was rather indurated. Various bulgings appeared here and there at the points of entrance of the large collateral veins; the vessel was converted into a complete cord. The pleural cavities each contained about three ounces of reddish limpid and transparent serosity; the lungs were entirely free from adhesions, they were of a reddish purple colour anteriorly, and purple behind; they contained much air in front, but were dense posteriorly. In this situation they were œdematous. They did not contain any granular matter or tubercles; the bronchial tubes were perfectly healthy. The two principal branches of the *pulmonary artery* contained coagula which were prolonged into the tertiary divisions of the vessel, where they terminated. These clots almost completely filled the calibre of the arteries. They were of a greyish colour marbled with black, of a cellular appearance, without distinct layers, and of firm consistence; they could not be easily torn; they were unattached except in about one-sixth of their circumference. Their surface was more smooth towards the point of adhesion, and was covered by a fine membrane, which became gradually lost in the lining membrane of the vessel. Upon being stretched, this membrane separated into minute cellular filaments. The adhesions formed by the clots were more extensive on the right than on the left side; beyond their tertiary divisions, the pulmonary arteries were wide; the pulmonary veins did not contain any blood.

It may be questioned whether the train of morbid processes developed in the above case resulted directly from inflammation of the pelvic veins; giving rise, either to the direct extension of the disease along the vascular tract, or to the entrance of purulent matter into the general circulation; or whether the cerebral, venous, and arterial inflammations were not the separate results of a vitiated condition of the circulatory fluid, giving rise to a disposition to acute inflammation in various parts of the blood-vessel system. In the absence of fuller details, I am strongly disposed to adopt the latter view. The deterioration of the fluids which results in ordinary cases of amenorrhœa and chlorosis, may not generally be sufficiently extreme to give rise to organic disease of any portion of the vascular system, but it is evident that, in extreme instances of this condition, where protracted suspension of an important excretory function has given rise to a state of extreme cachexia, attended with a highly morbid state of the vital fluid, there certainly does exist a marked tendency to the spontaneous development of acute and subacute forms of inflammation in various parts of the body, and more especially in the structures of the blood-vessel system. It would, of course, be out of place to enter more fully into the consideration of this subject at present, but the point is one that deserves a most careful investigation.

The two following cases, reported by Cruveilhier,* afford the most complete examples on record of acute inflammation of the pulmonary artery, as occurring in association with uterine phlebitis following labour.

A woman, ætat. 29, of a highly nervous temperament, was delivered of her first infant, at the Maternité, on the 12th of July, 1830. Considerable hæmorrhage followed the labour. The patient had a very anæmial

* Anatomie Pathologique, Tome 1, Liv. 2e, pp. 18 et seq. Pl. 1 and 2.

appearance; her face was pale, her pulse small and frequent, and she complained of great debility. There was severe hypogastric pain in the region of the uterus, augmented by the slightest pressure. Commencing uterine phlebitis was diagnosticated. 13th. The hypogastric pain had diminished, the febrile condition continued, but in a less degree. 14th. The secretion of milk was established, (the woman suckled her infant.) On the three following days there were copious sweats, accompanied by various nervous symptoms, headache, and a tendency to syncope. On the 18th, 19th, and 20th, she was more tranquil, but the pulse remained frequent. The hypogastric pains returned, and extended over the whole abdomen. The administration of pills containing calomel and digitalis produced frequent evacuations, which were followed by relief. On the 23d she complained of very severe pain on a level with the left false ribs: she stated that she had been liable to this symptom. Examination of the chest did not give any satisfactory result. The pain was not removed by frictions and cataplasms, but yielded to a blister applied on the 24th. From the 25th of July to the 3d of August the patient appeared to be convalescing; she suckled her infant with more success than on the preceding days. On the 3d new symptoms presented themselves: oppression, cough, difficult expectoration, anxiety, nervous excitement, and extreme frequency of the pulse. There was no tenderness in the hypogastrium. Exploration of the thorax gave no very definite indications of disease; percussion developed a natural sound; auscultation afforded only a slight gurgling at the base and posteriorly. The secretion of milk was suspended. On the two following days her condition remained unimproved: the face was sunken; there was oppression and cough, with great distress; the pulse was very frequent. After vomiting and purging had been produced by an evacuant, the patient ceased to make any complaint. There was now diarrhœa. The pulse became more and more frequent: there was no expectoration. She died on the 9th, twenty-eight days after her accouchement, and six from the occurrence of the last exacerbation of her symptoms.

Examination.—In the abdomen there existed no traces of peritonitis, with the exception of slight arborescent injection here and there upon the surface of the large intestines; otherwise, the intestinal canal appeared healthy. The uterine and ovarian, and nearly the whole of the branches of the external iliac veins, resembled hard cords, being filled with dense adherent and decolourised clots. The left external iliac, and the left crural veins, with every one of their divisions, contained coagula, which were less dense and less adherent, and which were evidently of recent date. At the base of the left lung were found two small purulent cavities, circumscribed and superficial, and distinguishable through the pleura by their yellowish-white colour. In the interior of one was a little adherent slough. Many other points of the surface of the lung presented indurations, with various degrees of redness; one of these resembled a small apoplectic effusion. The posterior halves of the inferior lobes of both lungs were rendered perfectly inadequate to respiration by infiltration of sero-purulent matter. Upon incising the lung in different directions, firm concretions were found filling the branches of the *pulmonary artery*. This vessel, when carefully opened, presented a sanguine concretion, which was decolourised and slightly adherent to the parietes; it branched with the vessel, penetrating into some of the very small ramifications. It was evident that the coagulation of blood had commenced in the arterial trunk, and had subsequently extended into the smaller branches, as the coagula in the latter were red and less firm; while, by their decolourisation and tenacity, the clots in the larger branches presented unequivocal evidence of their ancient date. Upon dividing the clots*, a collection of purulent matter, which presented all the characters of phlegmonous pus, was found in the centre of the largest coagulum.

In a woman who died fifteen days after her accouchement, and who presented, during the latter days of her life, a train of typhoid symptoms, Cruveilhier found the primitive iliac vein and its divisions filled with adherent sanguine concretions, which presented various degrees of age. They were white in the principal trunks, where they contained pus in their interiors, and became red and gradually less coherent in the extreme divisions. The lower extremities did not present any marked traces of œdema. The liver was of a light yellow colour, softened, and otherwise diseased; the uterus was equally flabby, but without structural alteration. There was pleurisy of the posterior border of the right lung, with ordinary diffused and

* M. Baron considers that in cases of this description the train of lesions in the artery occurs in a manner the reverse of that described by Cruveilhier; he believes that the coagulation commences in the extremities of the arteries, and extends backwards to the larger branches: this may possibly sometimes occur, but in the generality of cases the appearances of the various portions of coagula favour the opinion of Cruveilhier. It seems that almost any part of the tract of inflamed vessel may be the first to receive a deposit of adherent coagulum. Such deposits are very liable to occur in the large branches; they then form a kind of nuclei, which, so soon as they become sufficiently prominent, are increased by further depositions both on their cardiac and distal extremities.

lobular pneumonia. The pleurisy was evidently consecutive. The lung, when divided, presented divisions of the *pulmonary artery* filled with fibrinous concretions. These deposits were white, and contained pus in their interiors: they divided and subdivided with the branches of the vessel, becoming red and scarcely coagulated in the smaller ramifications. The concretions did not exist in the small vessels in the near vicinity of the portions of lung which were affected with lobular pneumonia. In the part of the lung which was the seat of ordinary inflammation (diffused pneumonia), the tissue of the organ was infiltrated with pus, and was in the condition of grey hepatization. Cruveilhier found in this subject a very rare pathological alteration—a considerable and irregular shred of lung was isolated in the middle of this organ, and was not attached, except by a very narrow pedicle formed by the air and blood-vessels. This portion of lung did not present in other respects any of the characters of gangrene.

Where fatal phlebitis occurs as the result of injuries and surgical operations, it is liable to be associated with all the lesions of the pulmonary tissues alluded to in the above histories: acute pulmonary œdema, purulent infiltration, lobular pneumonia, the characteristic form of circumscribed pneumonic abscess, and pleurisy, have frequently been observed in these cases. In a few instances the association of these lesions with inflammation of the pulmonary artery has been clearly displayed; but, as in the phlebitis which occasionally follows an operation, the patient usually sinks very rapidly under the prostrating severity of the disease, death may occur in these instances before the sensible evidences of inflammation of the vessel—such as the deposition of adherent coagula, &c.—have become established. It is also probable that, in many cases where the morbid appearances in the lung substance have been carefully noted, an extensively diseased state of the pulmonary artery has been entirely overlooked.

The following brief narrative, also by M. Cruveilhier, affords a good example of acute inflammation of the pulmonary artery occurring in association with a peculiar form of traumatic phlebitis.

In a man who died twenty-nine hours after amputation of the thigh, which had been rendered necessary by a recent injury, Cruveilhier found phlebitis of the medullary canal of the femur, capillary phlebitis, and apoplexy of the liver, and commencing phlebitic inflammation of the spleen. The lungs contained purulent cavities, which were principally situated at the base and posterior part of the right: they were generally small, and irregular in shape, projecting from the surface of the lung, and surrounded with a red and indurated areola. The superior lobe was entirely exempt. A section of this lung discovered some abscesses in its interior, and revealed a decolourised concretion, which filled and adhered to a secondary division of the *pulmonary artery*. In this, as well as in other cases which have occurred to him, Cruveilhier found that the veins of the stump, and of all the other parts of the body, although examined with great care in their trunks as well as in their smaller ramifications, did not present any change.*

It can scarcely, I think, be doubted for an instant, that, in the whole of the above cases, the patients were the subjects of acute inflammation of the pulmonary artery. In Cruveilhier's first case, the progress of the thoracic mischief which immediately led to dissolution occupied six days; and, although it is not improbable that a certain degree of insidious inflammation commenced in the pulmonary artery earlier in the disease, when the patient first complained of pleuritic pain on the 23d of July, the fatal attack of pneumonia and arteritis did not commence until nine days afterwards. It is clear that in M. Lediberder's patient, a deposition of fibrine must have taken place in some of the obstructed portions of the pulmonary artery previously to the occurrence of the fatal attack of dyspnœa. It is probable, however, that the vessel had remained tolerably free until a very short time previously to he woman's death. Cruveilhier's third case (an indisputable example of acute pulmonary arteritis) must be taken as evidence that decolourised clots may form in this vessel within twenty-six hours; and the character of the fatal attack which destroyed Lediberder's patient in an hour and a half, together with the recent character of the changes found in tho pulmonary tissues after death, appear to me to prove almost incontestibly, that this woman was destroyed by the acute attack of pneumonia associated with active disease, superadded upon sub-acute inflammation, of the pulmonary artery. In the remaining example, (Cruveilhier's second case) the entire duration of the disease was only fifteen days, but no mention is made of the precise time at which the evidences of thoracic mischief were first presented.

It will be found, then, that, in certain cases of acute phlebitis, the patients fall into a typhoid condition almost immediately after the

* This observation, although of great value as illustrating the now established principle that fatal results may be produced by inflammation of very minute veins, does not, of course, by any means apply to all cases. The most extensive inflammation of the large veins of the stump occasionally presents itself in persons who die with typhoid symptoms shortly after undergoing amputation.

commencement of the venous inflammation, presenting the collapsed and death-stricken aspect of persons whose systems are mortally contaminated by an animal poison; here, where death occurs, as it occasionally does, within a few hours after the onset of the attack, pus may be discovered in the circulating blood, which is indisposed to coagulate; the principal viscera are found generally softened, and present partial traces of incipient inflammation of the lowest character, such as indistinct ecchymosis, mottling, and irregular vascularity; the lungs appear congested in that defined manner which indicates the earliest commencement of lobular pneumonia, or have various portions of their tissues, and perhaps their entire substance, infiltrated with serous or sero-purulent fluid. This latter condition is not to be confounded with the state which occasionally precedes hepatization in ordinary cases of pneumonia; it is not the precursor of any second stage, but is a disease *sui generis*, and is essentially fatal in its character. In these cases, examination may fail to discover clear evidences of inflammation of the pulmonary artery; still this process may have commenced although the products of inflammation may not have had time to form. Where life is protracted for some hours longer, or when the patient continues to struggle on for a few days, the liver, spleen, and lungs are usually found to be the seats of circumscribed abscesses and purulent infiltrations, and these appearances, when occurring in the lungs, are generally associated with traces of pleuritis of the most asthenic character, principally affecting the surfaces of the lower lobes and diaphragm, and may also be attended with the evidences of acute inflammation of tracts of the pulmonary artery. When the whole, or the majority of these lesions occur in association with each other, the disease is always rapid in its progress, and is probably entirely beyond the aid of human science. It will be observed that inflammation of the vessels and substance of the lungs may be set up at any stage of the progress of low forms of phlebitis;—wherever the surface of a stump remains unadherent, and destitute of healthy discharge, and so long as the tract of an external vein, not previously the seat of varix, retains the slightest traces of inflammatory action, the danger of pulmonary arteritis and of secondary abscess of the principal viscera may be seriously apprehended.

In all such cases, attention to the renal and hepatic functions is most imperatively called for. Purulent infection is commonly regarded as an ultimate pathological condition, which may chance to occur in almost any case, and which is entirely beyond the influence of preventive means. A very long and practical investigation of the subject has, however, convinced me that (except in certain puerperal cases) the extremely low and unhealthy form of phlebitis which permits the entrance of purulent matter into the general circulation, never occurs, in this country, to any but those whose constitutional powers have long been impaired by various excesses or vicissitudes, and that it is then intimately associated with a more or less diseased condition of the principal secernent and depurative viscera. Under these circumstances, it may often be impossible to ward off the threatened attack, although its onset may be clearly anticipated, and our efforts will probably be as vainly employed to resist the rapid fatality of the disease when its progress has commenced: still, in the subjects of known or suspected renal and hepatic lesion, it always remains in our power to postpone surgical operations which are not demanded by immediate necessity; or, at least, where such delay is impossible, or where injuries have been accidentally inflicted, we may still be prepared to employ measures to ward off the threatened evil, or to meet it at its earliest approach; and, however feeble our most cautious efforts may eventually prove in the accomplishment of these objects, it still remains with us to guard the patient against many circumstances that may seriously aggravate the peril of his condition. Where an individual, whose kidneys or liver have long been in a condition either of organic disease or of serious functional derangement, becomes the subject of an accidental injury or surgical operation, any circumstance which tends to produce increased vascular action in his impaired organs is fraught with the most imminent peril to his life. In one, especially, whose vital powers have recently been lowered by the shock of a capital operation, the passage of a draught of cold air over the surface of the body, or even the employment of any of those mild diuretic and chologogue medicines, which are usually prescribed in such cases, may prove the means of setting up at once a train of acute internal inflammations of the most rapid and uncontrollably destructive character.

I am fully convinced that, if surgeons would resolve to adopt previously to all operations, and in every case of injury and wound, even in those of the most apparently trivial description, the precautionary measure, which I ventured to urge very strongly some years ago,*—of carefully examining the condition of the liver and spleen, and more especially of the kidneys, the occurrence of many of those formidable inflammations of the solid viscera and of various serous and vascular structures which so often follow external wounds, might be entirely averted.

The following case, by M. Bouillaud, affords

* Guy's Hospital Reports, New Series, vol. i. "Observations on certain of the Causes of Death after Operations and Injuries in London Hospitals, with a view to their prevention."

a very remarkable example of acute inflammation of the pulmonary vessels occurring as a sequence of phlebitis in a distant part. There appears to be every reason to believe that the pulmonary artery of this patient was the seat of morbid inflammatory change. This is stated to have been so in the heading of the report*; but, by a singular oversight, no allusion whatever is made to the condition of this vessel in the detail of the case. I have thought it right, however, to quote this case, as, even if it be regarded merely as an instance of pulmonary phlebitis, it still tends to illustrate, in a very interesting manner, the pathology of the class of diseases now under consideration.

A vigorous man, ætat. 31, suffered from pains in the thighs, which were at first suspected to be neuralgic, but they were attended by strong febrile symptoms, and afterwards gave place to œdema of the lower limbs. He died of an acute pulmonary attack.

On examination, the heart was found rather contracted, its cavities containing fibrinous clots, in which no appearance of pus was discoverable. There was redness of the internal membrane of the auricles, particularly of the left; an uneven and tubercular state of the internal membrane of the aorta, without any trace of reddening. Many of the branches of the pulmonary veins were filled and obliterated by coagula of altered blood, which were friable and grumous, resembling the dregs of wine, with an intermixture of pus. In some of the obliterated veins liquid pus was found; in others concrete pus; in a third set a false membrane adhered very intimately to the interiors of the vessels. The venous tissues were evidently thickened. One of the veins which contained the false membranes appeared to extend to a purulent depôt situated in the interior of the lung. On compressing the pulmonary tissue, small drops of pus and little cylinders of altered fibrine were forced from the extremities of the divided veins. The trunks of the pulmonary veins contained fluid blood, and their lining presented a redness similar to that of the left auricle, but without thickening of the venous tissues. In the left pleural cavity was found a large quantity of citrine serum, quite limpid, but the lower part of the cavity contained abundance of shreddy (fibrinous) deposit. The lung on this side was pressed towards the ribs, and, to a certain degree, atrophied by the compression of this effusion, and covered with false membrane, which was in some places adherent, in others loose; in some spots organized, in others amorphous. About a cubic inch of the base of this lung was occupied by a depôt of matter resembling wine-lees mixed with pus: this cavity did not exhale a gangrenous odour, and all around it there existed very distinct vascular injection. The bronchial tubes and their branches did not present any morbid appearance. There was not any effusion in the right pleura; the corresponding lung was generally distended with blood, but remained crepitant. Towards its anterior edge it was marked with red spots of partial induration, and with perfectly circumscribed purulent deposits—lesions which exactly corresponded with those which it is usual to find in individuals who die after capital surgical operations. Carefully dissected, the portions of lung which were altered in patches, or small indurated masses, offered all the anatomical characters of incipient inflammation. The tissue was easily broken down, as in hepatization, and the smaller vessels contained fibrinous coagula. In every one of the indurated masses infiltration of pus had commenced. Only one of the purulent collections was situated somewhat deeply in the substance of the lung. The bronchial tubes on this side were normal. There was remarkable distension of the inferior vena cava, about the spot where it receives the hepatic vein, where it was filled with coagulated blood. The surface of the concretion which distended it was covered with a material which appeared to consist of blood mingled with pus. Upon the internal surface of the vein there was a false membrane more or less adherent, which, in the spots where it was examined, was white or yellowish, or more or less deeply discoloured by blood: it was of the consistence of the false membranes which were found in the pleura. The presence of this false membrane was the principal cause of the thickening which the walls of the vein presented. Similar lesions extended into the iliac veins, and throughout all the deep veins of the right lower extremity, many of which contained pure pus, but without appreciable reddening of their lining membrane, which was only less transparent than in the natural condition. Many small depôts of pus existed around the obliterated and inflamed veins. A very large abscess was found in the muscles of the calf of the leg. The nerves of the posterior part of the leg appeared to be hypertrophied. The subcutaneous veins were healthy. The veins of the left limb were less affected than those of the right, and were permeable, although the iliac vein on this side participated in the affection of the vena cava and of the veins of the right limb. The vena porta was healthy. Little purulent collections were found in the cellular tissue surrounding the bladder, and could be traced to adjacent divided veins.

* The words used are, "Fausses membranes dans les divisions de l'artère pulmonaire."

The bladder was contracted, and contained a very small quantity of urine, together with a remarkably elongated calculus developed upon one of the ends of a hair pin, which, in three-fourths of its length, was not covered by any earthy deposit. The lining membrane of the bladder was remarkably thickened, presenting spots of capillary injection, and a roughened and papillated surface. Upon compressing the kidneys urine mixed with pus escaped.* The liver was healthy; the spleen was softened: its substance, of a reddish-grey colour, broke down under the slightest pressure, appearing to be infiltrated with pus and blood.

Whether purulent absorption had or had not occurred in the above case, it appears evident that a destructive form of inflammation was directly propagated from the inflamed veins to the heart and pulmonary vessels and tissues. It is probable that the phlebitis originated in the veins of the diseased bladder; and the extension of the irritation from that organ to the kidneys may have been the circumstance which determined the occurrence of such extensive asthenic inflammation in a person whose constitution had hitherto been good.

In some observations which I published in 1841 on acute aortitis† I had occasion to remark upon the tendency which the lining of the aorta has, in common with most of the true serous membranes, to suffer from acute and sub-acute inflammatory changes in persons of shattered constitution and intemperate habits; and especially during certain stages of Bright's disease of the kidney. I then stated, and subsequent inquiry has confirmed the observation, that by far the larger proportion of cases of acute inflammation of the aorta occur in patients of this description, whose principal excretory and secerment organs have long been undergoing insidious morbid change. Acute aortitis is, however, by no means a frequent complication of renal anasarca, although, in cases of this description, the entire tract of the systemic arteries almost invariably presents manifest traces of sub-acute inflammatory lesion. As regards the pulmonary artery, I am not acquainted with any perfectly demonstrative case in which a patient, the subject of any form of Bright's disease, has died from acute and general inflammation of this vessel; although it is by no means unusual to find adherent coagula of recent formation in portions of the artery where death has occurred from renal anasarca and its complications. In the next chapter, however, I shall have to refer to numerous instances where an apparently less active or sub-acute form of inflammation was found to have invaded the pulmonary artery in persons suffering from morbus Brightii. At present, I must be content with stating my belief that the vessel is liable to be attacked with inflammation of the most acute character in cases of this description.* That such disease of the artery is apt to occur in persons of highly intemperate habits is proved by the following narrative. Nothing remark-

* Unfortunately no further particulars are given respecting the true state of these organs; but the fact mentioned above goes far to prove that their pelves, at least, were in an inflamed constitution.

† Guy's Hospital Reports, vol. vi. pp. 312, 322.

* The following is an interesting example of inflammation of the trunk of the pulmonary artery occurring in association with a certain amount of renal disease. The opinion that these lesions existed in the relation of cause and effect may perhaps not be generally adopted. It is true that the congenitally faulty state of the right ventricle and its appendages must have rendered those parts in some degree liable to suffer from further impairment in consequence of comparatively slight causes of constitutional or local irritation. Still, I am inclined to believe that the inflammatory mischief which was set up in this vessel almost immediately after the occurrence of an external injury, was very probably determined by the faulty condition of the renal apparatus.

A milk-boy, ætat. 15, was admitted into the Royal Free Hospital, Feb. 20th, 1847. On the 12th he had bruised his left knee by a fall, and had since suffered from constant pain in that joint, and also, for two or three days before admission, in the right knee. On the day after the accident, he began to experience pain in the left side of the chest, and difficulty of breathing. When admitted he was much collapsed, and the extremities were cold and livid. The cheeks were of a deep purple colour, and the lips blue. The pulse was 124 in the minute, and extremely feeble. The tongue dry, and covered with a whitish fur. The respiration was peculiarly rapid and panting, and he was compelled to lie on the back, partly inclined to the right side, and with his head low. He complained of pain in the region of the heart, palpitation, and difficulty of breathing. Both knee-joints were swollen and tender, and there existed a red and swollen patch over the left trochanter. The respiration was puerile, and was attended with occasional mucous râles. As I shall have to refer to the state of the heart's actions, as ascertained by Dr. Peacock, in a subsequent part of this memoir, I shall omit them here. He gradually sank and died at eight o'clock on the following morning. The boy was much exhausted, and his intelligence so impaired, that it was impossible to collect from him any satisfactory account of his previous state of health. It was ascertained, however, that he had always been of a livid complexion, but was stout, healthy, and capable of a full amount of exertion, until February 1846, when he was thrown from a cart, and sustained concussion of the brain. He continued under treatment six days: the only peculiarity then observed in his appearance was slight lividity of the lips. Since that time he had been gradually getting thinner and weaker; he was constantly chilly, and very subject to take cold. He complained occasionally of palpitation, difficulty of breathing, and pain in the region of the breast; and his hands and face were always very blue, but especially so in cold weather, or when he was suffering from affection of the chest. His appetite was generally defective, and he occasionally vomited his food. His father is of a livid complexion, and has a "pigeon breast."

On examination, the brain was found healthy,

able was observed in this patient's abdominal viscera, but it must be strongly doubted whether, in a man of his advanced age and irregular life, the kidneys and liver could possibly have retained their natural integrity of structure and function. A workman, æt. 65, who had always enjoyed good health, though a great brandy-drinker, had been affected latterly with cough and mucous expectoration, which, however, did not prevent him from following his occupation. After working one day in the water, he was seized with shivering, pain in the side, and dry cough. He tried some remedies at home, and then applied to Dr. Hankel. On the 2d of June, he presented the following symptoms:—His countenance was pale and disturbed; the eyes prominent and brilliant; a short and dry cough; pain, and a sensation of compression about the middle of the chest; there was pain from dyspnœa, particularly at night, preventing him from sleeping; continued thirst; anorexia; white tongue; hot skin; pulse 100; respiration accelerated; gait slow and insecure. He had been eight days affected in this manner. There was an increase of fever during the night of the 2d. On the evening of the 3d, the agitation of the patient was much increased; he had dyspnœa; frequent cough, without expectoration, and continual desire to pass water; the countenance was more fallen, and the pulse weaker. In the afternoon of the same day his agitation was redoubled, and he got out of bed several times: at five in the evening, his anguish was so extreme that he left his room, complaining in a tremulous voice of his painful state, and wished to move to a room in the upper story for the sake of fresh air; his wife hearing him go hastily up stairs, ran up after him: he had just time to throw himself on a bed, was attacked with a slight rattle, and died on the instant. On examination, the eyes that, during life, had been prominent were now sunken. The lungs (marbled white and blue) did not fill the thoracic cavity completely. They appeared to the touch engorged with blood. On cutting into them, there flowed a moderate quantity of blood not very deep-coloured; the tissue of these organs felt sufficiently elastic; there existed old adhesions at their lateral and posterior parts. The heart was not distended with blood; it contained some concretions resembling polypi, particularly in the right ventricle; the lining membrane of the heart, that of the arch of the aorta, and its valves, were healthy. The *pulmonary artery* contained similar concretions: one of them was remarkably large and firm, but did not adhere in any part. The parietes of this artery were of a uniform intense redness, as if they had been rubbed with black-cherry juice. This redness could not be removed by washing, nor by scraping with the handle of a scalpel; it penetrated the whole thickness of the lining membrane of the vessel: the capillary vessels were not injected. This red tint did not extend to the right ventricle nor to the lungs. The other large vessels presented nothing similar. There was nothing remarkable either in the cranium or abdomen.* Notwithstanding the fact that, in this case, the coagula which were found in the pulmonary artery remained unadherent to the parietes, the character of the symptoms, and the absence of any very extensive inflammatory or obstructive lesions in the lungs and heart, appear to me to afford convincing evidence that the individual perished from acute inflammation of the vessel producing coagulation of blood in its interior, and consequently fatal obstruction to the pulmonary circulation.

Acute inflammation of the trunk and branches of the pulmonary artery occasionally presents itself as an idiopathic dis-

though much congested. Both lungs were engorged with blood, sparingly crepitant, and contained several masses in the state of pulmonary apoplexy. The heart weighed ten ounces; the foramen ovale was closed; the sinus of the right ventricle was divided from the infundibular portion of that cavity by a thick muscular septum, defective only at its centre, over a space of sufficient size to admit the forefinger. The cavity thus formed communicated with the aorta by an orifice three lines in circumference. The infundibular cavity of the ventricle gave origin, as usual, to the *pulmonary artery*, the orifice of which was very small, and was provided with only two valves, which were extremely thick and opaque. The coats of the artery were much indurated and thickened, and its canal was entirely obstructed by fibrinous coagula. At the sides of the vessel these coagula were of a dirty-white colour, and were laminated and firmly adherent to the valves and diseased lining membrane; but towards its centre they were softer and less discolorized. The obstruction occupied the whole trunk of the vessel, and extended a few lines in each direction. The distal branches were free from disease. The ductus arteriosus displayed a conical cavity, extending two or three lines from the bifurcation of the pulmonary artery: beyond this it was impervious. The pulmonary veins were natural. The left cavities of the heart were small. The aorta communicated with the left ventricle by an orifice of about the same size as that by which it arose from the right ventricle. The aorta was large from its origin to the sulcus marking the attachment of the arterial ligament: from that part its calibre greatly decreased. Its valves and tissues were normal. The bronchial and œsophageal branches were somewhat large. One of the kidneys was somewhat mottled. It appears probable that in this case disease was set up in the malformed pulmonary artery shortly after the occurrence of the first accident, but there is every reason to believe that acute mischief again attacked the already faulty and obstructed vessel some days previously to the boy's death.—*Medico-Chirurgical Transactions*, vol 30, p. 132. I regret that I have been compelled to abridge some of the details of this important case, which, with the observations appended by Dr. Peacock, are of great interest.

* Archives Générales, August 1834; from Rust's Magazin, t. xxxiv. Part II. p. 244.

ease in persons of apparently healthy constitution who have suffered from exposure to cold and wet. It is probable that in such instances the disease partakes of a rheumatic character; it is true that, rare as it is to meet with the evidences of acute inflammation of the pulmonary valves in cases of rheumatic fever, it is still more unusual to observe either pulmonary arteritis, or acute aortitis, during the progress of that malady. It appears, however, that, in common with nearly the whole of the true serous membranes, the lining of the trunk and branches of the pulmonary artery is occasionally liable to become inflamed in persons of rheumatic diathesis. I shall hereafter have to cite a case illustrative of this fact. In the following highly interesting example the almost general inflammation which attacked the vascular system of the unfortunate patient was probably of a rheumatic character; but the details are not sufficiently precise to enable us to decide that such was certainly the case.

M. Meli has recorded* the case of a mariner, æt. 24, tall, of strong constitution and sanguineous temperament, who had spent three days in a very cold snow-storm in a vessel where he was mostly engaged at the pump. He was violently chilled, and was afterwards attacked with intense fever, attended with much heat, thirst, great headache, sense of constriction in the throat, and great anxiety. The skin was the seat of a sense of burning, which passed into itching. On the 17th of January (the third day of his disease), he applied to M. Meli, who found the face flushed, the eyes suffused, and the pulse hard and tense. The patient complained of pain in the throat, difficulty of swallowing, and rheumatic pains, which were felt sometimes in the chest, and sometimes in the abdomen†. The patient lost some blood, and laxatives and antimonials were prescribed. Though, on the 18th of January, the patient was better, yet, on the 19th all the febrile symptoms were more intense. The skin was in several places red, and in others presented effusions of blood as after beating; the surface was painful, especially in the region of the larger venous trunks; and the slightest touch or pressure aggravated the pain‡. The pulse was very rapid, vibrating, and, in the left arm, dicrotous. The patient complained of great thirst, anxiety, throbbing headache, and strong pulsation over the whole body. The blood drawn on the previous day was covered with a thick buffy coat; the serum was very scanty, and the clot was firm and tough. As M. Meli considered, from the quick and violent beating of the arteries, and the redness and tenderness in the course of the veins, that the disease was inflammation of the vessels, he ordered another bloodletting and the use of foxglove. 20th.—The patient was somewhat better, but his night had been rather restless. The symptoms also continued, and on the 21st the extravasations on the skin were more numerous and larger; pure blood was discharged from the throat, and blood and bloody mucus from the bowels. The symptoms persevered, notwithstanding the use of local and general blood-letting and foxglove, with laxatives, until the night between the 29th and 30th, when the patient had a violent attack of shivering. He became very restless and anxious, and was convinced of the near approach of death. Dr. Meli found him with shrunken features and dark blue complexion; the pulse was intermitting, tremulous, and scarcely perceptible; the abdomen was tense and burning; the extremities cold. The patient soon afterwards expired, with violent and agonizing oppression. The body was examined on the two following days. It was emphysematous, and in different parts of the cellular membrane reddish serum was effused, and there were ecchymoses. The nostrils and rectum contained much blood. All the superficial and deep-seated veins of the lower extremities were filled with blood, and their walls were hard, thickened, and red-coloured internally. The arteries were in the same state. The trunks of the femoral veins and arteries were highly inflamed, thickened, and solid; and their coats were covered with networks of capillaries filled with blood. Their interiors were lined with layers of adherent plastic lymph, which, in different points, presented consistent purulent matter. The abdominal cavity was filled with much reddish serum, containing blood-clots. The peritoneum was much reddened, and covered with numerous fine red vessels. In several parts of the intestines and mesentery were ecchymosed spots. All the arteries and veins of the abdomen were reddened, and felt hard, as if they had been injected with the usual injection matter; and externally and internally plastic lymph was found effused. In several places they were closed, and united by the effusion of lymph; and in few places did they contain blood. In the interior wall of the aorta, two inches above its division into the iliac arteries, there was found an opening smaller than a lentile, but no laceration was seen in the trunk. The arterial walls were swelled

* Journal of Omodei, and Tiedemann's Von der Verengung und Schliessung der Pulsadern in Krankheiten, and Edinburgh Medical and Surgical Journal, vol. lxiv. p. 142.

† There is no reason to believe that these thoracic and abdominal pains were in reality rheumatic; they were doubtless attendant upon the acute disease of the great blood-vessels which was then in progress.

‡ A remarkably sensitive condition of the surface of the body has been observed by Dr. Bright in certain cases of acute arteritis.

and much thickened, the calibre of the vessel was diminished, and the inner coat was of a brownish red colour. At the point where the cœliac artery arises from the aorta there was a circular cording of the vessel, which extended into its division into the gastric, hepatic, and splenic arteries. This consisted of effused plastic lymph, which resembled a false membrane, and could be detached by the handle of the scalpel. The inferior vena cava was in like manner inflamed, much contracted in calibre, and to its inner coat were attached patches of false membrane which resembled valves. The part of the vena cava ascending behind the liver contained purulent matter. The arteries and veins of the mesentery were inflamed, hard, and red, and the vessels of the intestinal mucous membrane were much distended with blood. A similar condition of the blood-vessels of the stomach, liver, spleen, pancreas, kidneys, urinary bladder, and sexual organs, was observed. Their inner membrane was covered with thick fine networks of capillary vessels. The arteries and veins of the upper extremities were greatly inflamed, and quite in the same state as those of the lower. The *pulmonary arteries* and veins were also inflamed, especially the trunks, though less than the arteries and veins of the abdomen. The walls of the bronchial and œsophageal arteries were firmer and thicker than usual, and their inner membranes were inflamed. The coronary arteries were inflamed throughout, as also were the coronary veins; these vessels were externally covered with lymph which had exuded from the cellular membrane. The walls of the auricles and ventricles, and also the trunks of the arteries and veins, were covered with numerous capillary vessels. The inner surface of the left ventricle was lined with a coating of lymph which stretched over the columnæ carneæ, and partly hung free into the cavity of the ventricle, and the layer extended into the left auricle, and into the beginning of the aorta. The inner surface of the superior cava was lined with coagulated lymph. The inner surface of the throat was reddened, the velum was swelled, the uvula enlarged, and the whole surface was covered with numerous capillary vessels. Some clots of blood adhered to the mucous membrane. The vessels of the brain and its membranes also presented marks of inflammation.

The above case is probably almost unexampled in the extent and severity of the vascular lesions which it presented. It can scarcely be conceived that such an extraordinary amount of disease in structures which are by no means remarkably liable to be attacked with idiopathic inflammation, could have occurred simply as the effect of long-continued exposure to cold in a perfectly healthy individual. It is evident that this patient must have been previously the subject of some grave constitutional defect; such as the vitiated condition of the fluids which precedes the development of acute rheumatic disease, or the faulty state of the blood which attends the worst forms of sea-scurvy. Several of the morbid appearances observed in this case were not dissimilar to those usually met with in extreme instances of scorbutus; and it appears to be far from improbable that, in a young and originally vigorous man, who had been previously exposed to the ordinary causes of scurvy, the endurance of extreme cold and fatigue would excite diseased actions similar to those from which this individual perished. It is evident, however, that, whatever may have been the real origin of his disease, the man's death was immediately due to inflammation of the pulmonary artery, and the consequent obstruction to the circulation through the lungs.

Considerable stress has been laid by authors upon the occurrence of inflammation of the pulmonary artery, and fatal obstruction of its branches in cases of *pneumonia*. It is evidently considered that these lesions have occurred in the relation of cause and effect: my own observation has not led me to adopt this view, but, before arguing the question further, it may be as well to adduce the principal facts upon which the opinion usually held is founded.

M. Cruveilhier speaks of having found clots in the pulmonary artery in some cases of diffused pneumonia involving one-half or two-thirds of the lung.*

The following case has been expressly referred to as an instance of fatal obstruction of the pulmonary artery occurring as a consequence of pneumonia. It was communicated by M. Richelot to the *Société Medico-Pratique of Paris*, and has been briefly quoted by M. Baron in his valuable essay on obstructions of the Pulmonary Artery, already cited (p. 17). Mr. T., solicitor, ætat. 38, general health good; had been affected with a slight cough for the last twenty years; the chest, carefully examined, presented nothing extraordinary; hæmoptysis from time to time; dyspnœa on walking quickly. After a long journey on the 1st November 1837, he was seized with violent pain in the epigastrium, which yielded to the application of leeches, and other general remedies. 8th.—Hæmoptysis of pure red blood, dyspnœa; venesection was proposed but refused, and the patient continued his wonted avocations, though advised to refrain; a day or two after he presented all the symptoms of pleuro-pneumonia on the right side. Auscultation and percussion were not performed, from the refusal of the patient. Copious venesection, and the usual remedies, were had recourse to with success: nothing remained but slight dyspnœa. When convalescent, he was all at once attacked with approaching suffocation, and fainted away; having recovered his senses, he indicated the epigastrium as the seat of his suffering. This amelioration did not last long, for the feeling of suffocation returned: about half a pound of blood was drawn with difficulty from the arm; the face became livid; there was violent tenesmus; agitation, followed by prostration and death. *Autopsy, 32 hours after.* The parietes of the thorax presented the dark-red colour peculiar to asphyxia; the pericardium contained about three ounces of a sanguinolent serosity; heart normal, its cavities contained a small quantity of clotted blood; the left lung presented adhesions of old date; about two pounds of reddish serosity in the right pleura; a few fragments of the lung thrown into water floated on the surface; both lungs were of a blackish-red colour posteriorly and inferiorly, but no *engouement* nor hepatization. "The principal branches of the *pulmonary artery* were nearly obliterated by clots of black bood, several of which adhered slightly to the walls of the vessels, were soft in their centre, and surrounded by a portion of a somewhat lighter colour; the blood was perfectly liquid everywhere else." Cranium and abdomen in the normal state*. Dr. Richelot considered that death was produced by syncope, owing to cessation of the circulation caused by the obliteration of the pulmonary artery by clots of blood. We have therefore reason to conclude that the patient died *suddenly*.

Dr. Graves has recorded† the case of a man, ætat. 60, who came under his care with pneumonia and partial solidification of the right lung, with a complete absence of fever. The disease remained stationary for about three weeks, then there occurred a sudden change for the worse, and the patient died in about 26 hours. On examination it was found that, in addition to the solidification of the lower two-thirds of the right lung (the left lung was entirely free from pneumonic lesion), the pericardium was distended with a very abundant straw-coloured fluid. The membrane was in every way healthy. The heart felt very soft, and lay collapsed; its structure was pale, but otherwise normal. The *pulmonary artery* was occupied by a fibrinous clot, which presented the usual division produced by the branches of that vessel. There were only two valves, and these were coated with a recent deposition of lymph, in some situations almost a quarter of an inch thick. The valves were much thickened and opaque, contrasting in a remarkable manner with those of the aorta, which were quite free from disease. The lining membrane of both the pulmonary artery and the aorta presented its usual appearance, as did also the endocardium. There was some calcareous deposit on the tricuspid and mitral valves, but not more than is frequently observed in subjects of the same age. There was no anasarca or effusion into the pleuræ or abdomen. The hydropericardium and inflammation of the pulmonary valves had not been expected during life. Both had evidently arisen within thirty hours of the patient's death, contemporaneously with the sudden exacerbation of the symptoms.

These cases, together with many of the foregoing instances, certainly prove that acute pulmonary arteritis is likely to occur *in association* with various inflammatory conditions of the lungs and their appendages, but I do not consider that these or any other cases with which I have met, by any means establish the principle that pneumonia, in any of its forms, is liable to produce acute inflammation of the pulmonary artery. It is true that in some cases where very large portions of the lungs become consolidated, producing death from mechanical obstruction to the pulmonary circulation, branched coagula are found traversing the whole of the ramifications of this vessel, but these clots are always unadherent, and unattended with any of the marks of arteritis, and are evidently merely the effects of slow coagulation of blood in the vessel during the protracted agony of death from gradual suffocation. In none of the cases which have been narrated above was there reason to believe either that the pulmonary obstruction had been sufficient to determine the occurrence of coagulation in the affected vessels, or that inflammation had been propagated from the diseased lung substance to the arterial tissues. It was certainly noticed in Bouillaud's case that one of the inflamed veins passed from the near vicinity of a purulent cavity; but it will be recollected that in one of Cruveilhier's cases of lobular pneumonia,* it was distinctly mentioned that clots did not exist in the small vessels in the near vicinity of consolidated lung. In M. Richelot's case, so far from there being sufficient pulmonary obstruction to account for the extensive coagulation which occurred in the vessels, it

* Anatomie Pathologique, T. i. Liv. xi. p. 20.

* As cited in the Medical Times, vol. xii. p. 122, May 17th, 1845.

† Dublin Journal, vol. xxii. p. 388, and Clinical Medicine, p. 204 (a few particulars are given in the former account which are not found in the latter).

* Second case, p. 838.

does not appear that any portion of the lung was in a state of consolidation, nor is it by any means certain that this patient had ever been the subject of pneumonia at all: in this case the pleuritic effusion was large, but it existed only on the right side, while the vascular obstruction appears to have occurred equally in both lungs. In Dr. Graves' patient, also, the pneumonic consolidation was entirely confined to a part of one of the lungs, while a portion of pulmonary tissue, amply sufficient to admit of a tolerably free circulation, was found in a perfect respirable condition.

It has been argued that pneumonia is essentially dependent upon an inflamed condition of the smaller vessels of the lung. The opinion of Cruveilhier that lobular pneumonia is intimately connected with capillary phlebitis of these organs, may apply in certain instances; and, indeed, in the case of inflammation of any structure, it is, of course, impossible to deny that its capillary vessels are principally involved in the morbid process. Still, I am confident that pulmonary arteritis is by no means a frequent accompaniment of pneumonia, and that it still less frequently exists as a *result* of that lesion. I have examined numerous cases of pneumonia in every stage, and, I believe, in every form of the disease, and have failed to discover either the faintest trace of acute lesion in the wider pulmonary branches, or the slightest appearance of adherent coagula in the smaller ramifications. So generally, indeed, has this been the case, that I am in the habit of suggesting that recent pulmonary apoplexy may be distinguished, at a glance, from circumscribed red hepatization, by observing that, in the former disease, some of the arteries which pass into the consolidated portion of lung, are always obliterated by coagula; while in the latter the vessels are, as far as I have observed, almost invariably pervious. Particular allusion has been made by Mr. Paget* to the fact, that he has often found the branches of the pulmonary artery blocked up by clots formed during life in those who die of great *œdema of the lungs*.† There can be no doubt that certain of the cases which produce pulmonary œdema, have also a tendency to determine the formation of adherent clots in the pulmonary vessels: thus the two cases‡ which Mr. Paget cites in illustration of his principle, were examples of confirmed morbus Brightii, in which it was probable that the disease in the pulmonary artery and the œdema of the lungs, were merely the associated results of the condition of renal anasarca from which both patients suffered, if, indeed, the obstruction to the pulmonary vessels was not the older lesion of the two. I have frequently traced out the course of the pulmonary artery in examples of œdema of the lungs arising from various causes, and have been led to conclude that, as a general rule, the vessels remain perfectly free from all traces either of recent inflammation or of fibinous deposit, as far as they can be traced by ordinary dissection. More or less extensive obstruction of branches of this vessel has been observed as a usual attendant of *gangrene of the lung*:—it may probably occur either as a consequence or as a cause of that lesion. That complete obliteration of the vessels leading to the sphacelated part is not invariably present in these cases, is, however, proved by the fact, that fatal hæmorrhage has been known to occur from extension of the gangrene to a large pulmonary vessel which had remained unclosed.

The details of the following instance of acute pulmonary arteritis are too incomplete to be of any pathological value; but, as the case appears to afford a rather striking example of this disease, I shall quote it for the benefit of those who may have access to the work in which it originally appeared.

M. Tiedemann quotes* the case of a laundress, in whose body, after death, clots were found to extend from the right ventricle into the *pulmonary artery* and its branches, adhering to their walls, while the inner membrane was rough and flocculent, and presented in various parts small whitish specks, to which the coagulated blood adhered. In various spots, also, could be detected membranous shreds, which resembled coagulable lymph.

Kreysig and Otto have observed the interiors of the heart and blood-vessels bearing traces of acute inflammation in certain cases of children who have died from the exanthemata, scarlet-fever, measles, and small-pox. In many persons destroyed by the last-mentioned disease, Taunton noticed the traces of arterial inflammation in various stages and degrees. The same phenomena are observed in the arteries in some cases of repelled cutaneous eruptions.† My own observation does not furnish me with any case of the above description in which acute inflammation of the pulmonary artery has been discovered after death. Still, it is probably occasionally present, especially where the patients are cut off rapidly by the sudden occurrence of extensive thoracic inflammation.

* In his highly interesting papers on "Obstructions of the Branches of the Pulmonary Artery." Medico-Chirurgical Transactions, vols. 27 and 28.

† Loc. cit., p. 167.

‡ Cases 2 and 3.

* From L'Esperance, Mars 15, 1845.

† Review of Professor Tiedemann's work on "Arctation and Closure of Arteries in Disease." Loc. cit., p. 422.

SUB-ACUTE INFLAMMATION OF THE PULMONARY ARTERY.

The pathology of the class of cases which now falls under our notice is involved in considerable obscurity; it must, however, be admitted that the view entertained by more than one able pathologist by whom the subject has been discussed, is opposed to the conclusion which I have arrived at, namely, that in these, as in all other instances where *adherent* coagula are found obstructing the pulmonary artery, or, indeed, any other portion of the blood-vessel system, the formation of such deposits has been attendant upon a certain amount of inflammation in those portions of the vascular tissues.

A highly valuable series of cases of obstruction to the pulmonary artery was published by Mr. Paget in 1844 and 1845*. In the whole of the instances detailed by that gentleman, the cause of the coagulation of blood which was found to have occurred in branches of this vessel is ascribed either to (1) obstruction to the circulation through the pulmonary capillaries, (2) to extreme languor of the circulation, or (3) to the existence of some morbid state in the constitution of the blood itself, tending to increase "that adhesion of it to the walls of the vessels which constitutes, even in the healthy state, the greatest resistance which the heart's power has to overcome."†

Dr. Dubini, who has also investigated this subject, disallows the supposition that an inflammation (like phlebitis) of the obstructed vessels determines the formation of coagula in the pulmonary artery; and suggests that the cause of the coagulation during life is in the altered crasis of the blood: still, he thinks that there are many facts in opposition to this theory, "which," (he rather *naïvely* adds), "destroys every hope of success in the explanation of the genesis of the clots."‡

It is undeniable that the whole of the causes alluded to by these authors are frequently observed to have had considerable influence in determining the formation of the deposits in question; but that they are, under any circumstances, capable of occasioning the formation of *adherent* clots in the pulmonary vessels, quite independently of a certain amount of inflammation in the vascular tissues, can scarcely be admitted. Long delay to the pulmonary circulation, resulting from any mechanical obstruction to the access of blood to the systemic arteries, is evidently liable to produce more or less inflammatory lesions in the afferent vessel of the lungs, in consequence of its unnatural distension by an undue quantity of carbonized blood. In cases of fever, and in other diseases of an extremely asthenic character, the heart's action may become so greatly depressed as to favour the deposition of adherent coagula in various portions of the vascular system, but my own observation has not furnished me with any instance of this description where there was not full reason to believe that the vessels so affected had also been the seats of previous inflammatory lesion. It is now generally understood by pathologists that in patients suffering from extremely vitiated states of the circulating fluid, the causes which lead to death are almost invariably attended with inflammatory lesions of various tissues, characterised by the depositions of unhealthy fibrinous deposits in the affected parts. The influence of various humoral changes in determining the occurrence of asthenic inflammation, especially in the serous and vascular tissues, has long been recognized, and in any case where the blood is admitted to have been in a sufficiently diseased state to admit of its spontaneous coagulation in its proper vessels, we are, I think, fully at liberty to assume that the quality of the fluid was also sufficiently depressed and irritating to produce inflammation of the internal tissues of any portion of the vascular circle.

It is highly probable that, under any of the above circumstances, *non-adherent* clots may be formed previously to death in the vessels either of the pulmonary or systemic circulation; but it is certainly altogether opposed to the ordinary principles of pathology to maintain that coagula are capable of becoming adherent to any portion of the interior of the vascular tract, independently of a certain amount of inflammation of the arterial or venous tissues at that spot.

There are various forms of disease in which adherent coagula are liable to form more or less gradually in various parts of the afferent vessel of the lungs. Upon the grounds already stated, I have always attributed the production of these deposits either to the occurrence of acute inflammation in small portions of the vascular tract, or to the establishment of a more gradual or sub-acute inflammatory process in those situations. These circumstances are found to occur (1) in various instances of morbus Brightii; (2) apparently also in some few cases of rheumatism; (3) as well as in a very large proportion of that diversified class of diseases in which the pulmonary tissues

* Medico-Chirurgical Transactions, vol. 27, p. 162, and vol. 28, p. 253.

† Mr. Paget does not by any means deny the occurrence of coagula in the pulmonary artery in association with inflammation of that vessel; but it is necessary to mention that the case which I shall quote from his paper as an example of sub-acute inflammation of the pulmonary artery, does not appear to be regarded by the author in that light.

‡ As quoted by Mr. Paget from the "Annali Universali di Medicini, di Febbrajo. 1845."

become occupied by spontaneous apoplectic effusions. The whole of these cases are of sufficient pathological importance to call for a separate consideration.

Sub-acute inflammation of the pulmonary artery in cases of morbus Brightii.—The tendency to the formation of adherent coagula in the emulgent veins, and in many other portions of the blood-vessel system, in severe forms of Bright's disease of the kidney, has long been observed: these lesions are usually found in association with inflammatory effusions into one or more of the serous cavities, and both sets of organic changes are known to be alike due to a highly diseased and irritating condition of the circulating fluid.* The influence of this cause in producing cardiac and aortal disease cannot by any means be denied. And wherever adherent coagula are found in any portion of the pulmonary artery of patients who have died from renal disease and its complications (independently of the presence of any very distinct *previously existing* cause of obstruction to the circulation through the lungs),† it may, I submit, be confidently assumed that the affected portions of the artery have participated in the general tendency to inflammatory lesion of the serous and vascular tissues which characterises this disease.

The following case, which occurred within my own observation about ten years since, affords a striking example of the occurrence of inflammatory disease in the pulmonary artery, and in several other vessels in association with confirmed renal degeneration.

A stout labourer, aged 32 years, was admitted to Guy's Hospital with general anasarca, and slight ascites, of about a month's standing. His whole body was highly œdematous, the heart's action inordinate, and an abnormal sound was very distinctly audible over the region of the aortic valves. He suffered from intense dyspnœa, and frequent cough, with bloody expectoration: the urine contained albumen. The difficulty of respiration continued to become more and more urgent until his death, which occurred seven days after his admission. Upon examination after death, the left pleura was found entirely lined with a firm spongy layer of bloody fibrine, and filled with reddish serum. The lung contained three apoplectic clots of large size; and was compressed and rendered useless by the fluid in the pleura. The right lung was extensively hepatized, but contained air. Many branches of the *pulmonary artery*, in both lungs, were filled with coagula, apparently of several days' standing. The bronchial membrane bore traces throughout of acute inflammation. The heart was considerably enlarged; its right cavities were distended with blood, and a cystiform concretion of pale fibrine, about the size of a filbert, adhered within the right auricular appendix. The left ventricle was dilated, and slightly hypertrophied; the aortic valves were rather thickened and contracted, and one of them was completely filled with a globular pellet of firm coagulum. Portions of the aorta had small granular clots adhering to their surfaces: other parts of this vessel were reddened, and the lining membrane of its great sinus was raised by a few semi-transparent deposits. The structures of the liver and spleen were hard and contracted; the kidneys were enlarged, pale coloured, and easily lacerated: and in one of the emulgent veins I found fibrinous coagula, evidently of long standing.*

There may exist some doubt with regard to the true cause of the pulmonary apoplexy, and attendant lesions of the pulmonary artery, in this case. My own belief is, that they did not depend upon aortal obstruction, but were the results of inflammation and softening of branches of the artery occurring in coincidence with extensive inflammation in other parts of the vascular system.

* Mr. Paget has advanced the hypothesis that, in cases of morbus Brightii, "the diseased blood adheres more than healthy blood does to the walls of the vessels; the urea, or some other retained constituent of the urine, acting in the same manner, as experiments have shown that salts of baryta, and some other bases, will act, which, when introduced in even very small quantities into the blood, hinder or altogether prevent its passage through the small vessels, although the heart be acting with full force." This theory by no means satisfactorily supports the doctrine of the spontaneous coagulation of blood in the vessels of these patients. The most prominent and characteristic physical change which the blood undergoes in cases of renal anasarca is an unusual degree of fluidity, and a marked inability to coagulate firmly; hence the great liability of these patients to spontaneous hæmorrhages, and the danger which they experience from uncontrollable bleeding after operations and injuries. It is true that clots are more liable to form in the vessels of patients suffering from disease of the kidneys, and of the other depurative viscera, than in any other class of cases whatever, but this arises, not from a greater tendency in the blood to become coagulated, but from a greater liability in the tissues of the blood-vessel system to become the seat of severe inflammatory lesions.

† In the generality of cases of advanced renal disease, it is evident that the circulation is much interfered with in consequence of capillary obstruction in the various organs of the abdomen; but it is certain that, whatever influence this circumstance may have in *assisting* the deposition of coagula upon diseased portions of the arterial tracts, it alone can never prove sufficient to occasion a positive stagnation in the current through the pulmonary vessels.

* I have also recorded another case of renal anasarca, in which I observed appearances almost precisely similar to those described above, in both emulgent veins, portions of the *pulmonary artery*, and ascending aorta. Guy's Miscellaneous Inspection-Book, vol. xiv. p. 14.

Dr. Bright has furnished us with a very marked example of renal disease determining the occurrence of fatal obstruction in the pulmonary artery.*

M. R., a middle-aged woman, was admitted to Guy's Hospital, Nov. 8th, 1826, with anasarca. She had been two or three times in the hospital, during the last two years, with renewed attacks of the same disease, and had gone out relieved. She was now in the most hopeless and advanced state of disease. The effusion somewhat diminished under treatment; but, on the evening of the 21st, she became rather suddenly worse, complaining of great difficulty in drawing her breath; and, although assistance was immediately obtained, she died in a few minutes.

Sectio cadaveris.—Lungs tolerably healthy in structure; but it was found that the *pulmonary artery* was completely blocked up by a coagulum of fibrin of firm texture. The hand was not particularly large. The vena portæ, and its large branches going into the liver, were likewise obstructed with coagulum, nearly separated from the red particles, and the saphena vein was in the same condition. The liver was tolerably free from morbid change. The kidneys afforded very fine specimens of the confirmed granulated change. The spleen was healthy.

The following interesting case is abridged from Mr. Paget's report.†

A ruined wine-merchant, 55 years old, and who had drunk hard, had been in St. Bartholomew's Hospital since the 18th of April, suffering from stricture of the urethra and paralysis of the bladder. He was a bloated, unhealthy looking man, and had occasionally slight œdema of the hands and feet; but he was so nearly well that he never complained of more than the disorder of his urinary organs. It was once noticed that he had a cough; and he said that he was often subject to such an one, but thought it trivial. In the last week of his life he had sometimes spoken of having occasional shortness of breath, so that when he wished to sleep in the day, he was "apt to be taken with a panting;" and such an attack came on once or twice while he was walking in the ward; but these attacks were rare, they lasted only a short time, and they never appeared so serious as to elicit more than a passing remark from him. Within two hours of his death he talked in the same good spirits in which he had been all day. On the morning of the 4th of May, he was thought to look unusually well. In the afternoon, after a short sleep, he proposed to a patient near him to have tea; but first he went to the water-closet, and, on his return, presenting nothing unusual in his appearance, he sat down on his bed-side. A minute after, he fell heavily on his face; and, with a slight distortion of his mouth and of one arm, died before two minutes more had elapsed.

On inspection, it was found that some of the large bronchi contained a frothy mucus slightly tinged with blood. The left pleural cavity was obliterated by general and rather close adhesions, the right was free, and contained a small quantity of fluid. The lungs were large and deep-coloured, but, externally, appeared nearly healthy: their upper lobes were œdematous; the middle and lower lobes partially emphysematous, and containing a moderate amount of blood and serum. There was in no part of either lung a trace of pulmonary apoplexy, or of intense congestion. The right cavities of the heart were larger, their walls were of about the natural thickness. The tricuspid and pulmonary valves were healthy, and so was the *pulmonary artery*, in the size and texture of both its trunk and all its visible branches. But nearly all the branches beyond the primary divisions of the pulmonary artery contained clots of blood, which, from a comparison of them with those found in tied arteries, Mr. Paget judged to be from three to ten days old, those of the least age being in the largest branches. They were firm, cylindroid, and of various colours—black, brownish, pink, and yellow ochre, irregularly mixed. Some of them even much drier than recent clots, and grumous; others appeared invested and intersected by thin layers of white fibrine. Most of them filled the portion of the artery in which they lay, some did so to distension; a few of the driest and most decolorised adhered to the walls of the vessel, and left the surface rough and blood-stained when they were removed. The clots did not commonly extend continuously from any large branches of the pulmonary artery into many of its successively subordinate divisions: some of them were in the shape of thick cylinders from half an inch to an inch in length, which filled a branch of the artery, but were not even in contact with the similar clots which filled adjacent branches; some extended from a larger branch into two or three smaller ones derived from it, filling them all, but ending abruptly; only a few branched many times, and filled a larger series of the branches of the artery. No branch of the pulmonary artery less than half a line in diameter appeared to contain any of these clots, and the pulmonary veins were healthy and empty. The left auricle and ventricle were larger, and the walls were thicker, than in the healthy state: they

* Reports of Medical Cases, vol. i. p. 19.

† The case is very fully detailed by Mr. Paget (op. cit. vol. 28, p. 353), but for the sake of brevity I have omitted many of the negative statements.

contained a small quantity of black fluid blood. The mitral valves were opaque, but pliant, and probably efficient. The aortic valves were in a similar condition, and of large size, adapted to the aorta itself, which was uniformly slightly dilated and thickly beset with fatty, and a few earthy, deposits in its coats. The large systemic arteries and veins were all healthy and empty, or contained a little fluid blood. The entire digestive canal was healthy, as was also the liver and pancreas. The spleen was of middle size, and soft. In both kidneys the pelvis and infundibula were dilated, the papillæ flattened and expanded, and the texture generally, but not uniformly, indurated and contracted, so that their external surface was lobed, and in parts coarsely granular. The ureters were dilated and tortuous, the bladder very large, and its walls thin and weak.

Mr. Paget also relates two other cases in which similar morbid appearances were observed in the pulmonary artery in association with renal disease; but he very justly suggests that here the state of the heart and lungs may also have had a great share in producing those vascular lesions.*

In November, 1846, I was present at the inspection of the subjoined case. The details are unfortunately imperfect; but there is reason to believe that the individual's habits had been intemperate, and his liver and kidneys presented manifest traces of chronic disease.

A wine-cooper, ætat. 45, came under treatment at Guy's Hospital, suffering principally from cough with mucous expectoration. Some time previously he had been the subject of empyema of the left side, which had been relieved by tapping, at another hospital. There still remained a fistulous external opening, but this appeared to be confined to the parietes. Previously to his death he suffered from ascites and anasarca, and there were observed to be evidences of a large cavity in the centre of the left lung. On *examination*, the right cavities of the heart were found to be very capacious and thick. The trunk of the *pulmonary artery* was greatly dilated, as were the whole of its branches that remained open. The right lung contained tubercular deposits scattered throughout its substance; its lower parts were remarkably congested, and large portions presented traces of recent pulmonary apoplexy. One of the two primary branches of the artery on this side was completely obstructed by a strongly adherent coagulum, the lower portion of which, which had been exposed to the current of blood in the primary arterial branch, projected in the form of a rounded pellet: it was of a dusky purple colour, and was about the size of a common marble; but, immediately beyond this part, the vessel became greatly contracted upon the adherent clot, which internally had a semi-transparent horny appearance, like the outer layers of coagulum in very old aneurisms. A small portion only of the lower lobe of the left lung contained air; its remaining portion was compressed, tough, and solid, and reduced to a very small substance by an extremely thickened and cartilaginous state of the adherent pleural surfaces. Its centre was occupied by a large cavity, the surfaces of which appeared to have been placed nearly in apposition by compression. The pulmonary branches on this side were extremely large; two considerable tertiary branches were completely obstructed at their origins by adherent clots, which did not project from the orifices. Where the coagula were torn from their attachments, there merely remained a rough surface tinged with blood. The left ventricle was rather large, but it appeared small when compared with the right. The aorta was healthy. The liver presented an extreme degree of "hob-nail" contraction. The kidneys were large, and coarse in structure.

Occurrence of clots in the pulmonary branches in association with rheumatism.

Like the trunks of the systemic arteries, those portions of the afferent vessel of the lungs which are situated above its valves appear usually to enjoy an almost complete immunity from severe inflammatory lesions in cases of rheumatism. I have already, however, had occasion to quote an instance (M. Meli's case) which probably formed an exception to this rule. In the following example, also, there appear to be strong grounds for believing that the arterial lesions were due to that vitiated condition of the blood which is known to attend rheumatic cachexia. In this instance, however, the deposition of clots in the vessels appears to have depended upon a less acute form of inflammation than that which attacked the vascular system of M. Meli's unfortunate patient.

A respectable married woman, 29 years old, was admitted into St. Bartholomew's Hospital, under the care of Dr. Burrows, on the 5th of May, 1843. She had enjoyed good health till the previous October, when she had rheumatism, which affected rather severely all the large joints in succession, and lasted for six weeks. After her recovery, she had occasional wandering pains, and three weeks before her admission she noticed that her eyelids and face began to swell, and her joints, especially her knees and elbows, became swollen and painful, so that on the fourth day of her illness she was

* Cases II. and III., vol. xxvii. pp. 168-171.

obliged to confine herself to bed. On admission, the swelling of the joints had subsided, but they were stiff and tender; her feet and ankles were slightly œdematous. She was extremely weak and languid, complained of great thirst, and lay on her back, unable to lie on either side. She breathed 32 times in a minute, with some difficulty occasionally. Her pulse was 116, and soft. Her complexion was sallow, with flushed cheeks, dark lines around the eyes, and sharpened features. The lips were dry and pale, the tongue dry and furred, with a red tip. An abundant hot and acid-smelling perspiration was on the skin, and the urine was scantily but naturally secreted. Auscultation of the heart's sounds detected nothing unnatural. For three days after her admission no material change in her condition ensued. Nutritive diet, and gentle purgatives and diaphoretics, were given, and she appeared slightly improved; but the great depression of the system, the anxiety of expression, and the rapid pulse and respiration, persisted. She perspired profusely, and was very thirsty. For two days the abdomen was distended, tympanitic, and rather painful on pressure: on the next day she complained of great tenderness in the right iliac region; and on this day also (May 9th) the præcordial region was found tender, and on auscultation a distinct bellows-sound was heard at the base of the heart, accompanying the systole. The state of the joints varied from day to day. On the 10th, the cheeks and forehead were covered with an erysipelatous blush, and numerous red acuminated papulæ had appeared upon the chest; the urine was very irritating, and smelt offensively; sloughs had begun to form upon the sacrum, and the weakness and depression were increased. On the morning of the 11th, after passing a comfortable night, she was suddenly seized with a sensation of great tightness in the præcordial region, violent palpitation of the heart, and the most urgent dyspnœa. The attack lasted for an hour, and then she returned to nearly the same state as she had been in before it: but from this time she sank more rapidly. Upon auscultation, no respiratory murmur was heard below the right breast, and it was dull on percussion; the habitual dyspnœa became greater, the sloughs on the nates and sacrum extended, the abdomen became again tympanitic and tender, especially about the right iliac region. She vomited several times, and died in the afternoon of the 13th. Dr. Burrows observed that the symptoms were very obscure at the time of the patient's admission; he regarded the case as one of rheumatism occurring in a woman of feeble constitution. Upon the appearance of the remarkable symptoms on the morning of the 11th of May, he formed the opinion that she was attacked with passive pneumonia of the right lung, and he considered it highly probable that the constitution was suffering under the depressing effects of the typhus poison, for there were several severe cases of typhus fever in the beds adjacent to that of the patient at this time.

At the examination forty hours after death, Mr. Paget found all the blood which appeared in the ordinary course of the dissection, either fluid, or coagulated in soft black masses. The pericardium contained half an ounce of fluid; there were a few slender adhesions between the trunks of the large vessels, and on the surface of the heart were two white spots. The heart was of ordinary size, and its parts well proportioned, but its tissue was rather weak and pale. The valves on its right side were thin, clear, and healthy; those on the left side were a little thickened and opaque, but quite pliant. In the coats of the aorta were a few scattered fattty deposits; those of the *pulmonary artery* were healthy. Each pleural sac contained a small quantity of clear fluid, and at the apex of each lung there was an adhesion, with a few dry and shrivelled tubercles beneath it. The lower lobes of both lungs were slightly but generally œdematous, and gorged with blood. The upper lobes, with the exception of the small groups of old tubercles, were healthy. The air-passages were also healthy. Nearly half the branches of the *pulmonary artery*, from those of the second order to those of the fifth and sixth, (and probably to yet smaller branches), were blocked up by old coagula of blood. These were cylindrical, soft, and grumous, and in colour were a mixture of pale pink and dirty greyish white, with spots and blotches of deep crimson. They were not more numerous in one lung than in the other, and were irregularly scattered through all parts of each. They did not quite fill the vessels which contained them, but at various parts they adhered closely to the walls. The trunk of the pulmonary artery, and many of the branches, which did not contain coagula of the kind just described, contained fluid and softly clotted black blood. The abdominal and pelvic organs were healthy in their structure, but very pale and weak.* It appears to be rather uncertain whether the violent aggravation of this patient's symptoms, which occurred suddenly three days before her death, depended, as Dr. Burrows conjectured, upon the supervention of passive pneumonia, or upon the formation of soft coagula in the already greatly obstructed pulmonary branches. Either of these causes would have been sufficient to account for the great præcordial

* Mr. Paget's first Memoir, loc. cit. p. 174.

distress, palpitation, and dyspnœa, with which the patient was then attacked. It is highly probable that both these influences in some measure concurred to produce the symptoms in question, but the latter doubtless had the greater share in determining the fatal result.

On the occurrence of adherent coagula in the pulmonary artery in association with apoplexy of the lungs.

M. Cruveilhier appears to have been one of the first writers who observed the coincidence of coagula of long standing occupying a more or less extended portion of the pulmonary arterial tract with apoplectic extravasations into the tissues of the lungs. He remarks* that in a case of pulmonary apoplexy accompanying disease of the heart, he found all the arterial divisions which impinged upon the extravasations filled with concretions which ramified with the vessels. The clots which filled the smaller divisions of the artery were solid, and of a reddish colour; those which occupied the larger branches were hollow and decolourised. These clots did not adhere to the arterial parietes, neither did the latter present any appearance of inflammation. In 1838 I brought forward notes of a few cases† in which I had met with a similar train of morbid appearances; but it is to Mr. Paget that pathologists are indebted for an exposition of the frequency with which these lesions occur, and for a description of the anatomical characters which they usually present.‡ This gentleman observes that in all, or in a great majority, of cases of pulmonary apoplexy, especially in that form of it in which the blood collects in a defined and compact dark mass, the branches of the pulmonary artery, leading to the seat of effusion, are blocked up by coagula, which present the distinguishing characters of those formed long previous to death. In most cases (perhaps in all those which depend only on disease of the heart), he has remarked that the coagula are found in those branches alone in which the circulation is obstructed, but, in some instances, they extend further, the apoplectic effusion being inconsiderable in comparison with the size and number of the branches which are filled by coagula. These cases, he considers, make it highly probable that sometimes the formation of coagula in the pulmonary arteries, when found combined with pulmonary apoplexy, has been not consequent on the effusion of blood but coincident with it, or has, in some measure, preceded it.

It is almost needless to say that pulmonary apoplexy most frequently results from severe and long-continued causes of mechanical obstruction to the circulation through the lungs—such as chronic bronchitis, contraction of the mitral and aortic ostia, aneurisms of the ascending aorta, &c. It is evident, however, that the structure and arrangement of the pulmonary vessels are admirably adapted to enable those tubes to resist the injurious mechanical effects of inordinate distension during very lengthened periods. The remarkable pliability and tenacity of the branches of the artery, which impart to them a close similarity to the internal veins, have already been alluded to at the commencement of this memoir; and all morbid anatomists must have observed that, in their capability of undergoing extreme distension, the pulmonary veins are scarcely equalled by any other set of vessels in the body. This has evidently been provided for in the peculiar mode in which these veins are distributed throughout the lungs. Dr. Addison has demonstrated that the branches of the pulmonary veins do not accompany those of the pulmonary artery in all their ramifications, as was supposed by the older anatomists, but that they lie separately at the bottom of the pulmonary fissure, surrounded only by a loose cellular membrane, and resting merely upon the surface of the unbroken pulmonary tissue. These vessels are thus, throughout their whole course, found to be situated on the exterior of the aerial cellular structure of the lungs.* Hence the artery is admirably adapted to endure temporary or long-continued distension consequent upon obstruction to the transit of blood through the pulmonary capillaries; while, under circumstances of impediment to the free emptying of the pulmonary veins, both sets of vessels are capable of assuming the condition of reservoirs, in which the delayed blood is permitted to accumulate.†

* Anatomie Pathologique du Corps Humain, t. i. livraison iii. p. 3.

† Since published in Guy's Hospital Reports, No. 13: Observations on Acute Aortitis.

‡ Medico-Chirurgical Transactions, vol. xxvii. p. 163.

* Observations on the Anatomy of the Lungs, by Thomas Addison, M.D. Medico-Chirurgical Transactions, vol. xxiv. p. 153. 1841.

† The degree in which the pulmonary arteries and veins admit of being dilated, is generally displayed in extreme cases of contraction of the left auriculo-ventricular orifice, where care has not been taken during life to reduce very considerably the volume of the circulating fluid. The extraordinary amount of dilatation which may be produced in the pulmonary veins by causes of direct obstruction, is well illustrated in a case related by Dr. R. Townsend. A man, æt. 62, died asphyxiated, after having long suffered from frightful periodical attacks of suffocative orthopnœa. About twelve months previously to his death he had profuse hæmoptysis, but this did not again recur. On examination, the pulmonary veins were found enormously loaded with blood, being dilated to at least four times their natural size; those vessels, which are naturally no bigger than crow-quills, being as large as the fingers of a glove. This state of the veins was found to arise from the compression which they suffered at their entrance into the left auricle, the parietes of which seemed converted into one solid unyielding mass of tubercular matter, nearly an inch in thickness, developed in the tissues of the auricles; and, by the pressure which it made on the pulmonary veins, diminishing their calibre so much that a probe could with difficulty be passed through them into the auricle. In the narrative of this case no allusion is made to the presence of adherent clots either in the dilated veins or in the pulmonary artery, or right cavities of the heart, nor to the existence of any traces of pulmonary apoplexy, although that lesion was doubtless present at the time when the attack of hæmoptysis occurred. ("Contributions to Pathological Anatomy," and Medical and Physical Journal, vol. lxviii. p. 520.)

The question of the source of the extravasations in pulmonary apoplexy has long formed a subject of discussion with pathologists. The opinion formerly entertained that in these, as well as in some other forms of disease attended with hæmoptysis, the blood escaped from unbroken vessels by a process of exhalation, has probably been wholly abandoned, since a more careful investigation of structural anatomy has proved the impossibility of such an occurrence. The practical researches of Mr. Hilton have, I believe, enabled that gentleman to enunciate the principle that hæmorrhages invariably occur from the open mouths of vessels. It still remains, however, to determine from which set of pulmonary vessels these extravasations take place. The extreme frequency of large deposits of coagula in branches of the pulmonary artery would lead to the conclusion that it is from these canals that the hæmorrhage occurs. I am inclined to believe that, in some cases, especially where the extravasations are remarkably large, the blood is effused from arterial branches of considerable size. In one of the first examples of pulmonary apoplexy which I examined minutely, I felt convinced that many of the smaller branches of the artery had suffered rupture, followed by extravasation of blood into the substance of the lungs. Upon opening the vessels which led into the clots, their linings were found softened, and presented other very decided marks of acute inflammation: other portions of the artery also displayed similar changes. This occurred in a patient who died from the effects of long-continued bronchial obstruction. It is evident that in cases of this particular description, as the cause of impediment exists merely in the capillary circulation of the lungs, the extravasations must necessarily occur from some portion of the arterial tract, the venous system of the organs remaining perfectly free from undue distension. In a large proportion of instances, however, (as in Cruveilhier's case already cited,) the structures of the arteries, which are found obliterated, or obstructed by clots, do not present any very evident traces of recent inflammatory change: hence it is probable that, either the tissues of the vessels, having been long protected from irritation and over-distension by the attached coagula, have had time to regain their natural condition, or that the effusions have been due to rupture of some of the pulmonary capillaries. In discussing this question, M. Cruveilhier, after alluding to the obstructed condition of the arterial branches, mentions the fact that Laennec discovered coagula in some of the divisions of the pulmonary veins in a case of apoplexy of the lungs. This, however, I believe to be a rare and exceptional occurrence. Mr. Paget, who has evidently examined the lesions which attend this disease with great minuteness, makes no allusion whatever to coagula in the pulmonary veins, nor has such a condition presented itself within my own observation. It is evident that the pulmonary veins are very far more dilatable than the pulmonary artery; and, although they may occasionally suffer rupture*, this lesion very unfrequently, indeed, takes place, even under circumstances of extraordinary distension. I am very strongly disposed to believe that, in the great majority of instances of apoplexy of the lungs, the pulmonary artery, having long been submitted to inordinate distension, at length becomes irritated and inflamed by the continued presence of highly carbonised blood within its dilated and now inelastic branches, coagula gradually become deposited over tracts of the diseased and yielding vessel, which they alike defend from the contact of the blood and from the occurrence of ulceration or rupture in those situations. Subsequently, however, branches of the vessel give way, and permit the escape of blood into the pulmonary substance: the extravasation continues to extend as far as the elastic tissues of the lung will admit, and then, doubtless, the open vessel becomes compressed by the surrounding coagulum, and is subsequently further defended by the deposition of a solid clot in its interior. This view is not wholly theoretical, as it will be found that the morbid appearances, which have next to be described, afford very strong evidence of its validity. My own observation does not enable me to decide the question of whether pulmonary apoplexy ever

* M. Portal has recorded a case in which a rupture, nine-tenths of an inch in extent, occurred in one of the left pulmonary veins near the point of its emergence from the lungs. The sygmoid valves of the aorta were greatly diseased, and caused impediment; the entire heart was enormously dilated, the pericardium being inseparably adherent. The left pleural cavity was full of blood. This occurred in a person 21 years of age, who died during a fit of hæmoptysis, having suffered from symptoms of cardiac disease for three years previously. (Memoirs of the Royal Academy of Sciences at Paris, 1784.)

depends upon rupture of the bronchial arteries. It is, however, evident that the opinion which has been held by some pathologists that this lesion may result, in cases of hypertrophy of the left ventricle of the heart, from the violence with which the blood is propelled through these slender vessels, is wholly inadmissible. It is by no means improbable that where, in addition to long combined obstruction to the pulmonary circulation, the orifices of these vessels become, as frequently happens, greatly contracted, owing to the formation of bulky interstitial deposits in the subserous tissue of the descending aorta, their canals, being thus exposed to a very inordinate degree of distension, may become ruptured within the substance of the lungs, and produce appearances closely similar to those which characterise the ordinary forms of diffused pulmonary apoplexy.

The following case, by Mr. Paget, affords an excellent example of the various morbid appearances presented by the pulmonary artery in the severe forms of apoplexy of the lungs depending upon mechanical obstruction to the circulation.

A widow, 70 years old, thin and emaciated, came under treatment with symptoms of severe thoracic obstruction, supervening upon the aggravation of a winter cough. She sunk rapidly on the third day after her admission. Dr. Burrows remarked an extreme hurry of the circulation, with feebleness of the pulse, and great prostration of strength.

In the examination 36 hours after death, the lungs were found of ordinary size, but deeply wrinkled when collapsed. There were a few scattered adhesions on their anterior surfaces. Their tissue was generally light and crepitant, and in a few parts slightly œdematous; the air cells were of ordinary size, except about the lower and anterior margins of the lobes, where there was a moderate degree of emphysema. In a few parts the pulmonary tissue was compressed, and at the anterior and lower border of the left lung was a small patch of compact pulmonary apoplexy, with some diffused apoplexy around it. The bronchi were of ordinary size, their mucous membrane thin, pale, and firm; their tubes full of thick, semi-transparent, viscid mucus. In each lung, one of the superior, and one of the inferior main branches of the *pulmonary artery* were blocked up by a large, firm, mottled, clot of blood, which from itself, as a trunk, sent branches into two or more of the next order of branches of the artery. The colours of the clot were black, deep crimson, rusty, pink, and yellowish, in various irregularly mingled shades; they were moderately firm, of nearly uniform consistence throughout, and capable of being rubbed into a thick grumous substance; they adhered so firmly by parts of their surfaces to the adjacent walls of the vessels, that they could not be smoothly removed. The branches of the largest clots did not extend far into the arteries, but terminated abruptly in the arterial ramifications immediately proceeding from those in which they lay. But beyond their terminations, many smaller branches of the pulmonary arteries in all parts of the lungs contained short, firm, dry, mottled, and adherent clots, variously coloured. None of these were long or much ramified; none were continued through more than two branches, and many of them were not more than half or three quarters of an inch long; some were even shorter, and lay like bits of large clots upon the walls of the vessels; they had all the same characters as those already described, and the portions of lung adjacent to and beyond them were not different from the rest. Moreover, in several of the larger branches of the artery, there were appearances of clots of blood formed like those just described, and having been still further altered and organized. There were pale, semi-transparent, soft, and flattened narrow bands, attached firmly to the walls of the artery, and presenting all the characters of the organized clots which the narrator has sometimes seen adhering to the walls of divided arteries. They were from one quarter of an inch to an inch in length, and about one-tenth of an inch wide; a few were fixed in their whole length to the walls of the artery, but most of them by their ends only, so that a probe could be passed under them. Among the clots there were all gradations, between those last and those first described; and, in one instance, one of the largest of the more recent clots was continuous with a flat, semi-transparent, and adherent portion, like those which had existed for the longest time. (These appearances are well represented in a plate.) The branches of the pulmonary arteries which were thus obstructed were firm and polished internally, but in many places had fine, scattered, and grouped grains of yellow deposits in their coats. They were all of natural size, and the pulmonary veins were healthy. The cavities of the right side of the heart were considerably dilated and hypertrophied. Both of them were distended by soft and uniformly black coagulum, slightly adhering to their walls; and in the appendix of the right auricle the coagulum had become firm, mottled, and closely adherent, somewhat like those in the pulmonary arteries. The left auricle and ventricle were also dilated and hypertrophied, but in a less degree than the right; they contained a smaller quantity of similar soft, black coagulum. The tricuspid valve was healthy; the pulmonary valves were

large, and in small patches, opaque, but thin and pliant; the mitral valve was slightly thickened, and had several spots of soft yellow substance beneath its ventricular surface; the aortic valves were also irregularly thickened and opaque, with enlarged corpora Arantii, bearing small warty growths; but both they and the mitral valves might be deemed able to discharge their functions. The liver was small, tough, and granulated, and a great part of its surface was covered by long adhesions. The gall-bladder was full of calculi. The kidneys were small, and somewhat granulated, with adherent capsules, and numerous cysts full of transparent fluid. The large veins and the systemic arteries, as far as they were examined, were healthy.*

The morbid appearances described in the above narrative very closely indeed resemble those which I have myself frequently observed in cases of this description. I took the following notes after examining the lungs of a strong middle-aged man, who died, with bronchitis, hydrothorax, and extensive pulmonary apoplexy, at Guy's Hospital, in September 1846:—

A large secondary branch of the *pulmonary* artery was completely blocked up by a firm decolourised coagulum, evidently of long standing, the end of which hung into the cavity of the primary division of the vessel, in the form of a pedunculated, hollow, fibrinous concretion, of oval form, precisely similar to those cystiform fibrinous deposits which have been so accurately described by Dr. Hughes as adhering to the internal parietes of the heart, in many cases of cardiac obstruction.† While tracing out the branches of the vessel, my friend Dr. Gull pointed out to me two small blueish spots, which at first sight appeared to have been produced by effusion of blood beneath the arterial lining. It was impossible to distinguish with the unaided eye the polished surface which covered these thin deposits, from the epithelium of the vessel; still they could be peeled off, leaving a perfectly smooth surface of artery beneath. In examining one of the secondary branches of the vessel, I found that its calibre had become greatly contracted by the presence of a firmly adherent deposit of this kind, which extended to a distance of two or three inches. It occupied about one-half of the periphery of the artery, leaving a free, though greatly diminished, canal for the passage of blood. It appeared to be of considerable standing, being nearly decolourised, and of very dense consistence. It seemed probable that the transit of blood had gradually smoothed down the exterior of this deposit, until it at length presented a level and polished surface, which appeared to be perfectly continuous with the proper lining membrane of the artery.* The heart of this patient was hypertrophied, and his aorta rather dilated, but there was no marked degree of valvular disease. These appearances, it will be observed, closely resembled those which were observed by Mr. Paget in the preceding case. It can scarcely be doubted that, in both instances, the deposition of coagula, in the larger branches at least, was due to a certain amount of inflammation set up in the tissues of the over-distended vessels. Both cases are rather extreme examples, but they may be taken as fairly representing the general character of the morbid appearances which more or less extensive tracts of the artery will be found to present in the majority of instances of true pulmonary apoplexy.†

Occasionally some of the larger branches of the pulmonary artery leading to the apoplectic portions of lung are found completely coated with tubular fibrinous deposits, as was the case in a female who died with old obstructive and regurgitant mitral disease at Guy's Hospital in December 1845.

* Op. cit., vol. xxvii. p. 178.

† An appearance of this kind is represented in Mr. Paget's engraving already alluded to.

* I have never observed an appearance precisely similar to this in any of the systemic arteries. In these vessels, where a portion of fibrinous coagulum becomes deposited upon the lining of the tube, its surface usually remains to a certain degree granular and rough, and is always ready to receive fresh deposits from the blood. The only exceptions to this rule are the cystiform clots, which are produced in the cavities, and, occasionally, about the valves of the heart, and which generally present a smooth exterior; and the interiors of old and partially cured aneurismal sacs, the coagulum of which sometimes offers a dense and polished surface to the current of blood. In general anatomical characters the mode of deposition of coagula in the pulmonary artery bears a close similarity to that process as it occurs in the veins. In many cases, large coagula, which either coat or occlude portions of the larger branches, peel off with the greatest readiness from the interior of the vessel, leaving the epithelium smooth and, not unfrequently, free from discolouration. It is evident that here, as in the veins, the deposition of clots is designed as a means of protecting inflamed surfaces of the vascular tract, and of guarding them against distension, and the irritating contact of the blood; the circulation through these vessels is for a time either diminished or suspended until the inflamed surfaces have regained their healthy condition, or have ceased to be acutely diseased, and then the deposit is entirely absorbed or remains permanent, according to the degree in which it has previously become adherent to the vessel. Uneven and granular clots may frequently be observed in the pulmonary artery; indeed, it is probable that all fibrinous deposits in this vessel at first present rough surfaces which, if life be much prolonged, have subsequently a tendency to become smoothed and levelled.

† I have here restricted the term "pulmonary apoplexy" to those cases in which ulceration or rupture of portions of the vessels of the lungs is followed by the extravasation of considerable quantities of blood into the tissues of those organs, excluding those instances in which nearly the whole of the effused blood finds exit by the air-passages.

Here I found apoplexies in various portions of the lungs. The ascending trunk of the pulmonary artery was dilated, and coarse in structure, but free from opacity or interstitial deposit. One of its primary branches was coated, but not obliterated, to the distance of about an inch and a half or two inches, by a layer of partially decolourised clot, which closely adhered in some spots, and was merely glued to the interior of the vessel in others. Other fibrinous coagula which adhered to this led into the various ramifications of the vessel, but did not completely fill the larger ones. The lining membrane was discoloured, as if ecchymosed, at the spots where the clot adhered most strongly, and was perfectly clear where it was most loosely attached. The whole of the surrounding lung-tissue was filled with dark and rather softly coagulated blood, but no rupture could be detected in any of the larger vessels.

I have never seen any instance in which lesions of the above description extended throughout the entire tract of the pulmonary artery in cases of apoplexy of the lungs. Neither have I ever, under such circumstances, found the valves, or the first two inches of the vessel, occupied by clots, or bearing any traces of recent inflammation.

It will generally be observed that a more or less adherent clot occupies a considerable tract of the branch from which the hæmorrhage has occurred; but other branches may often be traced through the clot, which remain quite pervious, and present no appearance of having been at all injured in their structures by compression—a fact which appears to prove that the formation of clots within the vessels is not, in this instance, the result of external causes. The vessels which lead to the extravasations are usually most replete with coagula, but their entire tract is not always obstructed by these masses: very frequently the clots are deposited at intervals, leaving portions of the vessel an inch or two in length apparently free from disease.

It would appear that occasionally very large deposits of this kind occur in the vessel, and remaining for months, perhaps for years, as causes of obstruction to the circulation, aggravate considerably the symptoms from which the patient suffers, without tending immediately to hasten the fatal termination of the disease. This appears to have been the case in the following instance.

A woman, aged about 36 years, died at Guy's Hospital in August 1845, having suffered from extreme difficulty of breathing, with marked lividity of the countenance. She was incapable of lying down; there were the evidences of pleuritic effusion on the left side, and a loud superficial bruit was heard over the right ventricle. The pulse was small, rapid, and feeble.

On inspection, a large quantity of serum was found in both pleural cavities. The larynx was rather narrowed. The bronchi contained a brown viscid secretion; there was intense vascularity of the pulmonary mucous membrane; the lungs were congested, and partially emphysematous, and presented a certain degree of apoplectic mottling at various spots. The pericardium contained a moderate quantity of clear serum. The right auricle was exceedingly capacious, and its walls thick. The tricuspid orifice was contracted by deposit beneath its endocardium, forming a wrinkled edge rather less than two inches in circumference. The right ventricle was somewhat dilated: its walls were remarkably strong and thick. The *pulmonary artery* was rather below than over the average size: its left primary branch was obstructed, at its commencement, by a singular tongue-shaped mass, which projected upwards to the distance of about an inch and a half, having a broad base of attachment below, and gradually diminishing in size, and tapering to a point above. The lower and wider portion of this growth had the appearance of having been deposited between the coats of the vessel, as its polished surface was quite continuous with the lining membrane, but it had evidently been formed upon the interior of the artery in the manner which has been already described. This mass also formed a dark prominence on the exterior of the vessel, and it projected so considerably inwards as greatly to narrow, although it did not altogether occlude, the arterial canal. The most pointed part of this body was evidently formed of decolourised coagula of blood: the interior of the mass had the appearance of blood which had long been enclosed until it had become colourless and friable. No other deposits were discovered in the branches of this vessel. The left auricle was enormously dilated; the auriculo-ventricular orifice was extremely contracted and bony, barely admitting the handle of the scalpel. The lower mitral orifice was also contracted. The left ventricle was small: its walls were about equal in thickness to those of the right. The aortic valves were efficient, but rather thickened. The aorta was nearly of the average size. The liver and kidneys were congested, and rather contracted, but there did not appear to be more disease about these viscera than would inevitably result from the extreme and long-continued cardiac obstruction.

Although, in the majority of cases, pulmonary apoplexy, with its attendant lesions, is due to the influence of some fixed impediment to the circulation, it may evidently result from any cause which greatly diminishes the cohesion of the pulmonary vascular tissues, whether that cause be inflammation, over-distension, or a highly vitiated condition of the blood. Dr. Byron mentions* that the lesion in question was very frequently met with in the bodies of those who were carried off by the epidemic yellow fever of Gibraltar in 1828. He also refers to its occurrence in that form of low typhoid fever which was formerly known as "typhoid pneumonia," and narrates cases in which extensive extravasation of blood into the lung occurred, to all appearance, merely as the result of a species of hæmorrhagic diathesis. The occasional association of pulmonary apoplexy with gangrene of the lungs has been remarked by several authors, especially by Dr. Graves and Dr. Thomas Addison. The former gentleman denies that the effused blood is capable of producing inflammation, gangrene, or softening of the affected portions of the lung; the latter considers that the circumscribed gangrene of Laennec is commonly a mere effect or advanced stage of pulmonary apoplexy. It is evident that gangrenous disorganization of the substance of the lung may occur independently of any clear evidences of the previous or concurrent existence of apoplectic effusion: it is also certain that most extensive extravasations of this kind are frequently met with where the vitality of the surrounding lung substance does not appear to have suffered in the least degree from the presence of the adventitious mass of coagulum; still it cannot be denied, that, where the constitutional powers have been previously greatly lowered by asthenic disease, the disintegration and compression which portions of the lung sustain in consequence of these effusions may, not improbably, be followed by sloughing of the parts. Nevertheless, I am inclined to believe, that, in most of the cases where gangrene and apoplexy are found associated with each other, the effusion of blood has been the secondary lesion consequent upon the rapid destruction of vessels which have not become sealed in the ordinary manner previously to their implication in the gangrenous action. In the generality of cases of this kind the hæmorrhage probably arises from small vessels, the larger branches having become closed by coagula, or retaining sufficient vitality to be capable of resisting the destructive process. Death, however, has been known to result from the sloughing of a considerable branch of the artery occurring during the rapid progress of gangrene of the lungs.

* "Rare Cases," Dublin Journal, vol. xxvii. p. 67.

Occurrence of adherent coagula in the pulmonary branches in cases of malignant disease.

Mr. Paget was, I believe, the first author who fully directed the attention of pathologists to the fact that when the matter of medullary cancer or scirrhus passes into the blood, and circulating with it, is stopped in the capillary vessels of the lungs, the branches of the pulmonary artery may be to a great extent filled by it, and by coagulated blood or fibrine mixed with it. He offers the following case as a good example of this fact:—

A woman of drunken habits, 45 years old, had been in St. Bartholomew's Hospital with all the symptoms and appearance of one suffering from cirrhosis. She could assign no distinct date to the commencement

* *Note on Cases of Pulmonary Apoplexy.*—If, in the present state of pathological knowledge, it were necessary to bring forward a variety of facts in proof of the opinion that large extravasations of blood into the tissue of the lungs result from the active bleeding of open vessels, and not from transudation or exhalation, as was formerly maintained, very strong evidence would be found in those cases where large apoplectic effusions have produced rupture of the pleura pulmonalis, and proved suddenly fatal by hæmorrhage into the thoracic cavity. It is quite evident that such a lesion as this could not result from a gradual oozing of blood into the pulmonary tissue. Almost instantaneous death from the effusion of three quarts of blood into one of the great serous cavities (see case by Dr. Ferguson, in the first vol. of the Dublin Med. Transactions, p. 11) can only occur as the result of rupture of some considerable vessel. It is true that, in the case just alluded to, as well as in several others which have been collected in an interesting memoir by Dr. Paterson, ("Observations on Pulmonary Apoplexy proving fatal by rupture of the periphery of the lung by effusion of blood into the pleura"—Edinburgh Med. and Sur. Journal, Jan. 1846), the vessels from which the hæmorrhage proceeded were not detected. This, however, is not remarkable, when the torn and infiltrated condition of the pulmonary tissues in those cases is taken into account. The plan usually adopted by Mr. Hilton, of slowly injecting water from a large syringe into the vessels of the part or organ from which the bleeding is known to have occurred, is usually most successful in discovering the breach of vascular continuity. But even the failure of this means is by no means sufficient evidence that the hæmorrhage has not occurred from some considerable vessel which has either become closed, from the coagulation, subsequently to death, of the surrounding infiltrated blood, or from the formation of a plug in its canal upon the occurrence of the fatal syncope.

This class of cases also affords an interesting proof of the great influence which the elastic property of the tissue of the lungs has in controlling hæmorrhages which occur within those organs. Pulmonary apoplexy rarely, if ever, proves suddenly fatal when the open vessels lie deeply towards the interior of the lungs; on the contrary, when the lesion occurs near the surface and the pleura becomes ruptured, death from uncontrolled hæmorrhage appears to be the almost necessary result. Good specimens of clots deposited in the pulmonary artery, in association with pulmonary apoplexy, are preserved in the collection at Guy's, Preparations 1450 ss and 1450 oo.

of her disease, and died very much emaciated, jaundiced, and with considerable ascites and anasarca of the lower extremities. On examination thirteen hours after death, the author found the liver large, hard, yellow, and dry and adherent to a contracted great omentum. It contained several medullary tumors, composed of very soft tissue, out of which could be pressed a large quantity of pulpy matter of golden or king's-yellow colour, mixed with various shades of green and brown. A quantity of the same matter, but more nearly fluid, seemed loosely effused in the substance of the liver, and some more of it was contained in the large hepatic blood-vessels. In each of the lungs, irregularly scattered through every part, there were nearly a hundred small masses of medullary matter, of the same bright yellow colour as those in the liver. They did not form any tissue, but appeared loosely effused, so that, after any one of them was cut through, each part of it could be wiped away without injuring the structure of the lung. A similar yellow substance, but apparently thinner and more nearly fluid, filled a number of the small branches of the *pulmonary artery*, in such a manner, that, on a section of the lung, fine yellow ramified lines were seen, as if the arteries had been injected with chromate of lead and size, or some thick material. There could be no doubt that the cancerous substance had been conveyed with the blood from the liver to the lungs, where, being arrested, and obstructing the minute vessels, it had permitted fresh substance, with blood, to accumulate behind it. Mr. Paget further adds, that there is a preparation from another case of the same kind in the museum of St. Bartholomew's Hospital (Diseases of the Lungs, No. 19); but the characters are less marked, because the cancerous matter is uncoloured. He has notes of a third similar case; and in another preparation in the same collection (Diseases of the Blood-vessels, No. 100) the trunk of the left pulmonary artery is filled by a large firm mass, apparently composed of fibrine mixed with cancerous matter, which extends into all the branches that are preserved. This was taken from a lady, from whom, in the course of eleven years, Mr. Lawrence removed, at different times, both breasts, and three large tumors which formed upon or between the cicatrices left after former operations. In the first breast removed (in May, 1828) there appeared only an enormous enlargement of the mammary gland; in the second (in 1834) a large tumor, resembling most the carcinoma hyalinum of Müller, was removed. In the three following operations, performed in 1835, 1837, and 1839, the tumors removed were distinctly medullary. The patient died suddenly while she seemed in progress of recovery. In the examination, the left lung was found everywhere adherent, and extremely congested; the right lung was not adherent, but pale, œdematous, and somewhat emphysematous: its vessels were healthy. There were no cancerous deposits in either lung, nor any in the bronchial glands.*

The well-known appearances which usually so strongly characterize the solid coagula found in vessels which communicate with extensive fungoid deposits, and more especially the detection of cancer cells within them, will prove the best tests of the malignant origin of these clots. It will, however, be borne in mind, that, in advanced stages of cancerous cachexia, inflammation is occasionally liable to attack many of the larger vessels of the body quite independently of any impediment produced in their channels by the lodgment of malignant debris. The subjects of advanced cancer are always liable to the occurrence of insidious inflammation of internal surfaces and organs, in consequence of operations and other vicissitudes; and in those cases the arteries and veins certainly participate in this tendency to acute and subacute asthenic lesion.

I have been favoured with the following case of morbid growth in the pulmonary artery, by my friend, Dr. Edmund L. Birkett, in whose practice it occurred. It presents several interesting features; but it appears difficult to assign the precise cause to which the formation of the polypoid mass was due.

Caroline P., æt. 25, married, but without family, inhabiting an ill-ventilated room in a badly-drained part of Bermondsey, had been seriously ill fourteen days when Dr. Birkett first visited her (Feb. 18th, 1846). She had been frequently subjected to exposure to cold, and had, in consequence, experienced many attacks of inflammatory thoracic disease. When first visited, her aspect was anxious and distressed: there was great dyspnœa, amounting to orthopnœa; slight cough, but without expectoration; pulse quick, sharp, vibrating, and without strength. She complained much of palpitation, and of pain, occasionally very severe, about the præcordial region. The heart's action was tumultuous, its rhythm natural, its impulse stronger than natural. The chest, on percussion, was resonant, and the respiratory sounds loud, with occasional dry bronchitic rhonchi. There was also slight emaciation and irritability of stomach. During the whole period of the illness there was no material change, excepting as regarded the heart; for here, at a subsequent period, was observed a to and fro sound, superficial, and most audible a little to the right of the nipple, and not traceable along the course of the aorta. About the same time, the respiration, though distressed throughout, appeared to become so far relieved as to admit of her lying in the horizontal posture. The pulse retained the same character throughout, though becoming towards the last much weaker. The irritability of stomach was relieved. At one time the micturition was accompanied by pain, owing probably to the action of the nitre contained in the medicine. Her mental faculties continued perfect to the last. The treatment was local depletion, with counter-irritation over the region of the heart, with mercury, salines, and purgatives. She died April 2d. *Autopsy.*—Head not examined. Chest: a little clear serum on the left side; extensive old pleuritic adhesions; no signs of recent pleurisy; lungs congested posteriorly, of a dark red colour, and exuding, on section, frothy serum, but filled throughout with air; bronchial tubes natural, but containing some mucous secretion. Heart: no fluid in the pericardium, which was smooth, but white and opaque at parts; the heart was large, its nutrient vessels much gorged; the cavities were large, the parietes but little hypertrophied; the mitral valve was slightly thickened, but in other respects healthy, as were also the other valves. Within the *pulmonary artery*, at its point of divisoin, was a circular space as large as a fourpenny-piece, surrounded by a ring of vegetations, to which was slenderly attached a mass of the size of a large walnut, of a yellowish colour, and, in substance, resembling the roe of a mackerel. The liver was slightly congested; so also were the kidneys; the intestines were healthy. The anterior lip of the uterus was swollen and dark-coloured.

Adherent clots may be found in branches of the pulmonary artery in various forms of long-continued direct and indirect obstruction to the pulmonary circulation*; but in the generality of cases of this kind they will be observed to be associated with evidences of the previous existence of pulmonary apoplexy.

On the occurrence of non-adherent fibrinous clots in the pulmonary artery.

Most practical morbid anatomists must have observed that in very severe cases of pneumonia, bronchitis, mitral obstruction, disease of the valves of the systemic artery, and aortic aneurism, where death has been preceded by a greatly protracted suffocative struggle, a tenacious branched coagulum of pale elastic fibrine is occasionally found to occupy the entire tract of the pulmonary afferent vessel, by no means completely filling it, and not apparently adhering to any part of the arterial parietes, which remain free from all traces of inflammatory lesion. It now appears to be generally admitted that these, as well as the condensed masses of recent fibrine which are so frequently discovered in the cavities of the heart, are probably formed some time previously to death, when the delay to the circulation is becoming extreme, and the contractile power of the heart begins to fail.

Dr. Corrigan has recorded an interesting case in which this appearance was found in the pulmonary vessels of a patient who died in the depressed stage of fever.

M. W., aged 70, admitted into Hardwicke Fever Hospital, 20th of November, 1845. For the last one or two years he had been much exposed to cold, and had suffered greatly from poverty. Fourteen days before admission, he was seized with rigors, headache, and vomiting. He became worse from day to day, lay in a small close room, and received little attendance. Symptoms on admission:—Shivering, and cold extremities; pulse very weak; tongue dry, hard, and dark-brown; all secretions and excretions scanty; countenance vacant; intelligence imperfect: he lies on his back, and makes no complaint. Treatment: ether, ammonia, and brandy. 21st, 22d, and 23d—Sinking continued, notwithstanding that he had freely of wine, brandy, ammonia, &c. He lay in bed on his back, making no complaint, with a pulse hardly perceptible. On the 23rd, the tongue was moist, with slight mucous rattle in the throat. On the 24th, he showed more restlessness, with an inclination to attempt getting out of bed. On that day he died.

Post-mortem.—The lungs were healthy in texture, with slight bronchial effusion. The tissue of the heart was healthy. The left ventricle contained grumous dark-coloured blood. A bulbous shaped polypus lay in the right ventricle, not sufficient to fill it. This polypus, narrowing gradually as it passed into the *pulmonary artery*, extended along this vessel, being about the thickness of a quill, then divided at the bifurcation of the artery, and continued its ramifications along the arterial branches. The extensive polypus of fibrine was so firm that, taking it by the root which lay in the right ventricle, it was drawn out with all its attached branches, unbroken from the artery. It was not attached any where to the sides of the vessels, but lay like a cord along their centre. Dr. Corrigan considers that the structure of the polypus shewed that it had probably been

* Op. cit. vol. xxvii. p. 165.

* Mr. Paget found deposits of this kind in the pulmonary artery in a case where the pulmonary veins were compressed by a medullary tumor. (Op. cit., vol. xxvii. p. 173). Similar appearances are also not unfrequent in various forms of obstructive disease of the left heart.

for some days in process of formation. It was very nearly destitute of the colouring matter of the blood, except in its smaller ramifications. It was not of great size, as is generally the case in polypi of recent formation in the cavities of the heart, but was firm, comparatively hard, and small compared with the size of the cavities in which it lay. Dr. C. considers, however, that the most satisfactory proof of its formation having occupied some time, is probably to be found in its structure. It consisted of distinct layers of fibrine. The outer layer or layers were comparatively soft, and when laid hold of, and pulled on, could be most readily drawn off the fibrinous cord which constituted the middle of this long tree-like polypus. This centre fibrinous cord was very firm and pale-coloured. The distinct layers into which the polypus easily separated, resembled exactly the separate layers which are found in successive deposits of lymph in the trachea in croup. It appeared evident that it had either been the cause or the consequence of the sinking of the powers of circulation. But, whether the cause or the consequence, it is plain that, when once formed, it would be a serious hindrance to the circulation, and would most probably, or even necessarily, cause death by its constantly increasing size, and the proportionately increasing obstruction which it produced. Dr. Corrigan adds a practical hint of the necessity of giving wine early in cases such as this, when the gradually failing power of the circulation is the prominent symptom in typhus fever.*

The appearances presented by the laminated fibrine in the above case, seem to have so closely resembled those which are very commonly observed in the compressed yellow coagula found in the cavities of the heart, that they can scarcely, I think, be received as evidence that the coagula began to form in the ventricle and artery so early as Dr. Corrigan conjectures. Still the possibility of such an occurrence is not to be denied, and there is certainly very strong reason to believe that coagula of this description are, in all instances, formed previously to the cessation of the heart's action. Similar consistent branched coagula are occasionally found occupying the pulmonary veins in cases of obstructive disease. Two specimens of this kind are preserved in the collection at Guy's (numbered 1520[28] and 1520[32]).*

OBSTRUCTION TO THE PULMONARY ORIFICE CONSEQUENT UPON ENDOCARDITIS.

It has been shewn, at the commencement of this paper, that, during intra-uterine life, as well as for a certain period after the commencement of respiration, while the ascending portion of the pulmonary artery forms the principal conduit through which the systemic vessels are supplied with blood, its structures are far more liable to become the seat of extensive inflammatory lesions, than are those of any other artery in the infant's body. I have alluded to the fact that where, in consequence of obstruction to the circulation through the lungs or aorta, the ductus arteriosus remains permanently open, the tissues of the pulmonary artery appear to retain much of their original liability to morbid change, and have been found to present lesions closely similar to those which are usually confined to the aorta and its valves. I have already entered into a consideration of the causes which appear to give to the commencement of the pulmonary artery in youth and adult life, so remarkable a comparative immunity from those diseases which are so frequently developed in the main systemic artery; and it is certainly most interesting to observe, that, while the left side of the heart and its vessels are liable to various morbid conditions, the gradual advance of which does not, in the majority of instances, necessarily tend to the destruction of life, until after the lapse of a somewhat protracted term of years, the corresponding structures on the right side, which, if equally diseased, would necessarily become the seat of very rapidly fatal obstruction to the circulation, are capable of bearing the most severe vicissitudes, with a remarkable degree of freedom from injurious consequences. I have been most strongly impressed with this fact in the examination of the bodies of those who have died from the remote effects of rheumatic endocarditis. During a period of nearly fourteen years in which I have had extensive opportunities of observing cardiac disease in nearly all its forms, I do not recollect to have met with any single instance in which narrowing of the left ostia of the heart, the sequel of rheumatism, was associated with any proportionate amount of lesion at the pulmonary orifice. In common with other parts of the heart, the commencement of this vessel is doubtless liable to participate in the effects of rheumatic endocarditis, but the lesions which the parts sustain appear to be so very rarely of a permanent nature, that neither my reading nor my observation affords me any definite information on the subject.†

The number of fully detailed and indisputable recorded instances of inflammatory obstruction of the pulmonary orifice, occurring subsequently to the period of infancy, and independently of original defect in the

* Dublin Hospital Gazette, Dec. 15th, 1845. Vol. ii. p. 129.

* M. Hasse considers that the presence of the opaque coagula often found in the heart is to be regarded as the immediate cause of death.

† Except in those cases where the valves have been originally malformed.

valves, is so extremely small, that I am unable to arrange them in pathological order. In fact, after a very careful review of a considerable number of cases in which it might be presumed that disease of this orifice had been set up after the heart had attained its mature development, I have only been able to select two detached instances; and, even of these, the one which I shall now quote may not be considered as by any means a perfectly unexceptionable example. It is recorded in Dr. Edward L. Ormerod's valuable paper "On a Systolic Murmur in the Pulmonary Artery, and its application to the Diagnosis of Functional and Organic Murmurs."*

W. C., aged 28, first came under the care of Dr. Bond of Cambridge, (by whom the following notes were taken). He was a waterman, of temperate habits. Subject for the last two years to palpitation, of which he then complained, with a dragging sensation in the epigastrium, and dyspnœa on exertion, but he was able to retain the horizontal position to the last. The surface of the body was of a dusky purple, but he had no notable distension of the superficial vein, nor jugular pulsation. He had anasarca and ascites from time to time, and died gradually with these symptoms in December, 1839. There was dulness on percussion in the region of the heart for a space of two inches square. The heart's impulse was increased and extended. There was a loud harsh murmur nearly synchronous with the systole, loudest and harshest to the left of the sternum in the third intercostal space, but audible all over the cardiac region, and in the epigastrium. The sounds continued audible in the same situation, but not at the same time, till his death; for the murmur after the lapse of a year was found to have become diastolic. After death the heart was found nearly twice its natural size, and the pulmonary *valves* agglutinated into a thick cartilaginous ring, leaving a permanent orifice just large enough to admit the passage of an ordinary quill. All the other valves were healthy.

It is by no means improbable that the above was a case of congenital malformation of the pulmonary orifice, which, for many years, caused little impediment to the circulation, until it became aggravated by superadded disease. As, however, it is impossible to judge with certainty of the true nature of this particular class of cases from mere description, I have thought it as well to insert it at this place.†

* Edinburgh Medical and Surgical Journal, June 1846, p. 96.

† A case very similar to this is recorded by Professor Tiedemann, in his work on Arctation and Closure of the Arteries in Disease, as quoted in the Edinburgh Medical and Surgical Journal for June 1846, p. 127.

The following case of obstructive disease of the pulmonary valves, with which I have been kindly favoured by my friend Dr. Edward Lloyd, in whose practice it occurred, is probably the most remarkable instance of the kind on record, in the extent of the disease, as well as in several of the circumstances which attended its progress: it appears to me to afford an almost unique, though most instructive, example of cardiac disease:—

Amelia S., ætat. 38, came under the care of Dr. Lloyd, on the 3rd of February, 1845. She was a dark-complexioned sallow woman, with a very anxious and haggard expression of countenance; she was by occupation a work-woman at a dry-salter's, where she had been chiefly employed in packing galls, in lifting heavy weights, and in having to go up and down flights of stairs. She had always been an active person, and in these habits continued until confined by her last illness. Her habits were intemperate, and of late they had been more so than usual. In the preceding October, she was run over, but it was not certain that she received any material injury; still her head was cut, and her husband maintained that from that time she became less healthy than formerly. She, however, returned to work, and, up to Christmas-day, continued her usual avocations with her accustomed activity, frequently carrying sacks of two hundred-weight up and down stairs throughout the whole day. She had been subject to winter cough, and her husband appeared to think that she used sometimes to have attacks of fainting previously to her last illness, but her employer maintained that she was never short-breathed, and that he had never heard of her being faint: his expression was that "she could do the work of a man." Her attack commenced on Christmas-day, or soon afterwards. On that day she had a quarrel with her husband, when it is stated that he struck her several blows on the side, and she, from that time, complained of pain in that situation. This, however, does not appear to have attracted much attention, except that she was unable to go to work. On the Sunday following, she had another quarrel with her husband, when the neighbours interfered. On Monday she was very unwell, and on Tuesday she took to her bed, complaining of pain in her left side, which she ascribed to a blow from her husband. She now sent for Dr. Jewel: that gentleman observed the presence of a cardiac bruit. On the 3rd of February, when Dr. Lloyd first saw her, she had been ill five weeks; she lay on her left side, with an anxious expression of countenance, and peculiarly harassed though not difficult or laborious respiration, with every now and then a long deep-drawn breath, followed by a number of short inspirations.

Respirations 36-40. Pulse 130, very small, sharp, and slightly irregular. She complained of pains in her limbs, knees, and ankles, but particularly in the right arm, on examining which an abscess was subsequently found over the biceps, but she appeared to suffer chiefly from a severe pinching pain, which sometimes gave the sensation of a knife going through her on her left side. Skin hot; bowels relaxed; sensorium undisturbed. She was extremely irritable. The hurry of respiration increased when she was raised. She complained of cough, with some expectoration of a mucous character, not allowing her any rest from the pain it caused in her left side. She was said to have eaten nothing for a week, had no desire for food, and could not sleep at night. The extreme hurry of respiration was very remarkable indeed. The chest was resonant throughout; the respiration coarse and loud at the apices of the lungs,—there was no abnormal pulmonary sound, with the exception of a slight mucous râle heard over the right scapula. The heart's sounds were indistinct, heard more clearly at the apex than at the base: in the latter situation there was an abnormal sound, (which appeared to Dr. Lloyd to have the characters of a pericardial *frottement*), heard over a space of about two inches in diameter from the right nipple to the sternum, but not at all audible in any portion of the course of the aorta or carotid arteries. This sound was superficial and loud, but soft, with a distinct back stroke. No fremitus could be felt. She was ordered calomel and opium at night: serpentary and ammonia, and sago, beef-tea, and calves'-foot jelly.

On the 4th, she was much in the same condition; had passed a very restless night; her cough was troublesome; she expectorated frothy mucus tinged with blood. The abdominal cardiac sound was unchanged; the respiration was still extraordinarily hurried. Pulse 135, small and sharp. The urine was found to be slightly coagulable. There had been one loose motion during the night. The liver could be felt below the ribs. She now complained of pain in her right side on auscultation: a slight rubbing was heard in this situation. Emplast. Lyttæ, lat. sinist.; Hydrarg. Chlor. gr. i.; Opii, gr. i. nocte, et P.

On the 5th, she felt better; her face was less anxious. She had passed a comfortable night, and slept a good deal during the day. The skin was perspiring; she was extremely weak, and when she got out of bed she was obliged to lie on the floor to avoid fainting. She had done this for some time during her illness. The tongue was clean and moist; the blister had risen well. Pulse 130, small, and irregular in the duration of each beat. The cough was relieved, but respiration was still hurried. After a severe fit of coughing she became very cold and faint, and hot bottles and flannel were necessary to keep her warm. Her appetite was much improved: she had taken calves' foot jelly. The right arm was painful; the pain in the legs was less. P. M. Habt. Vin. ℥iv.

6th.—Was much agitated on the preceding night; since which she continued to grow worse. The countenance was more sunken; the respiration very hurried; pulse slower, 100, and intermittent; the cough troublesome; the heart's sounds were much more indistinct; and the abnormal murmur feeble. There was some dulness at the base of the left lung, with some œgophony. The tongue was rather brown; lips dry and parched; sordes on the teeth. She had not had the wine ordered, which was prescribed at last visit. Brandy ℥iv.; Infus. Serpentariæ C. et Tr. ejusd. c. Ammon. Sesquicarb. gr. v. quartis horis.

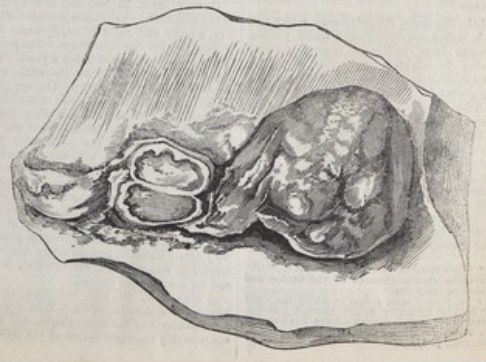
On the 7th, she was much lower; the abnormal cardiac sound was heard over a smaller space; the heart's sound was irregular and indistinct. Respirations, 40. Pulse, 114. Tongue brown; teeth covered with sordes.

On the following day she was much the same; the pulse was slower, about 90, very weak, and hardly to be felt. Respiration was very hurried; there was subsultus, and she was evidently sinking. She died on the morning of the 10th, having sunk gradually.

Autopsy.—The body was very spare and thin. The abscess on the fore part of the right arm was found to contain about an ounce of sanious pus, with some old coagula of blood. Recent and soft pleuritic adhesions were found on the right side of the chest, with pneumonic consolidation, approaching the stage of grey hepatization, of a small patch in the centre of the lung. The base of this lung presented a somewhat congested appearance, which was probably cadaveric. Both lungs, when cut into, yielded a large quantity of serum, mixed with air-bubbles. On the left side there were a few old pleuritic adhesions, with some fluid in the pleural cavity; the lung itself presented no other morbid change than œdema. The pericardium contained about two drachms and a half of fluid, and was perfectly healthy. The heart was of a normal size, and not at all gorged with blood. The left side was healthy; both aortic and mitral valves were sound. There was no hypertrophy or dilatation. The tricuspid valve was healthy. The right auricle contained a coagulum, but was not distended; the right ventricle also contained a small coagulum. On examining the *pulmonary artery*, a hard body was felt considerably obstructing its canal, but a probe was easily passed, and could be moved from side to side on the anterior wall

of the artery; it could also be passed between two bodies which blocked up the valves. On carefully laying open the artery, its orifice was found to present two tumors and one valve; the tumors corresponding to the posterior valves, and having a crescentic surface on their ventricular margin. One of these masses was about double the size of the other, the smaller being about the size of a large walnut. When placed in situ, it was found that each had smooth corresponding surfaces, evidently produced by friction between the two bodies. They were, externally, of a dark grey or brownish colour, having generally a somewhat warty and roughened surface. On laying them open with a scalpel, they exuded a gelatinous fluid of a very tenacious character. They were hard, and apparently organised, with fibres arranged in small convolutions. They were light-coloured internally: their bases or ventricular margins were apparently in a state of inflammatory softening, breaking down on the slightest touch, and separating from the substance of the artery without difficulty. The gelatinous fluid was examined by the microscope, and found to contain nucleated cells, not of a malignant character. The remaining valve was covered with some shaggy vegetations or fibrinous deposit: it was of normal size, and smooth and healthy on its arterial or inner surface. The coronary arteries were healthy; there were two openings to either artery. The omentum was attached to the abdominal parietes by old adhesions. The liver was much enlarged, stretching down to, and encroaching on, the umbilical region: it was congested and coarse in structure, and adherent to the abdominal parietes. The spleen was also large. The kidneys were larger than usual, and of coarse structure; their tunics were adherent, and they were very lacerable and pale. The ileum was congested, and presented some spots of ecchymosis: it contained nothing but a small quantity of bilious mucus.

Among other valuable remarks which Dr. Lloyd has appended to his report of the above case, he observes that it proves how great an amount of obstruction may continue to be borne on the right side of the heart, provided the lungs are sound, and in working condition. He considers that the heart itself shewed no evidences of obstruction, as there was neither hypertrophy nor dilatation, nor even engorgement of its cavities: in fact, there was a singular absence of marked evidences of cardiac obstruction both before and after death. There was no impediment to the return of blood from the jugulars, no blueness of the lips, congestion of the countenance, or œdema of any part of the body. Dr. Lloyd very justly accounts for this absence of cardiac engorgement and external congestion in two ways: first, the quantity of the circulating fluid was small, the patient being thin, spare, and at rest, and must have become smaller and smaller as the disease of the valves increased, from the woman's taking no nourishment of any kind, and also from the reducing effects of treatment; and, secondly and principally, he thinks this circumstance accounted for by the remarkably hurried action of the lungs, whereby a small quantity of blood was sucked up by atmospheric pressure from the ventricle at every respiration. This, frequently repeated, he believes, answered the purpose of the natural and slower respiration, and larger supply.



This remarkable specimen is preserved in the collection at Guy's (numbered 1413[52].) The artery has the appearance of being much dilated. Large portions of the clots have been removed, but the remaining masses are still of extraordinary size. All the valves are ruptured at their lower attachments, but this has occurred subsequently to removal, owing to the extremely softened condition of the parts.

The preparation marked 1413[50] in Guy's museum is the base of a heart in which the valves are more or less generally coated with layers of recent inflammatory product. Those portions of the ventricular surfaces of the *pulmonary* sigmoids which remain free from fibrinous laminæ appear to be pliable, and free from thickening or marked opacity. I have not succeeded in obtaining any history of the case.

Like the aortic sigmoids, the valves of the pulmonary artery occasionally, but rarely, present rows of minute semitransparent wart-like vegetations, which are usually arranged in the form of double festoons at the lowest and most projecting parts of their surfaces of contact. I have elsewhere stated* my grounds for believing that these little bodies are not mere adventitious deposits from the blood, but organised growths from the endocardial surfaces, and that, under circumstances of disease, they are intended to protect the outlets of the heart from undue attrition, as well as from the deposition of clots, and also to prevent the occurrence of adhesion between opposed surfaces of inflamed valves.

OBSTRUCTION TO THE PULMONARY ARTERY CONSEQUENT UPON EXTERNAL COMPRESSION.

Like the aorta, the pulmonary artery and its branches are occasionally found passing through large malignant tumors without having the slightest appearance of having been injuriously compressed by the surrounding growth.† Still the calibre of this vessel is liable to be seriously encroached upon by various morbid growths, and accumulations in the neighbourhood of the heart—such as pericardial deposits, large fluid effusions into the thoracic cavities, aneurism of the aorta, &c. The following cases afford interesting examples of these rare forms of obstruction:—

An instance is recorded in the Dublin Journal for May, 1841, in which extensive and thoracic effusion, the result of double pleurisy suddenly supervening upon chronic bronchitis, produced so much compression of the heart as to render it probable that death had resulted from complete flattening of the right ventricle. For eighteen hours previously to the unfortunate patient's death he suffered from frightful dyspnœa. The state of the pulmonary valves is unfortunately not described.

Compression of the base of the right ventricle and origin of the pulmonary artery by an aneurism of the ascending aorta.—About eleven years since, I watched the case of a middle-aged man, who was admitted to Guy's Hospital, with a pulsating aneurismal swelling upon the left side of the sternum. He suffered from hæmoptysis and extreme dyspnœa, and died in about a week after his admission. Upon examination, a large aneurismal pouch was found arising from one of the sinuses of Morgagni, and pressing forwards the base of the ventricular septum in such a manner as to place it completely in contact with the outer wall of the right ventricle: in this way the orifice of the *pulmonary artery* had become nearly flattened. Anteriorly this was completely the case; the left pulmonary sygmoid valve had become adherent by a fibrinous clot to the most projecting part of the tumor. At the parts where the compression must have been greatest, and where the surfaces must have been placed completely in contact with each other, the endocardium was deeply reddened, and, in parts, coated with fibrinous coagula. The heart is preserved in the pathological collection at Guy's, (numbered 147825).

The two following interesting parallel instances have been recorded by Dr. Elliotson:—

Owen S., ætat. 39, had been ill five years. When admitted, he had ascites, anasarca of the legs, a quick and rapid pulse, dyspnœa and palpitation, but *could lie down*. The palpitation and dyspnœa had lasted a year. The jugulars and other veins of the neck were distended to a great degree. On applying the stethoscope to the right side of the heart, or upon the sternum, a whizzing sound *(bruit de soufflet)* was heard, and it was ascertained, by feeling the pulse, that this sound was synchronous with the contraction of the ventricles. The principal *post-mortem* appearances were as follow:—The pericardium was adherent to the heart, and contained some portions of cartilage: there was a cartilaginous body in the substance of the wall of the right ventricle, where the pulmonary artery leaves it, and the artery was contracted in size to that of the brachial, there, and for some inches beyond.

A man, ætat. 60, who had been out of health some months, suffered from orthopnœa, anasarca of the arms, thighs, and legs, considerably increased action of the carotids and radials, and distension of the veins of the neck, with tenderness of the epigastrium. A loud and distinct *bruit de soufflet* was heard at the upper part of the sternum, at the moment when the ventricles contracted, proving that the obstruction must be at the outlet of one of those cavities, while the situation in which the noise was heard, and the distension of the veins pointed out the right as the one implicated. The only material differences between these two cases were, the circumstance that in the former the patient could lie down, while in the latter he could not, and the increased action of the carotid and radial arteries in the latter. On examination, the pericardium was found adherent to the surface of the heart in every part; the heart itself was enlarged to twice its natural size, and its substance was very much softened, and so changed in texture as almost to have lost its fibrous appearance. A part of this change might be owing to the decomposed state of the body, but certainly not all of it. The walls of the cavities were thickened, but not in proportion to the increase in size of the whole heart; the cavities themselves, and especially those on the right side, being much dilated. At the origin of the *pulmonary artery*, a fibro-cartilaginous structure, as large as a small egg, was found almost surrounding the artery, which was so much diminished in calibre that it would scarcely admit the little finger; beyond, the artery retained its usual size. There was also found to be a large aneurism of the aorta, which had burst.*

Obliteration of the right pulmonary branch by an aortic aneurism.—The collection at Fort Pitt contains a preparation taken from the body of a corporal, about 33, who died of phthisis pulmonalis, in which the right branch of the *pulmonary artery* is completely closed at its origin by the pressure of a small aneurism arising from the concavity of the aorta, near its base. The closure was permanent from adhesion. The inner coat of the pulmonary artery contiguous, had lost its natural smoothness. This disease of the vessels was never suspected during life, the symptoms being only those of ordinary phthisis†.

Congenital narrowness of the pulmonary artery.—Dr. Barlow has described a highly interesting class of cases of young patients who have suffered from birth from an imperfectly developed condition of the respiratory apparatus, the thorax being more or less narrow or deformed, the trachea small, and the lungs ill expanded, who, at or about the period of puberty, are liable to become the subjects of grave cardiac symptoms; and eventually to die from the effects of obstruction to the circulation. In such cases it is generally found that the left cavities of the heart and aorta are either normal in size or below the natural capacity.* The right cavities are more or less dilated and hypertrophied, while the pulmonary artery remains small, and, what is very remarkable, has occasionally been found by Dr. Barlow to be really below its usual standard capacity. This obtains so long as the vessel remains healthy.

I have myself observed a few instances in which the right ventricle had become hypertrophied in consequence of the long obstruction, and in which the pulmonary artery remained small, independently of any apparent organic fault in its texture;† but it is, undoubtedly, far more usual to find this vessel more or less dilated, in cases where the circulation through the lungs has been long and severely impeded.

The tissues of the pulmonary artery, being naturally far more extensible than are those of the aorta, probably do not undergo precisely the same changes of dilatation, &c., coincidently with the occurrence of hypertrophy of the right ventricle, as do the structures of the aorta under parallel circumstances. The right ventricle appears to be capable of becoming hypertrophied before the pulmonary artery has undergone any remarkable or proportionate dilatation; and this may be especially the case in those instances where (as Dr. Barlow argues) the vessel is small from originally faulty development. Should the patient's muscular system be weak (as usually happens in this class of instances), it is probable that the ventricle will also become dilated before a similar change occurs in the artery: the muscular tissue of the former depending more for its tone upon nervous influence than does the elastic fibrous structure of the other. The presence also of a freely acting safety valve on the right side of the heart, and a healthy condition of the abdominal vessels, will, at the commencement of these cases, have considerable influence in relieving the pulmonary artery; while, on the contrary, a close tricuspid, and obstructed abdominal circulation, would tend greatly to hasten its dila-

* Guy's Hospital Reports, vol. i. N. S., "Observations on the Diseases of the Orifice and Valves of the Aorta."

† Dr. Sims relates a case in which the right division of the artery passed through a malignant tumor, and was much dilated, but retained its texture. Medico-Chirurgical Transactions, vol. xviii. p. 290.

* Medical Gazette, vol. x. p. 221.

† Third Fasciculus of Anatomical Drawings selected from the Collection of Morbid Anatomy in the Army Medical Museum at Chatham. Plate vi.

* In some cases the left auricle and ventricle are dilated, while the aorta remains small, although free from any traces of organic disease.

† This has not merely occurred in very young individuals; in the case of the middle-aged man, the state of whose lungs I have described in the chapter on pulmonary apoplexy, the orifice of the pulmonary artery was nearly of the ordinary capacity, although some of its branches were greatly obstructed by old coagula. The heart was hypertrophied, and the aorta somewhat dilated.

tation. It must also be borne in mind, that in cases of this description death is not generally to be ascribed to the mere obstruction produced by the imperfect development of the pulmonary apparatus, but that it is usually caused by the superaddition of a certain amount of inflammatory or other disease in the originally defective lungs, which hastens on the fatal result long before the heart and its appendages have undergone those changes which they would ultimately have presented had life been considerably prolonged.

It is therefore not to be considered that the instances in question by any means disprove the generally prevailing rule, that obstruction to the pulmonary circulation has uniformly a tendency to produce dilatation of the afferent vessel of the lungs. And it is of course almost unnecessary to add that they do not in the slightest degree prove that pulmonary obstruction can, under any circumstances, have a tendency to produce a diminution in the capacity of this artery.

Dr. Barlow has also shewn that a condition of parts similar to that described in the above class of cases may result from the occurrence of adhesion of the pericardium at a period of life when the development of the thoracic organs is still incomplete.* I have also brought forward a series of instances which illustrate the fact, that complete adhesion of the pericardium, occurring in adult life, has a tendency to produce diminution in the size of the heart and its vessels in all cases where there is no valvular disease, and the pulmonary and systemic circulations remain tolerably free.†

All morbid anatomists are now acquainted with the fact, that in the majority of cases of pulmonary consumption the cavities of the heart are found considerably below the ordinary standard capacity; and that the pulmonary artery and aorta usually, to a greater or less degree, partake in this change.

Pathologists are indebted to Dr. P. M. Latham for an explanation of the remarkable and important principle upon which this adaptation is effected.‡ It is observable that in the larger proportion of cases of phthisis, even long after nearly the entire structure of both lungs has been rendered irrespirable by solid deposits and purulent excavations, there is usually, while the patient remains unexcited and at rest, an almost entire absence of severe dyspnœa. This often continues even up to the period of dissolution: the patient dying from hæmorrhage, exhaustion, cerebral complication, —or, in fact, from almost any other cause than mere suffocation. This is shewn to depend upon the very small quantity of blood which is propelled, at each systole of the contracted right ventricle, through the pulmonary vessels, as well as to the rapidity of its transit through the narrowed cavities of the heart. The colliquative sweats and diarrhœa, which are such frequent attendants of the later stages of this disease, are therefore not to be regarded either as processes which are wholly injurious to the system, or as altogether dangerous symptoms which it is absolutely necessary to repel by vigorous treatment: they must, on the contrary, be viewed as the principal means which nature adopts to relieve the heart and lungs from a large proportion of the circulating fluid—a relief which the latter organs require, not only in consequence of the great diminution of their aerating surfaces, but also on account of the impaired action of the bronchial exhalents which is usual in this disease.*

It is doubtless owing to a similar diminution in the bulk of the circulating fluid that the circulation through the lungs is usually so free in cases of malignant disease of those viscera. Large masses of softening cancerous deposit are occasionally found occupying very extensive portions of the pulmonary structure in the bodies of patients who have scarcely been at all subject to dyspnœa, and even in cases where no symptom has occurred to lead to the recognition of the disease during life. In such cases as these the heart and its vessels have usually been observed to have adapted themselves to the diminished volume of the blood.

Narrowing of branches of the pulmonary artery in the vicinity of phthisical cavities.—In extensive tubercular degeneration of the lung, even after softening and suppuration have commenced, it is often singular to observe branches of the pulmonary artery, of various diameters, passing through the apparently disorganised lung, their interiors remaining entirely free from the slightest discolouration or deposit, although the canals

* Guy's Hospital Reports, vol. v., New Series.
† Guy's Hospital Reports, vol. i., New Series.
‡ See Lectures on Diseases of the Heart, MED. GAZ., vol. iii.

* It has been observed by one of our leading pathologists that he regards a small heart as a bad coincidence in the case of tuberculous disease, adding, that he would rather have a large heart than a small one in connexion with phthisis. —[Clinical Remarks on a case of Tabes Mesenterica, by Dr. C. J. B. Williams: MED. GAZ., vol. xviii., p. 1490.]—I cannot but venture to prefer the doctrine of Latham to that of this high authority. It will, I believe, be usually observed that in cases of phthisis where the heart is large the respiration has been proportionably difficult, the pulse comparatively slow, and the patients to a greater or less degree subject to that depression of spirits from which the rapidity of the circulation in phthisis usually renders the victims of this hopeless malady so providentially free. The condition of the phthisical heart cannot be regarded as one of atrophy: all its cavities are usually small and well contracted; and the muscular tissue of its walls is firm, and well adapted to maintain short and rapid propulsive movements.

have evidently been in some degree encroached upon by the surrounding effused matters. In more advanced stages of disease, however, when phthisical excavations have become completely formed, complete obliteration of the arterial tracts, which are thus left in a state of isolation, generally occurs, while those portions of the closed arteries which intervene between the point of occlusion and the nearest pervious branch undergo a remarkable degree of contraction, which leaves them in the condition of very narrow conical cul-de-sacs. This latter change has been very accurately described by Dr. Baillie, who remarks, that when blood-vessels are traced into abscesses of the lungs, he has found them very much contracted just before they reach the abscess, so that the opening of their extremites has been closed up entirely. On such occasions it will require a probe to be pushed with a good deal of force, in order to open again their extremities. In these contracted vessels, he adds, the blood is coagulated as it is under similar circumstances in other parts of the body. This change of the blood-vessels he regarded as designed to prevent the occurrence of large hæmorrhages, which would certainly prove almost immediately fatal.

My own observation has led me to believe, with Cruveilhier, that the obliterated trunks of pulmonary vessels much more frequently form the principal substance of the bands which traverse the cavities of many phthisical excavations than the statements of Laennec and Andral would lead us to suppose. While the excavations are of moderate size, the obliterated arteries are usually seen passing across the cavities, in the form of greyish cords, covered externally with a coating of pus, fibrine, and particles of tubercular matter, and presenting, on transverse section, a close grained coagulum, having the appearance of soddened glue, perfectly identified with their degenerated tissues, and completely occluding their canals. These obliterated vessels are easily broken across, their structures having lost their tenacity; and, as the cavities increase in size, the isolated portions appear to become removed by sloughing, leaving only rounded extremities, which form slight elevations on the sides of the vomicæ.

It appears that in the generality of chronic phthisical excavations, the portions of vessels which pass across the cavities become perfectly obliterated. It is in the last degree improbable that any artery which has long remained in this manner completely isolated should ever remain sufficiently pervious to become either the seat of aneurism or the source of hæmorrhage. So far as I have observed, or can learn, these accidents are only liable to occur in vessels which are situated upon the walls of cavities, and which are not completely isolated. Still, it is not improbable that, where the excavation is very rapidly formed, a considerable tract of artery may become detached before the process of obliteration has been completed, and may then either suffer perforation or be actually separated during a violent paroxysm of coughing, as is described to have been the case in a somewhat marvellous case described in the Ephemerides Nat. Cur.

Dilatation of the Pulmonary Artery.

The fact that as life advances the pulmonary artery, in common with all other parts of the vascular system, is liable to become gradually and permanently dilated;* and that the establishment of fixed causes of obstruction, either in the lungs, left heart, or systemic circulation, are at all periods liable to produce a similar result, have already been commented upon in previous chapters of this memoir. It now merely remains to cite a few examples in which remarkable dilatation of this vessel was found to have occurred under somewhat unusual circumstances.†

We have seen that an unnaturally wide condition of this vessel is found in several classes of cases of cardiac malformation. The following instance presented at once the largest amount of simple dilatation of this vessel that has ever fallen within my notice, and one of the most singular congenital irregularities that has been known to occur in the heart of an adult:‡—

* General dilatation and loss of elasticity are the only visible physical changes to which the tissues of the pulmonary artery appear to be liable in extreme old age, independently of the operation of accidental morbid influences. In most of the hearts of very aged persons which I examined, the structures of this vessel retained, to the eye, a nearly healthy appearance; the valves especially presenting a striking contrast to those of the aorta. Morgagni describes the condition of the heart in a man of 98, in whom all the valves belonging to the orifices of the right ventricle were not only not rigid nor thickened, but even like those which belong to the pulmonary artery, they seemed to be formed of a thinner membrane than usual. The mitral valves, however, were found thicker than they ought to be; and the aortic valves were all bony, inflexible, and protuberating internally, &c. The arch of the aorta and its vessels were free from bony scales, which were however seen in the remaining part of the aorta.—Letter XXI. A. 15. It appears, in fact, that degeneration of the tissues of this vessel is not by any means a necessary attendant of old age, but is most frequently the result of over distension and other accidental morbid influences.

† Cases of too great capacity of the pulmonary artery are described by Cooper (in Farre), Richerand, Corvisart, Testa, J. F. Meckel, Hufeland, Lawrence, Young, &c.

‡ Full details of this case have been published by Mr. Alfred Taylor, in the MEDICAL GAZETTE, vol. xxxvi. p. 19.

A woman, 45 years of age, dropped suddenly in the street, near Guy's Hospital, and died, gasping and pallid, without any convulsions, immediately after she had been brought into the house. Some suspicion was entertained that she had swallowed about a drachm of nux vomica powder an hour before her death, but this was not proved to have been the case. I was present at the autopsy, July 12th, 1844. The body was lean, but scarcely had the appearance of being wasted. The extreme parts of the body were remarkably blue. Some of the muscles, especially the internal ones, were very pale. The chest was much deformed, the ribs being compressed laterally below the præcordial region, apparently by the tight lacing. The pericardium contained a considerable quantity of clear fluid. The heart was very large, owing entirely to the dilated condition of its right cavities, which were distended with dark semi-fluid coagula. The pulmonary artery was very conspicuous; there were some white patches, like cicatrices, upon its left side; posteriorly it was adherent to the aorta by strong bands.* The auricles communicated by an oblique opening capable of admitting the ends of three fingers; in the remains of the septum below this was an oblique passage, closed on the side of the left auricle; this was evidently the vestige of a foramen ovale: therefore there must have been originally two of these communications in this individual—one of which had closed, the other remaining permanently open. Four pulmonary veins were found opening into the right ventricle, as did the common termination of the coronary veins, which was furnished with a well-formed valve. The right auricle was very capacious. The tricuspid valve was not malformed. The right ventricle was capacious and thick. The *pulmonary valves* were bordered with a few vegetations; their tissue was unusually thick, but still retained its pliability. Immediately below its valves the orifice of the artery measured four inches and four lines in circumference; at the upper attachments of the valves it was about one line wider (the normal measurement of these parts being respectively 3$\frac{3}{4}$ and 3$\frac{2}{4}$ lines). Notwithstanding this dilatation of the vessel, there was no reason to doubt that its valves were perfectly efficient. The left auricle was less capacious than the right. The left ventricle appeared remarkably small when compared with the right; its parietes were by no means thin, but their muscular structure was soft. The aortic orifice was structurally healthy, but it was remarkably narrow, measuring somewhat less than two inches below the valves. The vessel was small throughout its entire course; but, immediately below the left subclavian artery, it was in a marked degree more contracted than elsewhere. Several enlarged bronchial glands were found near this spot; these may have encroached slighty on the aorta, or the dilated pulmonary artery may have somewhat compressed it, but there was no distinct structure here: and the undilated state of the left ventricle and ascending aorta proved that this narrowing of the vessel had not caused much impediment to the circulation. Neither the internal mammary, nor the intercostal arteries were perceptibly dilated. The lungs were in parts highly emphysematous, and were generally much congested. The liver was congested. The spleen small, and surrounded by adhesions. The kidneys were small, and tolerably healthy.

It was ascertained that this person was one of twins born at the eighth month. She had always been liable to shortness of breath, and to become blue upon unusual exertion. Her habits had been irregular and intemperate.*

It is evident that this female must have always been liable to great pulmonary congestion; and it is certainly most extraordinary, considering the nature of the congenital defect of her heart, and the artificial deformity of her chest, that she should have reached the middle period of life. It will be observed that in this case the orifice of the pulmonary artery had become widened to a degree considerably beyond that to which it is naturally dilatable—a circumstance to which I have alluded at the commencement of this paper, as producing a tendency to sudden death, upon the occurrence of any circumstances which occasion over-distension of the right cavities. It is evident that the degree of mental perturbation which could have urged this unfortunate individual to procure the means of committing suicide, was amply sufficient to produce fatal embarrassment of a heart so weakened and malformed.

* It is known to all anatomists that in some subjects the pulmonary artery is united to the ascending aorta, and that in others a considerable interval is found between these vessels.

* A case somewhat similar to this occurred to Mr. Harrinson, who, in 1844, presented to the Reading Pathological Society the heart of a child two years and two months old, who had been the subject of cyanosis. The right side of the heart was enormously distended with clots of fibrine; the auricle and ventricle were greatly enlarged. The auriculo-ventricular orifice was so much enlarged or dilated as to annihilate the functions of the valves. The *pulmonary artery* was greatly dilated. The ductus arteriosus was impervious. On the left side the auricle was very small, and the foramen ovale open; its edges rounded, and no valvular apparatus; the auricular appendages absent. No appearance of the entrance of any pulmonary veins; left ventricle very small. Mr. Harrinson conjectured that the pulmonary veins must have terminated either in the vena azygos or in the superior cava.

The average circumference of the pulmonary artery, at its widest part, is about 38 lines. Dr. Hope has described a case in which this vessel measured five inches half way up, and four and a half near the valves. Here the sygmoids were stretched, and were considered to be inefficient.* In such instances as these, the duration of the patient's life must obviously depend chiefly upon the strength of the right and left ventricles, and the degree of safety-valve action of the tricuspid.

It is usual to find the pulmonary artery with its valves more or less (sometimes extremely) dilated, coarse in structure, and irregularly thickened and opaque, in cases of old bronchitis and extensive emphysema of the lungs, &c., as well as in instances where death results from mitral and aortic obstruction.†

In cases of old bronchitis, mitral and aortic obstruction, &c., it is usual to find the entire tract of the pulmonary artery more or less inordinately dilated. In the generality of examples of this change, the branches of the vessel are observed to have undergone the greatest amount of structural alteration, presenting an almost varicose appearance, their anterior being thickened and opaque, and discoloured by striæ and patches of atheromatous deposit. In more extreme examples, the main trunk and valvular apparatus of the artery are also found to have undergone, in addition to their dilatation, a marked degree of thickening, and to have become the seat of a certain amount of opaque interstitial deposit—appearances which deprive the vessel of its characteristic appearance, and give it an aspect very closely resembling that of the ascending aorta. The following case, with which I have been kindly favoured by my friend Dr. Peacock, affords a very excellent example of the class of lesions now under consideration.

* I have never myself seen any case in which the pulmonary valves had become inefficient in consequence of retroversion, or from any other effect of dilatation independently of mechanical violence or congenital defect.

† Instances of too great capacity of the pulmonary artery are also described by Cooper (in Farre), Richerand, Corvisart, Testa, J. F. Meckel, Hufeland, Lawrence, Young, and others. Otto has also given the following references to recorded cases of aneurism of this artery.—And. Cæsalpin. Catoptrices, Lib. vi. cap. 20; Blancard, Anat. Pract. Rat. Cent. ii. Obs. 74; Fabrig. von Hilden, Observat. et Cur. Chir. Cent. ii. Obs. 89; Eggerdes, in Misc. Acad. Nat. Cur. Dec. 11, An. VI. 1687, p. 415; Morgagni, Epist. XXIV. Art. 36; Matani de Aneurismal. Præcordior. Morbis, p. 123; Bander, Observat. Med. Incis. Cadav. Illust. Frib. 1765; Bach v. Richter's Chir. Bibliotheck, vol. viii. p. 498; Essenchmidt, in Schmucker's Vermischt Schriften, vol. ii. p. 241; J. C. Stark, in Abhandlungen der Erlangen Physic zu Alexander, Trans. vi. 765.

Wm. Cassidy, æt. 72, cooper, admitted into the Royal Free Hospital, July 3d, and died July 5th, 1847. He was dying when I saw him. The face was pale, the cheeks livid, and the jugulars were distended and pulsating. The hands were cold and livid. He was greatly emaciated, and the abdomen and the cellular membrane, of the lower extremities more especially, were distended with fluid. The chest sounded dull on percussion of the level of the nipple on the left side; and on the right, from a somewhat lower point, a loud and rough systolic murmur was audible at the præcordia, and was followed by a flat second sound. This sound was most intense an inch and a half to two inches below the nipple, and external to a vertical line drawn from it. The pulse was unequal in strength, and occasionally intermitted. No history of his previous illness could be obtained.

The brain was below the average weight, or only 42 oz. 4 dwt. avoirdupois. There was some fluid beneath the arachnoid membrane and in the ventricles.

A considerable amount of fluid was contained in each pleural sac. The lungs were partly compressed by the fluid, and partly emphysematous: on section, they yielded much spumous fluid. The bronchi were of large capacity, and the cartilages unusually hard and thick. The dilatation extended throughout the bronchial ramifications, and the mucous membrane was generally reddened, and the tubes filled with a glairy mucous fluid.

There was a large, thick, and opaque patch of old lymph on the anterior surface of the right ventricle, which, when removed, left the pericardium beneath it smooth and glistening. The right auricle was studded with small nodular concretions of old lymph. There was but little fat in the heart. The heart weighed 14 oz. There was great dilatation and hypertrophy of the right ventricle, the cavity being 4 in. and 3 lines in length, and its walls three lines thick at the base; four and a half at the mid-point, and one and a half at the apex. The tricuspid aperture was very large, allowing of the passage of a ball measuring five inches in circumference. The pulmonary orifice was also extremely large, allowing of the passage of a ball measuring three inches and nine lines in circumference. The tricuspid and pulmonic valves were somewhat thickened, but not otherwise diseased. The trunk of the pulmonary artery was very much dilated, and the sinuses at its origin especially so. Its branches were throughout very large, and, though the lining membrane of the trunk was free from appearances of disease, that of the primary and secondary divisions, and the smaller branches, was the seat of extensive atheromatous deposit. In the smaller vessels the coats were

so much thickened that on section they remained wide open, and in some there were small, hard, and partly adherent pinkish-coloured coagula. The left ventricle was also hypertrophied and dilated, measuring three inches in length, and from six lines to two and a half lines in width. The mitral valve displayed some mammillary thickening, and the three fold was somewhat atheromatous. The aortic valves were thickened at their free edges, but not incompetent. The aorta around the angles of the valves was extensively ossified, and the orifice of the right coronary artery was nearly closed by a deposit of this kind. The other coronary artery, and the right throughout the remainder of its course, was not materially diseased.

The lining membrane of the aorta was extensively studded with atheromatous patches. The sinuses of Valsalva were much dilated, and the aorta generally was large, and its ascending portion elongated.

The liver was large, lobulated and coarsely granular. The kidneys large, mottled, irregular in shape, and studded with smaller or larger sized cysts.

The peritoneal cavity contained much fluid.

The occurrence of atheromatous deposit in the pulmonary artery, and especially in the branches of that vessel, is now generally recognised as a by no means unusual pathological appearance: it is usually associated with a greater or less amount of hypertrophy or dilatation of the right ventricle, and with a proportionate degree of congestion of the general venous system. These latter abnormal conditions may exist either as the direct results,* or as the mere coincidents of the arterial lesions.

Aneurism of the pulmonary artery.

The occurrence of aneurism in this vessel has rarely been observed: the great dilatability of the ascending portion of the artery appears to be the principal cause of its immunity from this lesion, while its internal branches are still further protected by the elastic support afforded by the pulmonary tissue. Indeed, when we take the true nature of aneurism into consideration, it becomes probable that the morbid appearances which will be described in the following cases were not due to a process strictly analogous to that by which aneurisms of the systemic arteries are produced: in fact, the condition appears to be more nearly allied to a form of varix which occasionally affects the larger veins.*

The following case occurred in the practice of Dr. Fletcher, of Birmingham†:—

A thin, pale girl, æt. 19, a screw-wormer, was admitted to the Birmingham General Dispensary. She complained of violent pain in the head, and of some pain in the left side of the chest, great dyspnœa, and trouble-some cough. Immediately under "(on ?)" the left of the sternum, between the second and third ribs, there was dulness on percussion over a space measuring nearly four square inches. Over this spot a very liquid pulsation was felt, accompanied by a purring thrill, so rough and so close to the surface, that it seemed almost to grate on the fingers. At the same place a hollow rasp sound was heard with the systole of the heart, and also for a moment at the commencement of the diastole, as if the blood was receding. The patient lived a year after these symptoms were observed, and, after repeated attacks of bronchitis and pneumonia, died rather suddenly.

Inspection.—The *pulmonary artery* was found dilated into a pouch, the interior circumference of which measured nearly six inches, and which pointed principally in the anterior direction, where the internal and middle coats were wanting, and where a fibrinous clot was found. One of the pulmonary valves was contracted. The aorta was first a little dilated, and then contracted, particularly where it was joined by the ductus arteriosus. There was also a permanent communication between the two ventricles.

Ambrose Parè has recorded the somewhat doubtful case of a certain tailor, who, "by an aneurisma of the *arterious vein* [pulmonary artery], suddenly, whilst he was playing at tennis, fell down dead,—the vessel being broken. His body being opened, Parè found a great quantity of blood poured forth into the capacity of the chest; but the body of the artery was dilated to the largeness of a man's fist, and the inner coat thereof was bony. For which cause Parè, within a while after, shewed it to the great admiration of the beholders, in the Physicians' School, whilst he publiquely dissected a body there."*

It is much to be regretted that the details of the following case have not been more satisfactorily given. I quote the account from a periodical†, having failed to procure a sight of the work in which it was originally published.

In September 1814 Dr. Harlan assisted Dr. Parrish in the examination of the body of Captain M——, a middle-aged person of robust constitution. His disease had never confined him to his house; and the day preceding his death he called on Dr. Parrish, and complained of dyspnœa and pain in the back of his neck. His symptoms were referred to stricture of the œsophagus; and Professor Winstar, who was consulted, advised the use of the probang: the patient deferred this operation, and, during the same night, was attacked with increased difficulty of breathing and exacerbation of all his symptoms. A bleeder being sent for, he lost some blood, with temporary relief; but in a short time afterwards he expired in convulsions.

The autopsy very unexpectedly developed an aneurism of the *pulmonary artery*, the sac of which extended so far back as to press upon the œsophagus, and produce symptoms of stricture in that part. There was considerable effusion in the lungs, and a lump of coagulated blood in the trachea, near the glottis, which most probably occasioned his sudden death. The effusion was occasioned by the rupture of the aneurismal sac, which was adherent to the air-cells of the lungs. Dr. Harlan justly observes that it was fortunate the probang was not employed.*

Mr. Fearn, of Derby, has recorded† the case of a patient who died from hæmoptysis, in whom an aneurismal sac of the size of a nutmeg was found jutting into a tubular extremity on the upper lobe of the left lung. The parietes of the sac were thin, and did not contain any fibrinous layers; a vessel, the size of a small crow-quill, leading from a considerable trunk of the *pulmonary artery*, was distinctly traceable into the sac.

Dr. Peacock has also met with an instance in which a pulmonary branch of the size of a crow-quill was found so dilated at one spot as to be capable of holding a bean. This dilatation was situated within a cavity hollowed out in the parenchyma of the lung containing blood; the lining membrane of the vessel was continuous. Several other cavities, containing pus, existed at the upper part of the lung. Dr. Peacock attributes this lesion to the loss of support which the vessel must have sustained, owing to the removal of the elastic substance of the lung in its vicinity. He also very justly denies the identity of this lesion with aneurism, as it occurs in the systemic arteries.

The preparation numbered 1450[cc] in the collection at Guy's, displays an artery of considerable size, traversing the wall of a phthisical cavity, and presenting two small aneurismal dilatations, one of which has burst into the cavity. The lining of the vomica appears to be unusually thin. Another branch, of equal diameter, occupies another portion of the wall of the excavation; its canal appears to be obliterated.

The lesion which destroyed life in the following case appears to have been closely similar to that form of rupture of the proper tonics of the aorta which usually gives rise to dissecting aneurism:—A Pontonier, twenty-one and a half years of age, who had enjoyed good health excepting slight dyspnœa during three months he had been in the army, after having engaged in carrying wood during the day, was roused in the night by a violent pain at the right of the sternum, with great dyspnœa, which subsided in a few minutes. He afterwards became much relieved. During the afternoon of the following day he asked for drink, became pale and collapsed, and died. On examination, a number of

* In one of his valuable papers on cardiac disease, Dr. G. H. Barlow alludes to a case occurring in his own experience, in which thickening and rigidity of the coats of the *pulmonary arteries*, extending into their smaller ramifications, had produced engorgement of the right ventricle, and its consequences. ["Select Clinical Reports," Guy's Hospital Reports, vol. v. N.S. p. 177.]

* The diseased state of the vena cava observed in the following case seems to have approached as nearly to the condition of aneurism as did the lesions of the pulmonary artery in any of the examples which have next to be cited.

Dr. Edward R. Squibb has recorded the history of a mulatto man, aged 37 years, who, for ten months previously to his death, suffered from a violent but remittent pain in the abdomen, with irritability of stomach, a sensation of obstruction after taking food, and occasional vomiting. Shortly before his death, the pain abated, and the patient felt able to sit up in bed. The exertion of rising up caused a feeling of great weakness and tendency to faint. He was immediately laid down, but expired in a few hours.

On examination, the abdominal viscera were found imbedded in and quite hidden by masses of coagulated blood, the viscera themselves appearing to be quite in a healthy condition. A rupture of the ascending cava was discovered just below the lower concave surface of the liver. The veins at this point had been very much dilated, and its coats much diseased and thinned. A semi-organized mass or clot, which was contained in the dilatation, was connected by its surface to the softened coats of the expanded vessel, and the rupture had occurred at the junction of the edge of this mass with the side of the vessel, and not at the projecting point of the dilatation. The tumor was on the anterior portion of the circumference of the vessel, and was overlapped by the lower edge of the liver, and by a portion of the stomach, which latter circumstance may account for the sensation of obstruction so constantly complained of during life.—*Philadelphia Med. Examiner*, Oct., 1846, p. 583.

† This case has been detailed at length by Dr. Fletcher, in the 25th volume of the Medico-Chirurgical Transactions; but for the sake of brevity, I have here given the account published by Dr. Blakiston in his recent work on "Certain Diseases of the Chest," p. 98.

* Book 1st, De Tumoribus, Cap. XXVIII. London edition, fol. 1649, p. 204.

† Medico-Chirurgical Review, July 1839, from "Medical and Physical Researches, by R. Harlan, M.D. F.R.S. London and Philadelphia, 1835."

* The use of this instrument cannot be too strongly deprecated in doubtful cases of dysphagia. I have met with two instances in which the œsophagus was greatly compressed by aneurisms of the descending aorta; and in each of which—the structures of the œsophagus having become completely removed—there remained a large rounded aperture, which was merely closed by thick layers of coagulum. Had a probang been introduced in either of these cases it must have inevitably caused instnataneous death, by entering the aneurismal sac.

† Lancet, Feb. 6, 1848.

calcareous concretions were found on the outer side of the *pulmonary artery*, close to its origin, and extending towards the right ventricle. The inner coat was separated from the elastic for the space of three inches from the point at which it joins the right ventricle, and torn into shreds, which projected into and narrowed the cavity. The whole lining membrane was coated with a thin layer of fibrine. At its origin from the ventricle, exactly in the situation where the concretions terminated, was a hole of the size of a fourpenny-piece, through which the blood had escaped into the pericardium; the neighbourhood of this opening was of a blood-red colour. The lungs were healthy and bloodless.*

Mr. Carling, of the London Hospital, has most kindly furnished me with the following highly interesting narrative of a case of traumatic diffused aneurism of the right branch of the pulmonary artery. The patient was admitted under the care of Mr. Luke.

"Edmund Taylor, a healthy and muscular young man, aged 23, was brought to the London Hospital about 5 in the morning, Sept. 30, 1838, having been stabbed in the chest with a long clasp-knife a short time previously. The wound was about an inch in extent, and situated at the front and upper part of the right side of the chest, near the junction of the cartilage of the third rib with the sternum. I was informed that instantly after its infliction there was a profuse hæmorrhage, and that the patient coughed up almost immediately a considerable quantity of blood. When admitted he was very faint, and there was a slight oozing of blood from the wound, but this ceased after the edges had been brought together with adhesive plaster. His breathing continued afterwards to be much oppressed, and for two days he coughed up occasionally a small quantity of blood. Pneumonia supervened, but it was checked by general and local blood-letting, and by antimony and mercury exhibited until the mouth became affected. He appeared to be going on tolerably well, when, on the eleventh day after the injury, he was seized with a fit of coughing, and upwards of sixteen ounces of blood suddenly gushed out from the wound. From this period there was a daily hæmorrhage, varying in quantity from one or two ounces to four or five; and he frequently expectorated small clots of blood. The blood which issued from the wound was thin, and sometimes mixed with matter like pus; and air always escaped when the part was dressed. Digitalis, the acetate of lead, and other remedies, were given to check the hæmorrhage, but with no effect. The patient got gradually weaker; and being exhausted by the daily losses of blood, died on the 29th day after the injury.

"The body was examined the day after death. It was much wasted and exsanguineous. The knife had passed in an oblique direction, dividing the cartilage of the third rib, and entering the chest a little to the right side of the anterior mediastinum. The whole of the front part of the middle lobe of the right lung was converted into a false aneurismal sac. There was a large irregular cavity, filled with coagulated blood, part of which was in layers, and deprived in a great degree of the red particles, and part loose and soft, and of a greyish-red colour. There was also a small quantity of coagulated blood. At the bottom of this cavity, towards the heart, was distinctly perceived an opening in a large vessel, capable of admitting a full-sized goose-quill;—the aperture communicated with the right branch of the pulmonary artery just as it divides into several branches for distribution throughout the lung. The pulmonary tissue surrounding this false aneurism was hepatized; and there were firm pleuritic adhesions on that side of the chest. The pericardium contained about three ounces of turbid serum, and there was a thin layer of loose lymph coating a great part of the heart. The left lung and the abdominal viscera were sound. The mammary artery was untouched, but the small intercostal vessel communicating with it was divided.

"It will perhaps excite some surprise that a considerable wound of one of the two main branches of an artery transmitting the whole of the circulating fluid of the body, should not have been attended with such a loss of blood as to have destroyed life in a short period. A wound of the same extent in the arteria innominata or common iliac arteries, less in size than the vessel wounded in this case, would no doubt have proved fatal in a few minutes. The safety of the patient may, however, be ascribed to the inferior power of the right ventricle of the heart as compared with that of the left. In the collapse which resulted from the profuse hæmorrhage consequent upon the wound, we may conclude that coagulation took place,—that the opening in the right branch of the pulmonary artery became closed, and bleeding arrested for a time. The action of the right ventricle, reduced by the hæmorrhage and the treatment afterwards pursued, appears to have been insufficient to disturb the false aneurism which had formed in the substance of the lung; but unfortunately it was subsequently ruptured in a violent fit of coughing. Although the aneurismal sac was of some considerable size it was smaller than might have been expected, considering the magnitude of the vessel wounded,—its near approximation to the heart,—the duration of life after the injury,—and the soft vascular and unresisting nature of the tissue which formed the parietes. But this circumstance may perhaps be explained by the inferior propulsive power of the right ventricle. A false aneurism of the aorta, under similar conditions, if not sooner destructive to life, would, I think, have attained in the same period a very much larger size. At the time that the bleeding recurred, eleven days after the infliction of the wound, the lung had been consolidated by inflammation. It was enabled, therefore, to offer considerable resistance to the effusion of blood and the extension of the aneurism."

It is singular that, while so much has been written upon the subject of injuries to the lungs, so few facts should have been hitherto elicited with regard to the precise manner in which lacerated and punctured wounds of branches of the pulmonary artery heal. It is probable that their closure is usually effected by the gradual infiltration of blood into the surrounding tissue of the lung, as well as by the collapse of that organ consequent upon the presence of blood or air within the pleura, aided by the depressed state of the circulation which results from the hæmorrhage, and from the depleting plan of treatment which is usually adopted. In gun-shot wounds, the closure of the vessels is doubtless greatly aided by the contused condition of all the tissues.

* Foreign Med. Review, Oct. 1842, p. 547: Dr. Helmbrecht, Casper's Wochenschrift.

RUPTURE.

Rupture of the main trunk and larger branches of the artery has been known to occur in several instances. This lesion appears usually to result from a diseased condition of the vessel, and is a far less frequent consequence of severe crushing injuries to the chest, than is rupture of the thoracic aorta. In the following case, however, the laceration appears to have been produced by a violence of this description:—

A healthy, robust youth, ætat. 22, was, in a scuffle, wounded in the neck; he fell down on the spot, and died almost directly. Very little blood flowed from the wound. On dissection, the wound was found to have penetrated two inches in depth, passing between the trachea and left subclavian vein, but neither these or any other important parts were injured. Nearly five pounds of black coagulated blood were found in the left bag of the pleura; the cavity of the pericardium also contained a quart of clotted blood. This membrane exhibited an irregular rupture, nearly two inches in length, at the point corresponding to the root of the left lung; the *pulmonary artery* was found to have given way just beneath the point where the pericardium is reflected upon this vessel: its parietes did not, however, exhibit any traces of morbid change.*

The following case has been recorded by Mr. Wm. Gunn, R.N.:—†

A seaman, ætat. 46, after suffering from pain in the head, extending down the neck and arm to the hand, which was benumbed, while making some considerable exertion, fell back without speaking, and in a state of exhaustion, from which he never recovered. On opening the chest, the left side was found completely full of blood, the lung being collapsed under the pressure. A rupture existed in the *pulmonary artery* about half an inch from its origin, and large enough to admit the point of the little finger. The artery was diseased at the point where it had given way.

Two highly interesting cases of rupture of the trunk of the pulmonary artery have been fully detailed by M. Ollivier.‡

A remarkable instance of this lesion has also been recorded by Mr. J. Adam, jun.§

The patient was a man of debilitated constitution, 52 years of age, who, for some time previous to his death, had been subject to various uneasy feelings, referred to the region of the stomach and head, with general derangement of the digestive functions, and latterly to irregular action of the heart, accompanied sometimes with a sense of constriction across the chest, increased on using any corporeal exertion, or too frequent indulgence in spirituous liquors. Latterly, when the fluttering action of his heart became distressing, he was obliged to have recourse to the recumbent posture for relief. Death occurred suddenly. On opening the thorax, a large quantity of dark grumous blood gushed out from the right pleural cavity, which was nearly filled with the effusion. Two polypi or coagula of blood were found in the right ventricle. The valves of the *pulmonary artery* were in a state of ossification. This vessel was extremely thin, and enlarged to nearly four times its natural size, being not less than seven inches in circumference. It contained an organized polypus, extending from the semilunar valves about four inches along the artery. A rupture had taken place in the "middle" of the artery, and it appeared that there was an opening in the centre of the polypus, through which the blood seemed to be transmitted from the ventricle to the lungs and aorta, and other large vessels issuing from its arch

* Medico-Chirurgical Review, July, 1848. From the Archives Générales.

† Edinburgh Med. and Sur. Journal, January, 1829, and London Med. Gazette, Vol iii. p. 545.

‡ Dictionnaire de Med.: art. Vaisseaux Pulmonaires.

§ In the Calcutta Medical Transactions, Vol. ij. p. 115.

were completely ossified. A similar condition of disease also extended throughout the remaining portion of the aorta. There was no apparent disease of the lungs.

Mr. Fearn, of Derby, has published notes of the case of a man, ætat. 63, who had been troubled with a cough for a long time, but in whom stethoscopic examination revealed nothing beyond the evidences of general bronchitis. The patient was suddenly seized with a tremendous attack of hæmoptysis. The bleeding, however, was restrained for a while by appropriate remedies, but it again returned, and the patient died from exhaustion, nearly four days after the first attack of hæmorrhage. On examination after death, it was found that the right *pulmonary artery* was ruptured, just at its entrance into the lung, and the pulmonary tissue was broken down by the quantity of blood which had been poured into it. There were no tubercles, nor any other adventitious deposit, in either lung.*

A case of sudden death from rupture of the pulmonary artery, is alluded to by M. Devergie. (Médecine Légale, t. i. p. 66). And another, occurring in a man 54 years of age, is noted in the Sixth Annual Report of the Registrar-General (p. 288.)

Ulceration.

The coats of the pulmonary artery occasionally become eroded and perforated in cases where aneurismal tumors of the arch of the aorta greatly encroach upon this vessel. A similar lesion is also produced in certain cases of phthisical disease; and sloughing of branches of the artery has occurred, though by no means frequently, in cases of pulmonary gangrene and pneumonic abscess.

In cases of aneurism of the arch of the aorta, especially where the dilatation principally affects the under and back part of the vessel, an ulcerated or ruptured communication is liable to be formed between the pulmonary and systemic arterial trunks. The lesion is, of course, by no means one of frequent occurrence, but several instances of the kind are upon record, of which the following appear to be most characteristic:—

Dr. Wells relates the case of a merchant, 35 years of age, who had suffered from apparent premonitory symptoms of phthisis, and from a slight attack of hemiplegia, which, however, had passed away when he consulted Dr. W. He then suffered from a noise in his ears, flatulence, and pains in his hands and feet, occasionally attended with slight swelling in the same parts. He did not complain of any unusual feelings in his chest. After considerable fatigue in walking, he was suddenly seized, between 8 and 9 o'clock P.M., while playing with his children, with a sense of great oppression in his chest. He soon after became sick, and vomited a matter streaked with blood. The surface was cold. Soon after midnight he was found labouring under a constant desire to cough, and was continually expectorating mucus tinged with blood. The body was moistened with a cold sweat, and his pulse was extremely feeble: sometimes it was scarcely perceptible. At 5 A.M. his pulse was very feeble and irregular; his breathing difficult; skin pale, cold, and covered with a clammy sweat; and he frequently tossed and writhed his body as if suffering great pain or uneasiness. The faculties of the mind seemed unimpaired. About a quarter of an hour later he became suddenly worse, and in a few minutes expired. Almost immediately before his death, he complained much of heat in his chest.

Autopsy, two days after death.—The blood-vessels of the lungs were very much distended, and there was also a considerable quantity of blood in the air-cells. Each cavity of the chest contained about ten ounces of a fluid highly tinged with blood. The pericardium contained about two ounces of a fluid similarly tinged. The tumor adhered to the *pulmonary artery* just before its division into the right and left branches. Within the circumference of this adhesion there was a narrow hole, by means of which a communication was formed between the two arteries. The cavities of the heart and great vessels were very much distended with blood.*

The following nearly parallel case is related by Dr. J. Reid:—

A gentleman, aged 53 years, who had been for some months complaining of slight bronchitic symptoms, with some precordial uneasiness, was suddenly seized with violent dyspnœa, rapidly followed by insensibility, and in about four minutes from the commencement of the attack he was dead.

On examination 48 hours after death, the heart and all its valves were found healthy. That portion of the arch of the aorta between the upper part of the sinuses of Valsalva and the origin of the arteria innominata was dilated into an aneurism capable of holding the fist. The dilatation chiefly projected to the left side, and there the coats of the artery were not only irregularly thickened, but contained numerous yellow patches. The left side of the aneurism adhered firmly to the trunk of the *pulmonary artery*, and a ragged irregular fissure, nearly an inch and a half in length, formed a communication between them. The

* Provincial Medical Journal, and Med. Times, Nov. 15th, 1845.

* Trans. of a Society for the Improvement of Med. Chir. Knowledge, vol. iii. p. 85.

aneurism contained only a few small clots of fibrine. The lungs contained a very considerable quantity of blood, and some serum.*

Although the particulars of the following case are detailed at considerable length, it is difficult to learn, from the description, whether the arterial lesion was primarily due to aneurism of the vessel or to erosion of its tunics. The rupture of so large an artery could scarcely have occurred under the circumstances.

Mr. ———, ætat. 36, of a consumptive family, suffered more or less from symptoms of phthisis from the autumn of 1841 to November, 1842, when he was attacked with active hæmoptysis. The blood was coagulated, and expectorated without effort. At the end of a month he sank from the exhausting effects of the hæmorrhage.

Examination.—There were adhesions between the pleura of the left side. The pulmonary pleuræ of both sides presented the peculiar spotted appearance which results from strumous deposit. The upper part of the left lung was entirely occupied by a large cavity containing about half a pint of grumous and coagulated blood. The walls of this cavity were composed of the parenchymatous structure of the lung, condensed and solidified by pressure. Upon careful examination, the left branch of the *pulmonary artery*, at the distance of two inches from its bifurcation, was found to open into this cavity by an aperture as large as a crow-quill. The communication of the artery with the abscess was considered to be the result of the coats of the vessel giving way, rather than the effect of the ulceration of the abscess, as the opening from the artery was funnel-shaped, and terminated in the smallest possible aperture. The trunk of the pulmonary artery was so large as at first to be taken for the aorta, for it had a complete curvature to the right as high up as the left clavicle. It was thought that this dilatation of the vessel was perhaps attributable to pressure of the cyst upon one of its main divisions. The patient had suffered from difficulty of breathing from childhood; but it is not mentioned that marked dyspnœa was one of his later symptoms. Indeed, the narrative does not clearly prove that the left division of the artery had been greatly compressed by the abscess.†

In some cases of congenital defect, where the arterial duct remains permanently open, the aorta and pulmonary artery are found perfectly in apposition, and communicating by a wide opening of somewhat irregular form. In distinguishing such cases, it will be necessary to observe whether the aorta is dilated at that spot, and especially to remark whether the valves and trunk of the pulmonary artery are thickened and opaque; as, where a communication has long existed between these vessels, the pulmonary artery usually takes upon itself much of the functions of the aorta, while its structures assume an appearance closely resembling those of the systemic artery.

In addition to the cases mentioned above, four preparations (numbered 14, 15, 87, 102) of the lesion under consideration are stated to be preserved in the collection at St. Bartholomew's Hospital; and one (No. 366) in the Hunterian Museum. A case in which an aneurism opened into the pulmonary artery has also been recorded by MM. Payen and Zeinck.* Another instance has also been contributed by M. Sue.†

Dr. Lee observed that, in a case where a large branch of the pulmonary artery opened into the cavity of a phthisical abscess, the vomica was not lined with that peculiar membranous structure which usually invests the walls of cavities of this class.‡

It is not usual for the arterial branches which pass upon the sides of pulmonary vomicæ to become occluded by coagula. It is probable that in these cases, wherever hæmorrhage occurs from the corrosion of a large vessel which has not previously become aneurismally dilated, the perforation of the artery is due, either to sloughing of the interior of the abscess, a not unfrequent occurrence in phthisis, or to the absence of that solid deposition which usually lies beneath the pyogenic membrane of phthisical cavities.

I was present at the examination of the body of a man, ætat. 58, who died, February 4th, 1845, in the accident ward of Guy's Hospital, from the secondary effects of a fracture of the elbow-joint. He had suffered from bronchitis, with fœtid expectoration, for some time previous to his death. The walls of the right ventricle were rather thin. Nearly the whole of the posterior portion of the right lung was in a boggy condition, the result of a low form of pneumonia; the pulmonary tissue being in this situation generally infiltrated with abominably fœtid sanies. This portion of the lung contained several cavities, one of which opened into a circumscribed cavity in the pleura; the lung surrounding this opening was in a sloughy condition. The trunk of the *pulmonary artery* was rather wide, and decidedly thin. Its surface was free from stain or deposit, but dull. The tissues of the vessel which traversed the diseased lung were generally

* Edinburgh Med. and Surgical Journal, vol. liv. p. 114.

† Mr. W. Crowfoot, Jun., of Beccles; Med. Chir. Transactions, 2d Series, vol. viii.

* Bulletin de la Faculté de Méd., 1819.

† Journ. de Méd. Contin. t. 24, p. 124.

‡ Dublin Medical Journal, vol. xxv. p. 168.

softened, especially in the inflamed parts. Several branches, rather larger than crow-quills, were more or less obstructed by dark adherent clots. In some branches these were evidently old, and nearly absorbed,—the vessels again beginning to be permeable by blood; in others, the clots produced complete obliteration, and were adherent to the entire circumference of the arteries which they occupied. Throughout most of the inflamed parts, however, many branches of the vessel remained perfectly free. A very considerable arterial branch was almost completely obliterated in two places by the pressure of cavities on either side. At each of these spots the vessel scarcely equalled a whipcord in circumference. The contracted portion which was most distant from the heart, was partially obstructed by some whitish fibrine, and the lining membrane of the other strait was vividly and deeply reddened, evidently by an intestritial effusion of blood. This vessel may have been capable of transmitting a very slender current of blood. The lesions which it presented did not appear to be particularly recent, being, in all probability, of several days' standing.

Another branch, of equal size, presented traces of severe inflammatory change, at a spot where it lay in the close vicinity of a sloughy cavity. Here the interior of the vessel bore traces of circumscribed thickening; its lining membrane was blackened, as if from the interstitial effusion of blood which had become partially absorbed. The centre of this discolouration presented a raised whitish defined spot, where the tissues of the vessel had lost their cohesion and appeared to be in a sloughing condition; the diseased tissue was not, however, sufficiently softened and detached to allow of hæmorrhage. This position of the artery was not defended by adherent coagulum: a small band which it gave off appeared to have been obliterated by fibrous deposit; but, although this had become nearly absorbed, the vessel still remained so greatly contracted as to be almost impervious.

It is probable that, had this patient's life been protracted for a day or two, or had the force of his circulation been greater, the separation of the sloughy portion of the artery would have been followed by fatal hæmorrhage.

Ulceration of the Pulmonary Artery (continued).

The lesions presented in the following remarkable case, which occurred within my own observation, were of so singular and so obscure a character, that I am doubtful whether to ascribe them to ulceration, or to softening of the tissues of the valves, the result of acute inflammation superadded to disease of old standing. Whether the destruction of the entire valvular apparatus occurred suddenly, or was the gradual result of chronic changes, must now, I fear, remain unexplained:—

E. C——, aged 19, a servant, of pale aspect and heavy appearance, whose habits had been rather irregular, was admitted to Guy's Hospital, Nov. 9th, 1851, with symptoms of renal anasarca, which had been increasing for the last four months, until five weeks prior to her admission, when the swelling of the body became nearly general. Upon her admission the anasarca extended over the whole of the body and the face. The breathing was 38, not painful, unless when she was on her back; pulse 106. Cough, from which she had suffered for some time, had been more troublesome at night for the last fortnight, but she did not expectorate. The urine was small in quantity, and albuminous. The right side of the chest, below the clavicle, was resonant on percussion; the left, in the same spot, dull, with cooing sibilant rales: posteriorly there was also dulness on both sides. The heart's sounds were regular. She appeared to improve under treatment for a few days, having perspired very freely, and slept better.

On the 13th, the pulse was observed to be irregular. For about three weeks from this date, the œdema, cough, and difficulty of breathing, continued to vary, as the skin and bowels were, or were not, acted upon. During the last two or three days of her life the heart's action was observed to be indistinct, and she became much depressed; the skin was dry; the pulse varying, at different times, from 88 to 100, and very feeble; the countenance swollen, with dark areolæ round the eyes. She complained of nausea, and much pain in the right lumbar region, passing up to the shoulder; but, on the evening of the day before her death, the bowels having acted copiously, she perspired very freely; the breathing still continued laborious, with much wheezing. On the day of her decease (Dec. 3d) the features appeared shrunk, the dark areolæ still surrounding the eyes. She had slept very little; the pulse 100, and feeble; the tongue rough and furred: the skin dry, the breathing laborious, and there was entire suppression of urine. Her debility was so great as to require a free administration of stimulants. She gradually became weaker towards the close of the day, sh inking down in the bed, and, at about five o'clock, P.M., she expired.

Sectio Cadaveris.—Each pleural cavity contained a little serum, and the right lung was partially coated with loose fibrine; the lungs were generally œdematous, and there were traces of pneumonia (grey hepatization) at the upper part of the right. The rima glottidis was œdematous; the heart did not appear enlarged; the walls of the right ventricle were rather thick, but its cavity was of very moderate size; it contained a good deal of dark coagulum. The *pulmonary valves* were almost entirely destroyed; no trace of pouches remained, and merely a few thin tattered slips adhered to the lines where the crescents had been attached. A flattened mass of fibrinous coagulum, which had in great measure lost its colour, adhered to the spot where the left and anterior curtains had joined. Between the origins of the right and left pulmonary branches an ovāl fibrinous mass, as large as a hazel-nut, adhered to the lining membrane of the vessel; it was firm, appeared to have undergone contraction, and contained earthy particles. It evidently interfered with the passage of the right pulmonary branch, which appeared narrow, and corrugated internally, and in a less degree than that of the left. The parts above and below the orifice of the vessel were of a purplish red colour: the lining membrane here was loose and irregular, detached at some spots, and raised by patches of atheromatous deposit in others; but the endocardium of the ventricle generally did not appear diseased. The walls of the left ventricle were rather thick, and its cavity by no means large; the aorta was small and healthy: the liver was enlarged,* soft, and much congested; the spleen was enlarged; the kidneys were white, large, flabby, and mottled.

It is much to be regretted that, in this case, owing to the very ordinary character of the symptoms, attention was not particularly called to the state of the heart during life. It was considered, by some of

* The specimen is preserved in Guy's Museum.

those who witnessed the inspection, that the destruction of the valves was the result of acute inflammatory softening; still, the disease seemed to be extremely localized, and it appeared to me that the reddening of the artery and parts adjacent was due rather to the imbibition of blood by the loosened and diseased surface, than to acute inflammatory change. From the circumstance of a mass of coagulum, partially deprived of its colouring matter, adhering to a spot which once must have given attachment to portions of the curtains, it is evident that the patient had survived the destruction of the valvular apparatus for several days at least,—possibly for a far longer period. The presence of atheromatous deposit, and of an earth-infiltrated clot in the ascending portion of the artery, showed that this vessel had long been the seat of extensive disease: this, together with the obstructed condition of the main branches of the artery, with the additional impediment to the pulmonary circulation which resulted from the pneumonic and pleuritic effusions, must have exposed the valves to constant and long-continued violence, under which, it is probable, they at length became ruptured and destroyed. The ill effects of this extensive lesion were, doubtless, in a great degree mitigated by the strength of the right ventricle.

The following is a brief abstract of cases involving remarkable abnormal conditions of the pulmonary artery, which have been recorded, or which have occurred, more or less, within my own observation (these latter are marked "*seen*") since the publication of the earlier chapters of this memoir:—

A single ventricle giving off a single artery (aorta), which distributes two branches to the lungs.—Male child, well developed, but cold and livid. Attacks of dyspnœa, in one of which it died, seventy-eight hours after birth. The father said to labour under heart disease. A single auricle of large size receiving the cavæ, two pulmonary veins, and the coronary vein. Auriculo-ventricular opening of large size, and provided with an imperfect valve. The ventricle well developed. The aorta, guarded by three similunar valves, gave off, from its posterior part, two pulmonary branches. Coronary arteries not given off at the aortic sinuses, but a single vessel, which appeared to have originated from the concavity of the arch above the pulmonary branches, passed down in contact with the left side of the aorta, and divided at its root into two branches, which were distributed upon the anterior and posterior aspects of the heart (a very similar arrangement observed in the heart of a calf. M. T. Weber, Observatio Anatomico Path. de Cord. Univent, e quo unus tantum truncus arteriosus surgit. Bonnæ 1832). M. Foster, Report of Proceedings of the Pathological Society of London, 1846-7. *Seen.*

An imperfect, but apparently similar, specimen from a cyanosed child, which died at the age of ten weeks, from convulsions of eight or nine days standing. The respiration had been free.—Dr. Crisp, Ibid.

Obliteration of pulmonary orifice.—The ascending portion of this vessel obliterated. The foramen ovale, ventricular septum, and arterial duct, remaining pervious in a cyanosed male child, eleven months and two weeks old. Dr. Peacock, London Med. Gaz., March 31st, 1848.

Also the following interesting, but imperfect, cases:—

Obliterated pulmonary artery.—Foramen ovale and ventricular septum pervious. Heart evidently that of a child at least several weeks old. Author, London Med. Gaz., Dec. 31st, 1847.

A dumb cyanosed female, ætat. 12 years, always delicate; subject to palpitation and dyspnœa on exertion, and to cough and pain in the side: death apparently from bronchitis. Both ventricles slightly hypertrophied, and of nearly equal thickness, opening in ventricular septum. *Ascending pulmonary artery* not discoverable? foramen ovale closed. Lungs of natural size. Unfortunately the arch was not preserved. (Case slightly alluded to in my memoir.)—Dr. Crisp, Trans. Lond. Path. Soc., 1847. *Seen.*

Pulmonary orifice contracted. Lungs partially supplied by enlarged bronchial arteries.—Man, ætat. 19, cyanosed. There appears to have been great contraction of the *pulmonary artery. Bronchial arteries and arteria comes nervi phrenici* unusually large and tortuous; the latter terminated in the lung. The lungs presented tubercles at different parts. *Foramen ovale open. Arterial duct* closed.—Mr. F. Le Gros Clark, Lond. Med. Gaz., June 4th, 1847.

Contracted pulmonary orifice. Patency of auricular and ventricular septa and arterial duct.—A cyanosed child, seventeen months old, which died from cerebral effusion. Entrance of the *pulmonary artery* small, and provided with only two valves. A free aperture in the base of the septum ventriculorum. The *foramen ovale and arterial duct* very nearly closed.—Dr. Peacock, Med. Gaz. Feb. 1848.

Contraction of pulmonary orifice. Auricular and ventricular septa perforated. Arterial duct closed.—In a dissipated man, about 28, Dr. Marcus found the *pulmonary valves* contracted and inefficient. The *tricuspid valve* might be regarded as wanting. Two small openings in the *ventricular septum. Foramen ovale* widely open.—Casper's Wochenschrift, 1849, and Med. Gaz. Dec. 1849.

In the case of a cyanosed boy 13 years of age, who died of pneumonia, the aorta arose entirely from the sinus of the right ventricle. The orifice and trunk of the *pulmonary artery* would scarcely give passage to a goose quill. Left ventricle very small, and had no exit except by an aperture of sufficient size to admit the point of the fore-finger, which opened into the right ventricle beneath the origin of the aorta. A small valvular opening in the *fossa ovalis*. State of the *arterial duct* not ascertained. —Mr. N. Ward for Dr. N. Parker, Pathological Society's Report, April, 1847.

Pulmonary orifice narrow, deficient interventricular septum, closure of arterial duct and foramen ovale.—Child, about two years and five months; cyanosis commencing three months after birth. Extreme contraction of *pulmonary orifice*; opening in the *septum ventriculare*; left cavity small; *ductus arteriosus* and *foramen of Botal* closed.—Dr. Peacock, London Pathological Society's Report, Dec. 7th, 1846.

Early tendency to closure of arterial duct.—Fœtus, born at seven months and a half, died in fifteen minutes after birth, in consequence of hæmorrhage from the umbilical cord. *Arterial duct* nearly closed, capable of admitting only the shank of a large pin.—Author, London Pathological Society's Report, 18th January, 1847.

Congenital closure of the aortic orifice, probably compensated by an open state of the arterial duct.—A female child, born at the full term of pregnancy. It lived two days, and died in convulsions, having been until their occurrence apparently in good health. The heart was natural in size and normal in situation; the cavities of the right auricle and ventricle were much enlarged. The *pulmonary artery* was in the same condition. All the carnæ columnæ on the left side were fused together, so as to obliterate the ventricular cavity, and present in lieu of it a solid mass. The only trace of ventricle was at the upper part of the latter, where there was a little hollow space, of a rounded form, just capable of holding a small pea, and presenting within, slight traces of chordæ tendineæ and mitral valve. The *ostium aortæ* was perfectly closed; and, on opening the ascending portion of the arch, two small folds of the lining membrane indicated the site of the sigmoid valves. The left auricle was natural, the *foramen ovale* large, and the coronary arteries arose at their usual place, and ramified in the usual manner.—E. Canton, Esq., Med. Gaz., March 1849, p. 561. Mr. Canton was so obliging as to afford me an opportunity of carefully examining this valuable specimen of exceedingly rare and curious malformation.

Open ductus arteriosus, associated with contraction of the aorta distal to the left subclavian artery.—Case of this malformation occurring in a male child born at the eighth month, and which died at the age of ten weeks,—Dr. Peacock, London Med. Gaz., Nov. 26th, 1847. *Seen.*

Case illustrating the earliest stage of the malformation usually known as "distribution of the descending aorta from the pulmonary artery."—Author, Report of Pathological Society of London, 15th March, 1847.

Permanence of arterial duct associated with disease of the aortic valve, and narrowing of the aorta opposite the left subclavian artery.—An interesting example of this malformation, occurring in a woman, æt. 34, is recorded by Dr. B. G. Babington.—Ibid. April 19th, 1847. *Seen.*

The *case of closure of the foramen ovale previously to birth*, alluded to in the memoir, is related in detail by Ebenezer Smith, Esq.—Ibid. Dec. 7th, 1846, p. 53. This highly important specimen is preserved in the Museum at Guy's. *Seen.*

Transposition of the pulmonary artery and aorta without transposition of the cavities of the heart. Foramen ovale and ventricular septum open.—This malformation observed in a cyanosed child which died at the age of three years. By M. Colnies.—*Archives Générales*, vol. v. p. 284, for 1834.

Rupture of pulmonary artery.—An interesting case of extensive rupture of this vessel, with laceration of the left auricle, in which the individual survived the injury nearly four hours, is related by Mr. Prescott Hewett.—London Med. Gaz., May 14th, 1847.]

Condition of the Cavities of the Heart in Cases of Malformation associated with error in the Pulmonary Artery.

Right auricle.—In nearly all instances of congenital cardiac defect this cavity is largely developed, being usually much dilated, sometimes enormously so; and generally with more or less thickening of its muscular structure. In some cases the pulmonary veins open into this cavity by uniting and forming two orifices; in others, one or two of those vessels terminate here, while one or more communicate with the superior cava: in one instance the whole of the pulmonary veins were found to enter this cavity. The valved, cribriform, and widely patulent states of the foramen ovale, are well known to anatomists. Occasionally the auricular septum presents two orifices

quite distinct from each other, and separated by muscular structure. In a case (Dr. Blackmore's) where the tricuspid was unusually imperfect, the orifice of the superior cava was defended by a thin valvular apparatus.

Right ventricle.—The tricuspid valve has been found in various ways malformed, in cases of narrowing of the pulmonary artery. Contraction of the right auriculo-ventricular orifice is a not unfrequent concomitant of arctation of the pulmonary ostium.* It appears to be a rule that, when the pulmonary orifice becomes obliterated, subsequently to the completion of the ventricular septum, the right ventricle undergoes so much contraction as almost to disappear entirely: hence, evidently, one form of single heart. In the larger proportion of cases of malformation of the pulmonary artery, the right ventricle is remarkably developed. In none of the severer cases that I have seen has it presented the tenuity of the anterior wall, and that external flattening which characterise this ventricle in its normal condition, but its cavity usually assumes more or less of a globular form: and (in the cases of obliteration and severe obstruction of the pulmonary artery) its walls are probably never thinner than those of the left ventricle, and are generally far thicker. In most cases of this description it is mechanically a matter of absolute necessity that the right ventricle should be of large capacity; but it is occasionally so large as to render it apparent that its condition is rather one of hypertrophy and dislocation than of merely unusual dimensions and muscular power. In cases of this description the ventricle rarely becomes attenuated and aneurismal: still, this extreme result has been observed.

Left cavities.—In all cases of obstruction to the pulmonary artery, the size and strength of the left auricle and ventricle are proportioned to the quantity of blood permeating the lungs. This does not always depend on the manner in which the pulmonary artery is malformed at its orifice: thus, in one case this vessel may be merely contracted, and the lungs receive a very small supply of blood: in another, the ascending pulmonary trunk may be wholly obliterated, and yet the pulmonary circulation may be comparatively freely supplied from aortic branches. In the latter case the left cavities will remain of fair size, perhaps nearly or quite equalling the right; in the former they will be found contracted in dimensions, and of weak muscular power. In fact, without examining either the lungs or the large vessels, the degree of freedom of the pulmonary circulation may generally be at once resolved, in cases of contraction or obliteration of the pulmonary artery with deficient ventricular septum, by observing the size and strength of the left cavities as compared with the right. In some instances, where the aorta has been disturbed directly over the septum, and the left cavities have been well supplied from the lungs, I have found the left ventricle fully as strong and as large as the right, having evidently taken an active part in propelling the blood into the aorta. Still as, in a very large majority of these cases, the supply of blood to the lungs is unnaturally small, and circulates with difficulty, it is usual to find the left auricle and ventricle remarkably small, and with rather thin parietes. Indeed, in many cases, the left ventricle appears to perform no other function than that of propelling a small supply of oxygenised blood through the open septum into the right ventricle, upon which nearly the whole onus of carrying on both the pulmonary and the systemic circulation devolves. Where the orifice of the aorta is obliterated, the ventricular septum being complete, the cavity of the left ventricle becomes nearly obliterated. I believe that, in several forms of congenital disease of the heart, attended with great pulmonary obstruction, death is hastened by the thinned and weakened condition of the left cavities, which have become so atrophied and powerless as to be incapable of contracting firmly upon any unusual quantity of blood that may be accidentally thrown into them.

A review of the examples of congenital heart disease, and of cardiac lesions occurring at late periods of life, which have been collected in the preceding pages, affords us an extraordinary insight into the many and beautiful resources which nature possesses in adapting the still uninjured portions of a diseased or deficient heart to the necessities of the circulation. There are no truths more important in physiology or pathology, none which convey to the physical student a more direct and striking evidence of the greatness of the Creator's plan in the construction of man's body, than the facts that life and circulation may continue under either of the following conditions:—When the right auriculo-ventricular orifice, the ascending pulmonary artery, the mitral opening, the aorta ostium, or the descending thoracic aorta, is perfectly obliterated; where the foramen ovale or arterial duct is closed previously to birth; or where, in adult life, the pulmonary valves have become totally destroyed. It is a consciousness of the gift of recognising such beautiful truths as these, and of obtaining, through them, an insight, however partial, into the right means by which nature should be assisted in her efforts to resist disease, that frees the labours of the pathologist from every embarrassment of fear and disgust, and leads him forward in his researches by the power of a resistless and inexhaustible charm.

* I believe that great narrowing of the tricuspid orifice rarely, if ever, occurs independently of contraction of other outlets of the heart. I have met with two remarkable instances of this lesion, in each of which the mitral orifice was also extremely narrow. A complication of this kind, of course, gives uncertainty to the diagnosis, and diminishes the prospect of survival.

Diagnosis and Symptoms.

When I find an accomplished physician and careful auscultator of the present day omitting from his treatise all consideration of the signs which characterise the diseases of the pulmonary artery, as being of little practical value, owing to the extreme rarity of those lesions;* and when, on looking over the materials which have been brought together in this memoir, I find that, in consequence, probably, of a similar opinion in the profession generally, scarcely more than two or three practical observers have considered the diagnosis of this class of diseases worthy of their attention,—I cannot but feel some degree of apprehension in bringing forward the following imperfect details in illustration of a subject regarding which so little positive information has hitherto been acquired. It is certain, however, that the diseases which affect the pulmonary artery are neither rare nor unimportant, and that, when present, they are generally characterized by certain distinctive rational symptoms and physical signs. The present chapter will merely embody a few scattered facts which I have been able to collect in elucidation of the subject. I trust that their evident imperfection will lead to more careful observation, and to the establishment of more definite results.

It would, of course, be almost impossible, and quite useless, to attempt to lay down any precise rules for diagnosticating every one of the various malformations to which the cardiac apparatus is liable; but a few general rules, founded principally upon the relative frequency of the principal forms of congenital lesion, and upon a knowledge of the periods to which life may be extended in each, may lead to the establishment of a series of diagnostic principles, which, although not by any means absolutely certain in their application, will frequently assist us in arriving at correct opinions.

It will probably never lie within the power of any physician to diagnosticate the particular set of lesions which are present in the vascular system of a cyanosed infant* within a few days of its birth. Its heart may be the seat of any conceivable kind of malformation: still, if the child has become cyanosed almost immediately upon the establishment of respiration, it is most probable that it suffers from closure of the pulmonary orifice, with imperfection of the ventricular septum.

Where the symptoms of morbus cæruleus are not developed until some days or weeks subsequently to birth, it is probable that the orifice of the pulmonary artery is narrow, the ventricular septum open, and the foramen ovale and arterial duct either closed or distinctly contracted; or the latter of these passages may have become narrowed or closed, while the former is widely pervious.

If the infant be upwards of a year old, it is in the least degree improbable that it suffers from the malformation usually known as "distribution of the descending aorta from the pulmonary artery."

If the child has survived its fourth year, transposition of the great arteries is scarcely to be suspected.†

When cyanosis is present at about the age of three or four years, it is probably due, either to great contraction, or closure of the pulmonary orifice, with ventricular communication. Should a single systolic bruit be heard superficially in the region of the pulmonary orifice, the case will almost certainly prove to be one of the former kind.

At the age of one month, or at any subsequent period, it cannot be judged with any probability that the heart literally consists of only two cavities.

In early infancy there are no means of diagnosticating between imperforation of the pulmonary artery and transposition of the two main arteries, except that the former irregularity is of far more frequent occurrence than the latter.

If the patient be above the age of seventeen years, imperforation of the pulmonary orifice can scarcely be suspected.‡

A person above the age of sixteen years, or a young adult, suffering from cyanosis of long standing, a bruit being audible in

* Practical Observations on Certain Diseases of the Chest, and on the Principles of Auscultation, by Peyton Blakiston, M.D., F.R.S., p. 223, 1848.

* In these remarks, I have not used the term "cyanosis" as merely characterising the symptom of lividity of the surface, but as comprising that train of phenomena which mark the existence of congenital malformations of the heart.

† In the three recorded cases of this malformation in which the longest survival occurred, the children died respectively at the ages of ten months, two years and nine months, and three years.

‡ Two individuals have presented this malformation at the age of ten years; one at twelve; and one at sixteen.

the region of the pulmonary artery, most probably has contraction of the orifice of that vessel, with perforation of the septum ventriculare.

If the individual has passed the period of early youth before becoming the subject of cyanosis; or if that symptom, formerly scarcely perceptible, has become considerably more apparent of late, it is, at first sight, probable that the disease is congenital narrowing of the pulmonary artery, the impediment having latterly been increased by thickening and further contraction of the parts, consequent upon superadded disease. In this case the ventricles probably do not communicate.

If the patient has passed the age of thirty years, the existence of congenital deficiency of the ventricular septum is highly improbable. Those who suffer from a congenital cause of obstruction which has originally been sufficient to arrest the development of the septum, very rarely, if ever, attain so advanced an age.*

If, in any given case of cyanosis, the symptoms be ascribed solely to a patent condition of the foramen ovale, the incorrectness of that diagnosis will certainly admit of proof upon examination of the body.†

The larger number of the above diagnostic rules will, doubtless, occasionally fail; but I believe that they afford as near an approximation to the truth as we are at present capable of obtaining when attempting to generalise upon a disease so multiform in its types, and, at the same time, subject to so few variations in its rational symptoms, as congenital malformation of the heart.

Having given very full details of the more prominent symptoms in most of the cases which I have cited, it appears quite unnecessary to enter into a separate description of the various characteristics of morbus cæruleus. It is almost impossible that any practitioner should fail to distinguish a case of this description. The diagnosis between ordinary cardiac disease and congenital malformation is, however, a matter of some importance, and will be discussed hereafter.

The amount of positive information which has been adduced relative to the auscultatory signs of the various malformations of the heart, is exceedingly small.

Considerable attention has of late years been paid to the physical signs which characterise obstructive disease of the pulmonary orifice. Dr. Ormerod, who has published a highly interesting and suggestive paper on the subject,* evidently regards as the most distinctive sign the presence of a murmur heard loud at the base of the heart, and which may or may not be also traceable along the aorta, but most clearly audible in a line from the third left intercostal space at the edge of the sternum, towards the middle of the left clavicle, for the distance of from two to two and a half inches. This is unquestionably the situation at which there is reason to expect that a bruit arising from obstructive disease of the pulmonary orifice should be most intense; but where, as is usually the case in these instances, the position of the heart is more or less abnormal, owing to the enlarged condition of the right ventricle, and where the cardiac action is much excited, the bruit is so liable to have its point of greatest intensity altered, and to be diffused over a very large portion of the thoracic cavity, that I apprehend it will, in a considerable proportion of cases, be found extremely difficult to assign to it by any means so definite an anatomical boundary.†

* In the four cases of this defect (the lesion being unquestionably congenital) where life was most prolonged, three of the individuals died respectively at the ages of twenty-two, twenty-four, and twenty-five years; and in an example observed by Dr. Fletcher, and already cited, an opening as large as a goose-quill was found in the ventricular septum of a man sixty-three years of age. In one rather questionable case the person is stated to have died at the age of twenty-seven. Richerand relates a case of imperfect septum ventriculare in which the patient attained the age of forty years; and Louis cites the instance of a general officer in whose heart a similar malformation was discovered. However, as Dr. Todd very pertinently remarks, there does not appear to have been any contraction of the pulmonary orifice in either of these two cases. I greatly doubt that an individual in whom a large and free congenital communication exists between the ventricles can reach the period of middle life.

† In the analysis of 180 cases of cyanosis, M. Aberle, of Vienna, found that two-thirds of the individuals were males.

* "On a Systolic Murmur in the Pulmonary Artery, and its Application to the Diagnosis of Functional and Organic Murmurs." Edinburgh Med. and Surg. Journal, 1846, p. 95.

† In a discussion which arose subsequently to the reading of Dr. Ormerod's paper at the Medico-Chirurgical Society, Dr. C. J. B. Williams objected that the rule laid down by Dr. Ormerod did not sufficiently distinguish aortic from pulmonary arterial murmurs. "When an aortic murmur was weak, it might be heard at the mid-sternum, yet not be loud enough to reach the arteries of the neck; but when made louder by anything exciting the actions of the heart, then it was heard in the neck, and showed its aortic origin. This is commonly the case in anæmia, in which the loose, ill-filled state of the arteries, makes them bad conductors of sound; and this, he suspected, would apply to most of the cases" (of "functional disease") "described by Dr. Ormerod as those of pulmonary murmurs." (LONDON MEDICAL GAZETTE, June 6th, 1845). Dr. Ormerod's observation has led him to conclude that a murmur may be heard in the pulmonary artery in acute rheumatism, as also in many cases of anæmia and its kindred maladies, which murmur he believes there are good reasons for concluding not to arise from organic disease, as well on account of the variable nature of the murmur itself, as, most conclusively, on account of the rarity of disease of the pulmonary valves.

In chronic cases, and where the bruit is single, the *superficial character* of an abnormal sound heard over the base of the right ventricle, and in the course of the pulmonary artery, becomes very distinctive of its origin in that vessel.* The *tone* of the bruit, of course, varies greatly in intensity, according to the nature of the lesion which produces it. An abnormal sound originating in this artery is usually systolic, being occasionally so much prolonged as entirely to mask the second sound.†

* In cases of phthisis, Dr. P. M. Latham has frequently met with a systolic murmur occupying the space between a line drawn from the left side of the sternum along the upper edge of the second costal cartilage, and continued an inch along the second rib, and another line drawn from the sternum along the lower edge of the third costal cartilage, and continued an inch along the third rib. This sound is a gentle bellows murmur, which cannot be perceived either in the præcordial region, or in the course of the aorta, or in the carotids. Dr. Latham considers that, where, from direct examination of the lungs, a suspicion only of tubercular disease can be entertained, the murmur in the space indicated must always contribute to confirm it. He supposes the pulmonary artery, in its first divisions, to be the seat of the murmur, but doubts whether it becomes such in consequence of its own disease, or by reason of pressure or impediment reaching it from diseased lung. I am not aware that the above observation has been explained by dissection; nor am I acquainted with the existence of any perceptible organic condition in the heart or pulmonary artery in cases of phthisis which could account for the occurrence of this murmur. Whether it depends upon pulmonary or pleural lesions, a want of relation between the size of the ventricle and the capacity of the artery, or upon other causes, has yet to be explained. Loose adhesion of the lung to the pericardium, emphysema of the pulmonary edges, which is common in phthisis, and especially where portions of the edges are occupied by tubercular deposit; recent pleuritic granular exudation, and the presence of an abscess in the anterior margin of either lung, but particularly of the left, may, in phthisis, produce abnormal sounds synchronous with the movements of the heart, almost undistinguishable from those connected with organic disease of that organ: but all these sounds are, as far as I have observed, more or less modified by respiration. I have never discovered any morbid condition of the pulmonary artery, either in these or in any other cases of phthisis, which appeared capable of producing a bruit.

† This was noticed in a patient of Dr. Peacock's, whose case I have already cited in the chapter on Acute Inflammation of the Pulmonary Artery, reserving the physical signs for insertion in this place; as, although the cardiac lesions were somewhat complicated, the auscultatory phenomena appeared to have agreed pretty closely with the rules laid down by Dr. Ormerod. "The chest yielded a clear sound on percussion, except in the præcordial region, where the resonance was impaired over a larger space than natural. The liver could be felt extending a little below the edges of the ribs; the sternum was arched and prominent, more especially towards its base. A loud murmur was heard in the præcordia, accompanying the impulse of the heart. It was of a soft, blowing character, and was most intense at the cartilage of the third left rib near the sternum, or at a point half an inch above the nipple, and between that body and the sternum. It was here prolonged throughout the whole period, so as to render the second sound inaudible. From this point it continued to be heard very distinctly, though decreasing in intensity, along the upper part of the sternum, in the subclavian and carotid arteries" (this circumstance may have been due to the existence of a very free communication at the base of the ventricles), "and on the left of the spine, in the interscapular and dorsal regions. It was also heard, less distinctly, in a line from between the nipple and sternum, towards the middle of the left clavicle. Below the level of the nipple the murmur became shorter and more feeble; and, at the point of pulsation of the apex, towards the epigastrium, and on the right side of the lower half of the sternum, it was followed by a very clear second sound."

A diastolic bruit has rarely been observed as marking regurgitant disease of the pulmonary orifice. In one of Dr. Ormerod's cases which I have cited, a loud, harsh systolic murmur became diastolic previously to the patient's death—probably in consequence of a gradual failure in the power of the right ventricle. In Dr. J. Bell's case, already detailed, the sounds of regurgitation were quite distinct, and apparently attended with an abnormal first sound. Here, however, there was reason to believe that the blood passed downward *from* the narrow and valveless pulmonary artery to the right of the ventricle. In the case of aneurism of the pulmonary artery, with contraction of one of its valves, related by Drs. Fletcher and Blakiston, the sound appears to have been double. "Immediately on the left of the sternum, between the second and third ribs, there was dulness on percussion over a space measuring nearly four square inches. Over this spot a very liquid pulsation was felt, accompanied by a purring thrill, so rough and so close to the surface, that it seemed almost to grate on the fingers. At the same place a hollow rasp sound was heard with the systole of the heart, and also, for a moment, at the commencement of the diastole, as if the blood was receding."*

* The signs observed in the above case agree well with those given by M. Aran (partly, apparently, on Hope's authority, and partly theoretically) as the characteristics of *Aneurism of the Pulmonary Artery*.

"*General symptoms.*—Those of hypertrophy, with dilatation of the heart, which generally accompanies this morbid state. Dilatation of the pulmonary artery can only be confused with dilatation, or with sacciform aneurism of the aorta; but never in either of these" (two latter) "cases do we find pulsation between the second and third ribs. Even if an aneurismal tumor of the arch of the aorta should have destroyed the cartilages of the second and third ribs, it would not be possible to make a mistake in diagnosis, because the aneurisms of the aorta are very large, and the murmur which they produce is grave and distant, instead of being acute and superficial; and, besides, the tremor would be perceived above the clavicles, which is never the case in dilatation of the pulmonary artery."—Practical Manual of the Diseases of the Heart, translated by Harris.

Attempts are frequently made to diagnosticate the presence of those deficiencies in the ventricular and auricular septa which so frequently attend congenital narrowing of the pulmonary orifice. Where, however, as usually happens, these communications are wide, and not associated with a diseased and roughened condition of the adjacent endocardial surfaces, I am not acquainted with any evidence in favour of the belief that their presence occasions the development of abnormal sounds: indeed, it should hardly be expected that, where, owing to the presence of some permanent cause of obstruction, the course of the circulation has been diverted through one of these apertures, any abnormal sound should attend the transit of the fluid, so long as the communications continue to be direct, smooth, and perfectly free. In some cases where deficiency in the ventricular septum exists, independently of any narrowing of the arterial orifices, the abnormal aperture is liable to become greatly contracted, and the surrounding endocardium is then usually found to present more or less irregularity of surface, wrinkling of the membrane, warty vegetations, or raised fibrinous deposits. Here it becomes doubtful whether the systolic bruit which is generally heard at the base of the heart is due to the forcing of a slender stream through the narrow communication, or to the passage of the currents, on their way to the arteries, over its irregular margins.

The rational symptoms which distinguish impediment to the entrance of this vessel are usually more characteristic than the descriptions of Tiedemann,* and of other authorities, would lead us to believe.

It has been shown that, although, in congenital cases, blueness of the surface usually attends obstructive disease of the pulmonary artery, this is not present unless the impediment is very considerable; and the case observed by Dr. Lloyd, although it can only weigh as a rare and exceptional instance, proves that the utmost amount of impediment may be established in the pulmonary artery of an adult without producing livor of the surface, providing the lungs act freely, and the volume of the circulating fluid is small.

An habitually small and rapid, but *regular*, condition of the pulse, usually associated with a more or less excessive action of the heart, is so generally present in cases of congenital cardiac defect, that it cannot be regarded as pathognomonic of contraction of the pulmonary orifice; still, it becomes an important aid to diagnosis in cases where narrowing of this vessel occurs in the adult.*

Diminution in the volume of the pulse, attending aggravation of dyspnœa and palpitation, is, of course, always to be regarded in this, as in every other form of heart disease, as an unfavourable sign, as evidencing superadded pulmonary or cardiac obstruction.

A very characteristic and highly interesting leading pathognomonic sign of obstruction to the pulmonary artery appears to have almost altogether escaped the attention of writers on heart-disease. I allude to the distinctive circumstance that, in a very large proportion of cases, individuals suffering from great narrowing of the pulmonary artery seek the recumbent or horizontal position, either habitually or during paroxysms; while those who are the subjects of any other form of pulmonary obstruction, or of impediment to the circulation through the left heart, breathe most freely when the shoulders are raised, and the body is placed almost vertically. This circumstance has been noticed incidentally by the observers of several of the foregoing cases, but it never appears to have received its true weight, or its pathological explanation.† It is evident that,

* For Prof. Tiedemann's account of the symptoms of narrowing of the pulmonary orifice see Edinburgh Med. and Surg. Journal for April 1846, p. 442.

* Dr. Williams observed that the pulse is not affected in cases of obstruction to the pulmonary orifice (as quoted by Copland, Dict. of Pract. Medicine, art. Heart, § 78). It is true that the pulse seldom intermits or is irregular in these cases, except occasionally, where there is regurgitation, or where the power of the heart begins to fail; but the smallness and rapidity of the pulse are too characteristic, in these cases, to allow it to be believed that the above opinion is still entertained by that eminent observer.

† This is abundantly shown in the instances which I have cited. Thus, in Dr. Lexis's case, the girl experienced paroxysms of strong suffocation, which she endeavoured to relieve by bending the body forward.

Dr. Pulteney's patient "could escape the fit by instantly lying down on the carpet on his left side, and remaining immoveably fixed in that posture for about ten minutes."

In Dr. Ramsbotham's case, where the pulmonary artery and aorta arose from the right ventricle, with patency of the ventricular septum, the child "lay on her back, or on the left side, with her legs drawn up to the body, and the back bent to a semicircle."

In a patient of Dr. Peacock's "the respiration was peculiarly rapid and panting, and he was compelled to lie on the back, partly inclined to the right side, and with his head low."

In Dr. Birkett's case it is noticed that latterly "the respiration, though distressed throughout, appeared to become so far relieved as to admit of her lying in the horizontal posture."

In the case by Dr. Bond, of Cambridge (cited by Dr. Ormerod) the man "was able to retain the horizontal position to the last."

Dr. Lloyd's patient "lay on her left side;" and, "when she got out of bed, she was obliged to lie on the floor to avoid fainting. She had done this for some time during her illness."

In Dr. Elliotson's case there was dyspnœa, with palpitation, but the man "could lie down."

Mr. Adam noticed that, in a case of dilated

whatever distress of breathing occurs in uncomplicated cases of this description results from impeded and insufficient access of blood to the lungs; and that hence the recumbent position affords the aid of gravitation to the contractile efforts of the heart, and, at the same time, renders the supply of arterial blood to the brain more free than it could otherwise be. This symptom cannot, however, be assigned merely to cases of congenital narrowing of the pulmonary artery: it is characteristic of all circumstances which impede the free access of blood to the pulmonary vessels. Thus it occurs in certain instances of tumors in the anterior mediastinum, and where extensive pericardial deposits and effusions compress the anterior surface of the heart. I have also observed it in two cases of aneurism of the root of the aorta, pressing forward to the sternum, and interfering with the pulmonary artery.*

It is a matter of great importance to be prepared to distinguish between those cases, so well described by Dr. Barlow, in which narrowness of the chest gives rise to cardiac obstruction, and those in which congenital disease of the heart has occasioned ill-development of the other thoracic organs. Thus, nothing could be less rational than to recommend athletic sports for a narrow-chested child, the subject of congenital heart-disease; while almost equally ill results would accrue from denying proper exercise in the case of a person under fourteen or fifteen years of age, whose only discoverable physical fault was an ill-formed chest, attended with shortness of breath, and a liability to occasional palpitations.

Little difficulty attends this diagnosis during childhood; but, where the cases present themselves for the first time towards the age of puberty, rather more discrimination will be required. Here the following facts may be taken into consideration:—In cases of congenital disease of the heart in which the symptoms first become severe at puberty, the chest is not usually extremely ill-formed: the orifice of the pulmonary artery is mainly in fault, and a systolic bruit in this situation is usually present: whereas, in those cases of Dr. Barlow's where a bruit occurs (which by no means always happens), the abnormal sound may usually be traced to the mitral valve. At this period of life, however, the diagnosis is rather a matter of interest than of utility, as the principal mischief in either case is irreparable, and a knowledge of the precise nature of the cause of obstruction cannot materially influence treatment. The remark has been introduced here merely with a wish to enforce very strongly the necessity of distinguishing between the two sets of cases during childhood.

Permanence of the arterial duct is so generally associated with some other grave organic and obstructive lesion, either in the aorta or in the pulmonary artery, that it appears doubtful whether any perfectly distinctive signs of its existence can be obtained. Dr. Williams, however, states, as some ground of diagnosis in cases of this defect where there is no other very serious lesion to obscure it, that the murmur accompanying the first sound of the heart is *prolonged into* the second, so that there is no cessation of this murmur before the second has already commenced. From this sign—taken, of course, in connection with other symptoms—he has been able, in at least two cases, to diagnosticate this lesion; and post-mortem examination proved the accuracy of this diagnosis.*

Prognosis in Cases of Cyanosis, &c. involving a Faulty Condition of the Pulmonary Artery.

Some leading data for prognosis in cases of congenital cardiac defect may be drawn from the earlier remarks in the chapter on Diagnosis. Thus it will be found that,

pulmonary artery, obstructed by an "organised polypus," the patient was latterly obliged to have recourse to the recumbent position for relief "when the fluttering action of the heart became oppressive."

I believe that the only cases on record, of narrowing of the pulmonary artery, in which it is mentioned that the patients were compelled to remain in the erect position, are the two by Dr. J. Crampton and Mr. Marshall. In the latter the exception is accounted for by the fact that latterly the patient suffered from large ascitic effusion. I have not been able to meet with the original report of the former case, and can only find that, in addition to the cardiac defects, the whole surface of the body was extensively anasarcous, and that the measurement of the right side of the thorax exceeded that of the left by half an inch. It is probable that here also some grave cause of pulmonary embarrassment exterior to the heart was in operation.

* In some cases of aneurism of the root and arch of the aorta there is considerable obstruction to the circulation (independently of the interference of the tumor with surrounding parts); the patient being consequently liable to suffocative attacks. In others the transit of blood is free, and the breathing little embarrassed, almost up to the time of the patient's death. In these two cases the aneurisms were evidently of the latter kind; while the second of Dr. Elliotson's cases (quoted in the chapter on Obstruction to the Pulmonary Artery consequent upon External Compression) was of the former description. There the pulmonary artery was extremely narrowed by a fibro-cartilaginous deposit, while there was a large aneurism of the aorta. In this instance the obstruction to the systemic circulation counterbalanced the defective pulmonary supply; and, consequently, the individual could not lie down.

* London Medical Gazette, May 7th, 1847, p. 824.

in cases of "*distribution of the descending aorta from the pulmonary artery*," death occurs within the first year. In *transposition of the great arteries* (unattended with general transposition of the viscera), life has not been known to be protracted beyond the fourth year, while death usually occurs much earlier. The highest age which is known to have been attained in *imperforation of the ascending pulmonary artery* is 16 years; but here, also, life is rarely so protracted. I can only discover five cases of this lesion in which the individuals survived the age of a year and a half.*

The subjoined analysis of 159 cases of congenital organic defect of the heart, or cyanosis (from Dr. Aberle, of Vienna), affords a fair ratio of the probability of survival under these circumstances:—

Death, in the first 24 hours	. .	4 cases.
„ in the first fortnight	. .	16 „
„ before the end of 1st month		7 „
„ second to third month	.	6 „
„ third to sixth	. . . .	8 „
„ sixth months to one year		12 „
„ one year to two years	.	7 „
„ two years to three	. . .	9 „
„ three years to six	. . .	11 „
„ six to eight	. . . .	11 „
„ eight to eleven	. . .	13 „
„ thirteen to sixteen	. . .	12 „
„ sixteen to twenty	. . .	8 „
„ twenty to twenty-five	.	10 „
„ twenty-five to thirty	. .	6 „
„ thirty to thirty-five	. .	5 „
„ thirty-five to forty-five	.	5 „
„ forty-five to sixty	. . .	4 „
„ at eighty	. . . . .	1 „
		159

We find here, that, previous to the expiration of the first year of existence, 57, or 35 per cent., of the cases proved fatal. Upwards of two-thirds (108) of the individuals died before the age of eleven years; between that age and twenty-five years 30 more had fallen from the list; and, of the remaining 21, five only passed the age of 45 years.

Every epoch of the brief life of an individual suffering from a grave congenital defect is fraught with danger to the patient, and with the deepest anxiety to his physician. The commencement of respiration is attended with imminent danger of fatal embarrassment to the circulation, the heart now becoming suddenly burthened with functions which it is destined never to perform with freedom; and, at the period when the closure of the arterial duct and foramen ovale should be effected, the struggle for existence becomes still more protracted, and equally precarious. The third period of danger is proved to attend the first dentition. Hooping-cough, scarlatina, and measles, fall with double weight and fatality upon these unfortunate little patients. Another season of trial is ushered in by the approach of the second period of dentition; while, in any year of childhood, death may occur from hydrocephalus, bronchitis, pneumonia, pulmonary tubercular disease, diarrhœa, or gradual aggravation of those agonising paroxysms which characterise the disease. Thus it will be perceived to be indeed a matter of almost hopeless difficulty for a cyanosed child to struggle on to the age of puberty; but that life should be extended beyond the period at which that change is effected, is an anticipation which can scarcely be entertained, even by the most sanguine.

The unfortunate little subject of these defects is, in almost every instance, unable to take part in those active and clamorous sports which the young child in vigorous health instinctively pursues, for the development of his respiratory organs and muscular system. Hence, as the period of maturity approaches, when the other organs of his body have acquired nearly their due bulk and proportion, and when, as the result of habitual inactivity, a preponderance of fluids almost invariably exists, the narrow and ill-formed chest and deficient pulmonary apparatus present causes of nearly inevitably fatal obstruction to the circulation through the imperfect and relatively feeble heart.*

The observations of Farre and others lead to the belief that corresponding cardiac defects are liable to occur in several children of the same parents. Several children of one family may be born with various cardiac defects; or several children may be born still, and one with cardiac malformation. Our evidence upon this subject, however, is extremely scanty.

Occasionally, malformation of the heart may have caused the death of the fœtus in utero. Dr. Hunter argued that many miscarriages at the earlier or later months may depend upon some undiscovered malformation, perhaps of this kind.

In those females suffering from severe congenital disease of the heart who survive the age of puberty, menstruation usually occurs late. Whether women thus circumstanced have ever become the mothers of healthy children subsequently to the establishment of that function, I have not been able to ascertain: in fact, the marriage of such persons must be an extremely rare occurrence. Great cardiac obstruction appears almost equally to determine the late development of puberty in the male. In all the slighter forms of congenital cardiac defect, occurring in patients of either sex, it is probable that the offspring does not suffer in any evident degree. In a case of great contraction of the pulmonary orifice which I have quoted from M. Cherrier, it is mentioned that the man married at the age of 28 years, and had two children, who died of scrofulous affections at the age of three years. This, however, may have been merely an accidental coincidence. It is to be regretted that our information upon this subject also is so extremely scanty. A few additional facts would be of very high physiological interest.

Persons who are the subjects of congenital obstructive disease of the heart are always liable to suffer from temporary or organic cerebral lesions, consequent either upon deficient supply of blood to the brain, the circulation of an ill-depurated fluid through the vessels of that organ, or mechanical obstacle to the emptying of its veins. Most of the leading symptoms in these cases are cerebral—drowsiness, torpor, giddiness, vertigo, headache, loss of consciousness, syncope, &c. After the first months of infancy a large proportion of deaths in cyanosis result from various kinds of cerebral disease; such as watery softening, hydrocephalus, abscess of the brain, congestion, or other grave lesions of the nervous centres, producing convulsions and convulsive attacks, hemiplegia, and temporary or permanent paraplegia.

The rule, in cases of cyanosis, is that the intellect is deficient, mainly from a faulty condition of the brain, and partly also, no doubt, from the want of mental training which is usually, and almost necessarily, attendant upon prolonged ill health in childhood and early youth. The recorded instances of ordinary or precocious development of intellect in these subjects are extremely rare, and might, probably, be found to have been placed on record chiefly upon the authority of partial friends. The development of the intellectual faculties, however, is not, as some writers have argued, proportioned in these cases to the degree in which the heart is malformed, but to the degree in which the blood is arterialised, and to the quantity of that fluid which is supplied to the brain.

These patients rarely perish from the gradual symptoms which usually characterise obstructive disease of the heart. They are liable to die suddenly from failure of the heart's action, or to sink rapidly when bronchitis, or pneumonia, or any other cause tending to embarrass the lungs, comes into operation; but they are not affected with dropsy (except in those cases where cyanosis occurs comparatively late in life, as the result of lesions superadded to congenital malformation), and œdema very rarely presents itself as one of the earliest (noticed) symptoms, as is often the case in ordinary heart-disease. In congenital cyanosis, the venous system seems to adapt itself to the overloading, from delay, which it constantly experiences; the right auricle, cavæ, and entire venous system, appear to have been of unusual capacity from the first, and the veins of the liver especially are capable of containing a vast quantity of delayed blood. Hence there is a less tendency to the occurrence of dropsical exudations here than in cases where obstruction to the circulation commences at a later period of life. Although dropsical effusions rarely proceed to extremity in cases of this kind, their development, where they do occur, must be regarded as an evidence of the most excessive and serious degree of obstruction. Œdema of the legs and feet is apt to appear in the advanced stages of morbus cæruleus, and is liable to be attended with gangrene,—less on account of extensive distension, than owing to sluggishness of the circulation in parts remote from the heart.

The lungs, mesenteric glands, and other internal organs of persons suffering from cyanosis, are extremely liable to become the seats of tubercular deposit. This is accounted for by the supposition of an authority, whose name has escaped me, that it depends upon the deficient and imperfect supply of arterial blood to the parts.

It is singular that so able an observer as Rokitansky should have failed to notice this very ancient and well-supported fact. He observes, "All forms of cyanosis, or rather all the diseases of the heart, great vessels, and lungs, adapted to produce cyanosis in a greater or less degree, cannot co-

* In 26 cases of obliteration of the orifice or ascending portion of the pulmonary artery, collected in this memoir, death occurred at the following ages:—Still-born (1); 7 days (1); 13 days (1); 23 days (1); uncertain, but under 1 month (1); 4 weeks (1); 6 weeks (1); 7 weeks (1); 5 months (1); 5½ months (1); under 6 months (1); six months (1); 8 months (1); 10½ months (1); 11¼ months (1); 11 months (1); 8 years (1); between 9 and 10 years (1); 10 years (1); unknown (1); 16 years (1).

* Persons with very slight and apparently uninjurious congenital defects in the heart and its appendages, rarely survive the period of greatest muscular strength (25 years). Under such circumstances, the heart is peculiarly susceptible of acquired disease, and the lungs are extremely liable to sudden and extensive inflammations. It is extremely rarely that we meet with any congenital defect of the heart (except valvular foramen ovale and corded sigmoid valves) in a person advanced in life. For particulars regarding the influence of deficient thoracic development in producing dangerous impediment to the circulation through the right side of the heart, in cases where that organ is not malformed, see Dr. Barlow's valuable paper in the 7th volume of the Guy's Hospital Reports, as well as some observations by the author in the same volume.

exist with *tuberculosis*. Cyanosis affords a complete protection against it, &c."*

Many of the cases of cyanosis adduced in the earlier portions of this memoir wholly negative the opinion of Rokitansky, and prove that, on the contrary, the subjects of congenital malformation of the heart are rather excessively prone to suffer from tuberculosis at all ages, but especially where life is somewhat unusually prolonged.†

Hæmorrhages from the nose, mucous membrane of the bowels, and lungs, are liable to occur at all periods of the lives of cyanosed patients, but are most frequent towards the close of existence, when congestion is greatest, and the obstruction to the circulation resulting from the advance of structural impairment of the organs is at its height. The lighter forms of hæmorrhage are usually followed by temporary relief; but hæmoptysis, as it involves an extremely severe lesion, and almost invariably results in these cases either from tubercular or pneumonic degeneration of lung-substance, from bronchitis, or from some new and serious impediment to the pulmonary circulation having its seat in the pleuræ, left heart, or systemic vessels, must always be viewed with the utmost apprehension.

Hepatic, splenic, gastric, and enteritic congestions, may be regarded almost as the necessary and physiological attendants upon congenital cardiac obstruction, and are constantly liable to be determined into states of more or less active disease. A foul tongue, offensive breath, imperfect digestion, a general tendency to defective nutrition, bad or capricious appetite, a faulty biliary secretion, with constipated or irregular bowels, are the almost inseparable concomitants of the state under investigation; and hence the danger attendant upon diarrhœal and dysenteric attacks in these patients.

Whenever the urine is found to be albuminous in a case of acute exocardial or endocardial disease, the prognosis is bad, as here it rarely fails to happen that the kidneys are the seat of fixed organic lesion, of which the cardiac mischief is not the cause, but the result. Possibly, though not very probably, patients thus affected may recover for a time, but, as far as I have observed, they most frequently sink under the immediate attack, or only survive it for a very brief period, during which the abatement of symptoms is extremely partial. This arises from the fact that, in acute cardiac and arterial diseases of renal origin, the lesions are generally of the most extensive and the gravest character. In all forms of chronic heart-disease where the urine is albuminous, when the patient is first seen the prognosis is invariably bad, especially where the duration of the renal affection is not known; and so long as this symptom resists judiciously-employed treatment, the patient's condition must be regarded as very critical. The sudden appearance of albumen in the urine of an individual suffering from organic heart-disease, is always a most discouraging symptom; but the practitioner should not on that account neglect to employ systematic endeavours to relieve its cause, as it may depend upon little more than excessive congestion of the kidneys, which may, not improbably, be removed, when the bulk of the circulating fluid, or of any ascitic effusion which may be present, has become diminished, or when the renal functions have become lightened by moderate excitation of the cutaneous and intestinal actions.

The secretion of scanty and high-coloured urine, as evidencing renal congestion, is a frequent characteristic of cyanosis; true Bright's disease is, I believe, only noticed in these cases as a very rare and accidental complication. The subjects of cyanosis could scarcely live through those vicissitudes or excesses which occasion the commoner forms of mottling and granular disease of the kidney. Hence, whenever failure of the renal function, or an albuminous state of the urine, occurs in these cases, it must be viewed with much apprehension, as indicating the superaddition of more or less severe structural alteration in organs which have never performed their functions with healthy freedom. It is here that the most delicate discrimination of the pathologist, and the nicest judgment of the therapeutist, must combine in an endeavour to relieve the weak and overloaded heart, to remove bronchitis, that the pulmonary transpiration may be rendered as free as possible, to increase the action of the skin, to exclude from the diet and medicine everything that may act directly or indirectly as an excitant to the suffering organs; and, in short, so carefully to free the kidneys from all excessive duty and additional causes of irritation, that the congestion may have a tendency gradually to subside, and to leave the organs capable of performing a moderate function with comparative ease.

In concluding this perhaps unduly lengthened memoir on diseases of the pulmonary artery, I should feel that I owed an apology to those who may have done me the honour to accompany me throughout my investigation, did I not believe that I have rendered some service to medicine in proving that a class of diseases which has hitherto been considered of such unfrequent occurrence, so barren of interest, and so destitute of importance, as scarcely to deserve a separate place, or even more than a passing notice, in systematic works, will, upon being observed with proper attention, reveal a series of physiological and pathological facts of the highest interest, and a set of therapeutic indications of no trivial moment.*

Chittagong, Bengal, November 1st, 1850.

THE END.

WILSON AND OGILVY, 57, SKINNER STREET, SNOWHILL, LONDON.

* As quoted by Dr. Stillé, Philadelphia Med. Examiner, April 1849, and Ranking's Retrospect, vol. x., from Handbuch der Path. Anat. Bd. ii. p. 510.

† See *Dr. Ramsbotham's* case of obliteration of the pulmonary artery: death at the age of sixteen years from tubercular disease. *Dr. Shearman*, imperforate pulmonary orifice in a girl, ætat. 8 years. Lungs found very full of grey tubercle. *Dr. Cheever*, marked contraction of the pulmonary orifice in a boy aged 13½ years; cyanosis from the first months of infancy. Tubercles were found pretty generally diffused through the substance of the lungs. *M. Schuler*, cyanosis: death at seven weeks; scrofulous degeneration of the mesenteric glands. *Mr. Houston*, contraction of the pulmonary artery, with cyanosis; death from phthisis and bronchitis at the age of 3 years. *M. Cuillot*, cyanosis from birth; death at 11 years and 7 months. Lungs condensed, and in an advanced state of phthisical degeneration. *M. Lexis*, extremely contracted state of the pulmonary artery in a girl 5½ years of age. The pulmonary tissue contained a few tubercles. *Mr. Napper*, contracted pulmonary artery, with cyanosis, in a child aged 5 years and 10 months. Lungs tubercular. *Messrs. Aran and Bennett:* a female, ætat. 20. Great contraction of the pulmonary artery; very aggravated cyanosis; great consolidation of the lungs by tubercles; death from hip-disease and erysipelas consequent upon caries of one of the ribs. Case occurring at *Guy's Hospital:* lad, ætat. 16; contraction of the pulmonary artery; cyanosis. The right lung contained miliary tubercles; the left was engorged with blood, and contained at its apex a tubercular excavation, apparently of long standing. *Dunglison:* a female, ætat. 21; congenital contraction of the pulmonary artery, with cyanosis. Both lungs studded throughout with crude and miliary tubercles, none of which were softened. *Dr. Craigie:* a man, ætat. 44; pulmonary valves contracted so as to form a ring, which could only admit the point of the little finger. Both lungs contained tubercles, and were much consolidated: the apex of the left was occupied by a large tubercular cavity. *Mr. Le Gros Clarke:* case of a man, ætat. 19; great contraction of the pulmonary orifice; cyanosis. The lungs presented tubercles at different parts.

* It was originally my intention to have concluded these observations with a concise abstract of views relative to the treatment of cardiac diseases. Finding, however, that my remarks extended themselves beyond the due limits of this already very lengthened paper. I have embodied them in a separate treatise—"On the Management of Diseases of the Heart, and of Aortic Aneurism"—which is now publishing in Calcutta, and which, I trust, will shortly be submitted to the profession at home.



PISIFORM LITHIC CONCRETIONS OCCURRING IN A CASE OF ALBUMINURIA.

BY SPENCER THOMSON, M.D.

[*Extracted from the Monthly Journal of Medical Science.*]

THE annexed drawings represent microscopic views of a series of uric acid deposits (the "pisiform lithic concretions" of Dr Prout[1]), which occurred during the progress of a case of albuminuria, under the care of the writer.

The patient, a farm-servant, a stout muscular young man, æt. 21, of sanguine temperament, first applied upon the 24th of June of the present year. He attributed the commencement of his illness to cold taken six months previously, having got very wet, and dried his clothes upon him before a fire; at the time, he suffered from rigor and other pyrexial symptoms, and has never felt well since. He had been two months under treatment at the time of his application, but apparently no attention had been directed to the urinary affection, when first examined by the writer. He had been engaged for some days previously in mowing, during the extremely hot weather, perspiring profusely, and drinking plentifully of beer. The chief symptoms were, extreme debility; sleep broken and unrefreshing; mouth dry and disagreeable on waking; tongue posteriorly coated with thick white fur, anteriorly rather red, moist, papillæ prominent and broad; thirst constant; appetite very bad; great weariness of the lower limbs, more particularly after meals; bowels irregular; no particular complaint of pain; skin, when not perspiring from exertion, dry and hard; urine pale straw colour, acid, sp. gr. 1·025, the amount of albumen coagulated by heat or nitric acid, rendered it too solid to pour from the tube; breathing short, but chest sounded well all over; sounds and impulse of heart rather louder and stronger than natural; pulse 72. The patient was ordered at once to stop work,

[1] Prout on Stomach and Renal Diseases, fourth edition, p. 193.

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to wear flannel, to abstain from all stimulants, and put upon a regulated diet, chiefly farinaceous, with little or no fatty matter; warm baths every night for some time were resorted to; diaphoretics, alkaline salines, mild purgatives and alteratives were employed, along with two cuppings from the loins, followed by counter irritation; latterly mild tonics, serpentaria, tartrate of iron, uva ursi, &c. &c. were administered.

On first stopping work, the pulse fell to 56, and continued at that standard for some days; it then gradually rose to 72; the skin was very dry, but soon became moist under the warm bathing, and has continued so ever since, and the appetite was much improved. The only symptoms complained of during the treatment, directly referable to the urinary organs, were ardor urinæ during the continuance of uric acid secretion, and subsequently, aching in the groins, especially after exercise. At the present date, August 1, all symptoms of disease appreciable by the patient have disappeared; he eats, drinks, and sleeps well, feels strong and fit for work, to which, subject to regimen, he returned a week since. The urine, however, still remains coagulable, though not to the same extent as formerly; it is pale, clear, acid, sp. gr. 1·020, average quantity about three pints, occasionally deposits uric acid. The patient says his feet had always perspired very freely previous to this illness, they are now habitually dry.

The resemblance of the above case, in most of its features, to many others of albuminous urine renders comment unnecessary, except upon the peculiarities of the renal secretion. The sp. gr. 1·025 presented by the first specimen of urine examined, was doubtless owing to the loss by perspiration at the time, as, since then, 1·020 has almost uniformly been the density of the night's secretion, the daily quantity passed varying from forty-eight to fifty-six ounces. Notwithstanding the apparent abundance of albumen, it was evident that the above density was due to the presence of other constituents of the urine; for, after the separation of the former ingredient by heat and filtration, the density fell but one degree. Urea was present in good quantity; the uric acid, in the first specimens, was all precipitated spontaneously; by the addition of a stronger acid, not a single additional crystal was obtained; examination led to the belief that a large excess of hydrochloric acid was the cause of this precipitation. The urine, pale when passed, became much deepened in colour after standing a day or two. Decomposition was remarkably retarded; some of the urine kept for three weeks in an open vessel, at the end of that time, retained its acidity, and had not become in the least offensive;[1] healthy urine, under similar circumstances, becoming very offensive in forty-eight hours.

[1] The tendency to resist the usual progress of decomposition is common to urine depositing uric acid.

The first specimens of the pisiform uric or lithic acid concretions which were observed, presented to the naked eye the appearance of bright orange looking grains; under the microscope they appeared as delineated in figs. 1 and 2, the lustre being rather waxy; some, such as fig. 1 *a*, and fig. 2 *c*, approach very nearly the form of the usual rhombic crystal, others again, as fig. 2 *d*, represent the rounded form of the true concretion, all these latter encrusting hair-like filaments. In a specimen of urine passed a few days after that, from which the above were taken, the concretions appeared to remain suspended by threads of mucus; on placing a collection of these under the microscope, they appeared as represented at fig. 3; concretions in various forms encrusted simple filaments, some of which were apparently cylindrical, others flat and twisted upon themselves, as represented at fig. 3, *a a a*; besides these simple filaments, however, there were numerous other compound penniform, apparently articulated filaments, as represented fig. 3, *b b b*—on these were no encrustations. Amid the earlier deposits, two species of animalculæ occurred rather abundantly, fig. 3 a^* and b^*; the first a^*, slow in motion, the body slightly opaque, and continued into a long transparent proboscis; the second, b^* oval in form, motion rapid, and presenting traces of internal organization, which probably a higher magnifying power would have rendered better defined. Fig. 4 represents a few more of the encrustations. After the appearance of the deposit represented at fig. 3, the uric acid gradually began to disappear, and to become more normal in its crystallisation; figs. 5 and 6, taken from successive specimens of deposit at a few days interval, show the gradual approach to the common form of uric acid crystal, represented at fig. 7, these last, very small and thin, having been deposited from urine passed only a day or two previous to the entire disappearance of the free acid. Fig. 2 *a* and *b* represent a large organic or cellular globule formed in serous urine.

Fig. 1.—Pisiform uric acid concretions.
Fig. 2.—Pisiform concretions, most of them on filaments.
c.—Irregular crystallization between two filaments.
a and *b*.—Organic cellular globules.
Fig. 3.—Pisiform and amorphous concretions mingled with compound filaments.
a^* and b^*—Animalculæ.
Fig. 4.—Concretions on filaments.
Fig. 5.—Uric acid deposit, approaching normal condition.
Fig. 6.—Uric acid deposit, approaching to normal crystallization.
a.—Irregular crystalline mass of uric acid.
Fig. 7.—Normal form.

Not having met with any explanation of the origin of these pisiform concretions, the following, in the absence of direct proof, may be considered at least probable:—It appears that the true concretion is always formed upon one of the simple hair-like fila-

ments; at least, they are so generally found connected, we may be allowed to suppose that those apparently disconnected have become detached accidentally. The concretions in question are said to accumulate largely in the pelvis of the kidney.[1] It is not irrational, then, to explain the cause of this accumulation, by the supposition, that these filaments, which serve as a nucleus, have some attachment to the gland, and thus retain, for a certain time, the concretion formed upon them, which is meanwhile rounded by attrition and the constant washing of the urine. Respecting the compound filaments, I offer no explanation: they seemed, in the specimen in which they occurred, to be the cause of the suspension of the deposit in the urine.

Although Dr Prout describes these uric concretions as of frequent occurrence in serous urine, it is probable that they, and the consequent symptoms, were increased by the quantity of malt liquor which had been consumed. During hay and corn harvests, uric acid lithiasis is extremely common among the farm labourers in this part of England, in consequence of the large allowance of malt liquor, especially if the beer happens to be rather old and acid, or very new. The same disease, from the same cause, is of very common occurrence among the brewers' men of Burton-on-Trent.

Note.—The probable origin of the pisiform or concrete condition of uric acid, will, it is imagined, account for the great tendency to the formation of calculus which accompanies this form of excretion. It is evident that the retention of these concretions by their filaments, either in the pelvis of the kidney, or in the lower part of the ureters, must afford the greatest facility for the farther deposition of uric acid.

Haunton, near Burton-on-Trent,
August 2, 1846.

[1] Prout, p. 195.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

Dr. & a Parkes
with the Author's kind regards

ON THE

IDENTITY OR NON-IDENTITY

OF THE

SPECIFIC CAUSE OF TYPHOID, TYPHUS, AND RELAPSING FEVER.

BY

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[*From Volume XXXIII of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

C. AND J. ADLARD, BARTHOLOMEW CLOSE.

1850.

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COMMUNICATED BY

WILLIAM SHARPEY, M.D., F.R.S.

Received November 20th.—Read December 11th, 1849.

There are certain diseases which have peculiarities common to all, so characteristic, that, although we are ignorant of their intimate nature, and even of their exact seat, yet they naturally group themselves together and form a class admitting of one general description. This class exhibits equally natural subdivisions. The species, of which these subdivisions are formed, are distinguished from each other by peculiarities even more marked, if possible, than the class in which they are included is from all other classes. The great class to which I refer is that of—

Acute febrile diseases, having a determinate duration, and dependent for their origin on specific causes.

It includes, as distinct species, smallpox, measles, scarlet fever, typhus fever, typhoid fever, and relapsing fever. For many years the first three were confounded under one name, and it was only after the publication of Dr. Withering's essay that measles and scarlet fever were regarded as distinct affections, i. e. distinct as to their course, their symptoms, their lesions, and their causes. The three last-enumerated diseases are yet, by many, looked on as but varieties of one

disease, which merely presents differences in its phases according to epidemic constitutions, individual peculiarities, and hygiènic conditions. The great work of Louis on the Typhoid Affection, by affording a standard of comparison, materially lightened the labour of separating from that disease those which had previously been grouped with it.

The paper of Dr. Gerhard, in the 'American Journal of Medical Sciences' for 1837; the cases collected by Dr. Shatmak, and so ably analysed by M. Valleix; and the excellent paper of Dr. Stewart, in 1840, rendered it highly probable, although they did not prove[1] that typhoid fever and typhus fever were absolutely distinct from each other, i. e. were two species of disease, and not varieties of one affection.

In the 'Monthly Journal' of the present year I have attempted to determine absolutely the question of their identity, by an analysis of the course, the symptoms and the lesions of structure found after death in a certain number of fatal cases, collected by myself during one epidemic. It appeared to me that the conclusion which flows from that analysis is, that, so far as concerns their course, symptoms, and lesions, no two diseases can be more distinct. But not only do the diseases, i. e. smallpox, measles, and scarlet fever, with which I have classed these fevers, differ from each other in course, symptoms, and lesions, but they differ also with respect to the nature of their exciting cause. That cause is specific. We can generate inflammation of any organ at will by a variety of means; but by the application of *one* cause only can we excite smallpox, measles, or scarlet fever. In like manner, typhoid fever, typhus fever, and relapsing fever must require for their production the application of distinct specific causes, if they be distinct diseases belonging to the same class as smallpox, &c.

[1] See Christison on Fever, Library of Medicine; Watson's Lectures, 2d edit.; and especially vol. xii, British and Foreign Medical Review, in which the arguments adduced by the above-mentioned and other authors in favour of the non-identity of typhoid and typhus fever are ably criticised, and the following conclusion arrived at: "That they are the *same species* of disease, but *different* varieties of that species."

The peculiarity which entitles a cause to be termed specific, is that of exciting in those exposed to its action one, and only one, species of disease. Further, all specific causes, the products of individuals labouring under disease, can excite in other individuals only diseases resembling in all essential characters those present in the individual from whom they themselves sprung. Herein lies the test, the *experimentum crucis* by which the absolute non-identity of smallpox, measles, and scarlet fever is proved; for if the same cause, i. e. the poison generated by either, could not only produce the disease from which it had its origin, but also the other two, then the three affections would be regarded as varieties of one disease, and not as distinct species; just as *scarlatina simplex* and *anginosa*, and *scarlatina sine eruptione*, are varieties of each other; and just as *rubeola vulgaris, rubeola sine catarrho*, and perhaps it may be said, certain catarrhs without rubeoloid eruption are varieties of each other. We know, however, that smallpox, measles, and scarlet fever, owe their origin to different specific causes, and therefore we assert that they are distinct diseases. If the same difference in the specific cause of any two other affections be observed, then, however trivial the differences in their symptoms, they too must be held to be distinct diseases; *à fortiori*, will this be true of diseases differing from each other so widely as typhoid, typhus, and relapsing fever, in course, symptoms, sequelæ, and pathologico-anatomical lesions?

The object of this paper is to inquire, whether the specific cause of the three diseases just enumerated is identical?

The materials used for the solution of the question are the cases admitted into the London Fever Hospital during the years 1847, 1848, and 1849.[1]

The diagnosis of relapsing fever rests on the peculiarity of its course and symptoms; of typhoid and typhus fevers, on the skin eruption when present. The following are the diagnostic symptoms of these affections:

[1] To the medical officers of this institution, and especially to Dr. Tweedie, I am indebted for the liberality with which I have been permitted to make unrestrained use of the cases admitted into its wards.

Relapsing Fever.—Sudden rigors, headache, skin hot and dry, tongue white, urine high-coloured, bowels regular, occasional or frequent vomiting, loss of appetite, absence of abnormal physical abdominal signs. In severe cases, jaundice, profuse sweating on about the seventh day, followed by apparent restoration to health; on from the fifth to the eighth day, reckoning from the apparent convalescence, repetition of the original symptoms, with greater or less severity; again terminating in sweating, and then permanent convalescence.

Typhoid Fever—*Rose spots.*—The eruption in typhoid fever appears from the seventh to the twelfth day of the disease, very rarely later, and still more rarely at an earlier period. The characteristic spots are frequently preceded for a day or two by a very delicate scarlet tint of the whole skin.[1] The eruption itself consists of small spots irregularly scattered over the anterior and posterior surface of the trunk. The number of spots on the surface at one time ordinarily varies from six to twenty; sometimes there are very few, at other times, but infinitely more rarely, they are so thickly seated that scarcely an interval of normal cuticle is left between them.

The separate spots are circular, and of a bright rose-colour; this hue passes insensibly at their basis into that of the surrounding cuticle. Their usual diameter is about two lines. They are somewhat elevated; but, although perceptible to the finger passed lightly over the surface, they possess none of the seed-like hardness of the first day's eruption of smallpox, nor are they so prominent and perceptible to the touch as the papulæ of lichen. Their surface is rounded, lens-shaped, never acuminated. No trace of vesication can be detected on their apices. If tolerably firm pressure be made on these spots, they entirely disappear; but they resume their distinctive colour and elevation as the finger is being withdrawn. The above characters are preserved by each spot from its first appearance till it disappears. When, however, the duration of a spot is prolonged to five or six days, it usually becomes before that time very small, and less bright in colour; still, however, it disappears on pressure. The ordinary duration of each spot is about two days, but it varies from two to six days. Fresh spots appear every day or two from the outset of the eruption, till from the twenty-first to the twenty-eighth day of disease. This successive daily eruption of a few small, very slightly elevated, rose-coloured spots, disappearing on pressure, each spot continuing visible for three or four days only, is, so far as I know, peculiar to, and absolutely diagnostic of typhoid fever.

[1] This tint closely resembles, as I have elsewhere remarked, that of the skin of a person soon after leaving a hot-bath. It is important to be acquainted with it, because when it is more marked than usual, and sore throat is also present, it may be mistaken for the rash of scarlet fever.

Typhus Fever—*Mulberry rash.*—The eruption in typhus fever appears on from the fifth to the seventh day, and reaches its maximum amount in a day or two. It occupies the trunk and extremities, and occasionally the face. It consists of distinct spots and a subcuticular rash.

The frequent absence of one of these elements of the mulberry rash, the different proportions they bear to each other, the depth of hue of either, as well as the changes they undergo in their physical characters, cause considerable variations in the appearance of the rash in individual cases.

1*st. Distinct spots.* The spots vary in number. Sometimes they are very few, and pretty equally diffused over the whole surface; at others, while there are but few spots on the anterior surface of the trunk, the posterior is covered; or again, they may be innumerable anteriorly as well as posteriorly, ordinarily they are very numerous. Their size varies from a mere point to two, three, or four lines in diameter. Sometimes two or three spots coalescing, give rise to very large irregularly-shaped patches. Each spot passes through two, and in many cases, three stages.

First stage.—The spots on their first appearance are slightly elevated, somewhat flattened on their surface; have a dusky pinkish-red colour, somewhat like the stains of mulberry juice; and disappear completely on pressure, resuming their distinctive appearances as the finger is being

withdrawn. *Second stage.*—In from one to three days these spots undergo a marked change; they are no longer elevated above the level of the cuticle; their hue grows darker and more dingy; and instead of disappearing on pressure, they only fade, i. e. when the finger is firmly pressed on them they grow paler, but do not entirely disappear. In some cases the spots, after reaching this stage, pass into faintly marked, reddish-brown stains, and then vanish. *Third stage.*—In many cases, and especially those that are severe, the spots reach a third stage; their centres become dark purple, and are unaltered in appearance by the firmest pressure, although their circumferences still fade; frequently entire spots, circumference as well as centre, change into petechiæ.

The duration of each of the above-described spots is from its eruption till the termination of the disease. But a few large, almost scarlet patches, are occasionally seen on the back of the hand on the fifth or sixth day of the disease; these usually disappear altogether in a day or two.

The Subcuticular Rash.—When the trunk is covered with mulberry rash, many of the spots are usually pale, very imperfectly marked as spots, and run into each other; these spots are seen indistinctly, as if situated beneath the cuticle; or as the vulgar say, are "not well out." They give to the skin a mottled aspect, and on this mottled surface, as on a ground, the darker, more distinct, and decidedly marked spots are situated. Like the distinct spots, the subcuticular rash is deepest coloured on the most depending parts of the body.

The subcuticular rash may precede or be preceded by the distinct spots, i. e. the eruption is for a day or two very pale, and then some spots grow more distinct, or a few well-marked spots first appear, and then after a day or two the rash becomes more abundant. The diagnostic characters which separate the spots of typhoid from those of typhus fever are then derived from the colour, shape, duration, and the changes in physical characters which each spot severally experiences in the course of these diseases.

Number of cases admitted into the London Fever Hospital during the seven nine months of 1847; with which they laboured.

All the cases in which two or more persons suffering from typhus fever, typhoid fever, and relapsing fever, were admitted from the same house, into the London Fever Hospital, in 1847.
This table exhibits the age, sex, degree of intimacy of the individuals, and the nature of the disease under

Months.	Rose spots.	Mulberry rash.*	Months.	Ages of males.	Ages of females.	Degree of intimacy.	Nature of disease.	Remarks.
			May	30, 32 .	28	Lodgers	Typhus fever ...	All mulberry rash.
			July	25 ...	20	Husband and wife	Relapsing fever ..	No spots.
				17 ...	20, 33 .	Brother, sister, and sister-in-law	Typhus fever ...	
				14, 19	.....	Brothers	Typhoid fever ...	Both had rose spots.
			September	21 ...	24	Brother and sister	Typhus fever ...	Both had mulberry rash.
				.....	16, 21 .	Niece and aunt	Typhus fever ...	Both had mulberry rash.
				22 ...	24	Husband and wife	Relapsing fever ..	No spots.
				16 ...	34	Mother and daughter	Relapsing fever ..	No spots.
			October ..	55 ...	35	Husband and wife	Relapsing fever ..	No spots.
				40 ...	36	Husband and wife	Relapsing fever ..	No spots.
				13 ...	13	Brothers	Typhus fever ...	Both had mulberry rash.
			November	.....	21, 23 .	Sisters	Typhoid fever ...	Both had rose spots.

* I have no means of determining these particulars for 1847.

Number of cases admitted into the London Fever Hospital, during the separate months of 1848, with		
Month.	Rose Spots.	Mulberry Rash.
January	8	30
February	8	27
March	5	43
April	4	48
May	3	47
June	10	35
July	11	37
August	11	25
September	22	32
October	16	18
November	13	38
December	7	10

All the instances in which two or more cases of typhus, typhoid, and relapsing fever were admitted from the same house, into the London Fever Hospital, in 1848.
This table shows the age, sex, degree of intimacy of the individuals and the nature of the disease under which they laboured.

Month.	Ages of Males.	Ages of Females.	Degree of Intimacy.	Nature of Disease.	Remarks.
January	22	22	Husband and wife	Typhus fever	The man had mulberry rash, the wife no spots.
	30	24	Brother and sister	Typhus fever	Both mulberry rash.
February	—	11, 33	Mother and daughter	Typhus fever	The child had no spots.
	14	7, 11, 43	Mother and three children	Typhus fever	All mulberry rash.
	13, 50	55	Father, son, and sister-in-law	Typhus fever	All mulberry rash.
	5, 15	13, 44	Mother and three children	Typhus fever	The infant was convalescent, the mother had mulberry rash, the two elder children no eruption.
March	8	30	Mother and son	Typhus fever	Child had no eruption.
	13, 19	16, 20, 45	Mother and four children	Typhus fever	All mulberry rash.
	5	13, 44	Mother and two children	Typhus fever	Mother mulberry rash, children no eruption.
April	—	8, 18, 20, 24	Sisters and lodger	Typhus fever	All mulberry rash.
	18, 27	—	Slept in the same room	Typhus fever	Both mulberry rash.
	—	26, 49	Mother and daughter	Typhus fever	Both mulberry rash.
	—	9, 17, 35	Mother and daughters	Typhus fever	All mulberry rash.
	—	12, 42	Mother and daughter	Typhus fever	Both mulberry rash.
	20	16	Brother and sister	Typhus fever	The boy had mulberry rash, the girl no eruption.
May	20, 21	19	Brothers and sister	Typhus fever	The sister had no eruption, brothers mulberry rash.
	—	11, 18, 33	Occupied the same room	Typhus fever	The child had no spots, the others mulberry rash.
	19, 58	39	Father, mother, and son	Typhus fever	All mulberry rash.

Month.	Ages of Males.	Ages of Females.	Degree of Intimacy.	Nature of Disease.	Remarks.
June	17, 25	—	Slept in the same room	Typhus fever	Both mulberry rash.
	11, 38	—	Father and son	Typhus fever	Father mulberry rash, son no spots.
	12, 15, 42	29	Father, sons, lodger	Typhus fever	All mulberry rash.
July	19	20	Brother and sister	Typhus fever	Both mulberry rash.
	—	30, 40, 45	Inmates of the same ward at workhouse	Typhus fever	All mulberry rash.
	9, 13	—	Brothers	Typhus fever	Elder mulberry rash, younger no spots.
August	15, 44	—	Father and son	Typhoid fever	Both had rose spots.
	—	11, 36	Mother and daughter	Typhus fever	Both mulberry rash.
	47	47	Husband and wife	Typhus fever	Both mulberry rash.
	12, 17	—	Brothers	Typhus fever	Both mulberry rash.
	11, 14	50	Mother and children	Typhus fever	All mulberry rash.
September	4, 10, 13, 42	6½	Father and children	Typhus fever	Father and daughter had mulberry rash, the three boys no spots.
	—	5, 6	Sisters	Typhoid fever	Both had rose spots.
	47	11, 33	Husband, wife, and child	Typhus fever	All had mulberry rash.
	10	12	Brother and sister	Typhoid fever	Both had rose spots.
	11	13	Brother and sister	Typhoid fever	Both had rose spots.
October	16, 46	—	Father and son	The father typhus fever / The son typhoid fever	Mulberry rash. / Rose spots.
	19, 21, 29	7, 14, 20, 22, 60	Inhabitants of one house	Typhus fever	The youngest child had no spots, the other seven had mulberry rash.
November	26, 54	23, 49	Whole family	Typhus fever	All mulberry rash.
December	7, 23, 52	16, 34	Whole family	Typhus fever	All mulberry rash.

Number of cases admitted into the London Fever Hospital, during the separate months of 1849, with

Month.	Rose Spots.	Mulberry Rash.
January	9	24
February	6	18
March	5	22
April	4	15
May	4	16
June	11	12
July	16	6
August	15	8
September	15	11
October	23	6
November	10	5

All the cases in which two or more persons suffering from typhus fever, typhoid fever, and relapsing fever were admitted from the same house into the London Fever Hospital in 1849.

This table exhibits the age, sex, degree of intimacy of the individuals, and the nature of the disease under which they laboured.

Month.	Ages of Males.	Ages of Females.	Degree of Intimacy.	Nature of Disease.	Remarks.
January	9, 37	. . .	Father and son	Typhus fever	Both had mulberry rash.
	25	22, 30	Husband, wife, and lodger	Typhus fever	All had mulberry rash.
	26, 30	25, .	Husband, wife, and lodger	Typhus fever	All had mulberry rash.
	14, 50	47 .	Husband, wife, and child	Typhus fever	All had mulberry rash.
February	10, 48 .	. .	Father and son	Typhus fever	Both had mulberry rash.
	9, 14 .	16, 38, 53	Mother, children, and aunt	Typhus fever	The child, æt. 9, had no spots. All the rest had mulberry rash.
March	10, 17 .	. . .	Brothers	Typhoid fever	Both rose spots.
	6, 8, 29	. . .	Father and sons	Typhus fever	All mulberry rash.
	. . .	53, 61	Occupied the same room	Typhus fever	Both mulberry rash.
	24, 24, 28, 29, 31	26, .	Same room, lodging house	Typhus fever	All mulberry rash.
April	6, 21	33, 40	Mother, children, and nurse	Typhus fever	All mulberry rash.
	12	9, 16, 21	Brother and sisters	Relapsing fever	
May	21 .	21 .	Husband and wife	Typhus fever	Both had mulberry rash.
June	. .	12, 44	Mother and daughter	Typhus fever	Both had mulberry rash.
	. .	20, 30	Sisters	Typhoid fever	Both had rose spots.
July	. .	18, 18, 51	Visitors, lived in the same court	Typhoid fever	All had rose spots.
	21, 15 .	9, .	Brothers and sister	Typhus fever	All had mulberry rash.
August	42 .	49, .	Husband and wife	Typhus fever	Both had mulberry rash.
	10, 13, 16	. .	Brothers	Typhoid fever	All had rose spots.
	16 .	13, 54	Mother and Children	Typhus fever	All had mulberry rash.
September	. .	13, 16, 54	Mother and Daughters	Typhus fever	All had mulberry rash.
	40 .	47, .	Husband and wife	Typhus fever	Both had mulberry rash.
October	. .	23 .	Master had fever, diarrhœa, and passed blood	Typhoid fever	Rose spots.
November	40, 40 .	12, 34	Husband, lodger, wife, and child	Typhus fever	All had mulberry rash.
	9, 12, 22 .	. . .	Brothers	Relapsing fever	

Into these tables are collected *all* the cases in which more than one of a family, or more than one inhabitant of a house, suffering from Typhoid, Typhus, or Relapsing Fever, were admitted into the London Fever Hospital, during the periods specified.[1]

It will be observed, that in 1847 there were admitted into the London Fever Hospital two or more cases of typhus fever from each of five separate localities; that two cases of typhoid fever were received from either of two localities; and two cases of relapsing fever from each of five distinct localities. Twelve cases of typhus fever having been brought from five houses, four cases of typhoid fever from two houses,

[1] I ought to qualify the expression "*all* the cases," because it was not till the year 1848 that the diagnosis between these diseases was made with reference to *all* the cases admitted into the Hospital. The consequence is that not only are there no data for determining the numbers of each of the three affections received into the Hospital during 1847, but there are no data for determining, in many instances, to which of the three diseases any given case ought to be referred; i. e. the past histories of particular cases were only partially obtained, the exact locality from which the patient came was not recorded, the name of the street, or even parish, being often all that was ascertained. I have, therefore, been obliged to omit very many cases received into the Hospital in 1847; but I have included *all* those of which the records available for my purpose permitted the diagnosis to be made, and the locality from which the patient came, to be learned. In 1848, comparatively very few cases were admitted of which the diagnosis was not recorded, and the exact residence ascertained; and during 1849, the greatest care was taken to ascertain the locality from which each case was received. Since the middle of 1848, the diagnosis of the cases here used has been made or verified in nearly every instance by myself. In 1849 in every case. Before the middle of 1848, the characters of the spots were in many cases recorded by my friends Mr. Sankey, or his assistant, Mr. Humphrey, i. e. if two of one family entered the Hospital, the notes of one of the cases were frequently taken by either of those gentlemen, while I kept record of the other; this fact I regard as valuable, because it was only on collating these notes, eighteen months after they were made, that I became aware of many of the facts embodied in this paper.

and ten cases of relapsing fever from five houses, in the course of six months. During the same time not a single example was observed of either disease communicating the other, or of cases of the three diseases, or even of two of them, being generated by the same cause. All these diseases, be it remembered, prevailing in this city at the same time.

During the year 1848 there were admitted into the London Fever Hospital two or more cases, one of which presented the symptoms of typhus fever, from each of thirty-four localities. These thirty-four foci of disease yielded on the whole 101 cases. During the same year, more than one fourth of the cases of fever received into the Hospital were examples of typhoid fever, therefore one fourth, i. e. twenty-five of the 101 cases ought, if typhoid and typhus fevers are but varieties of each other, to have presented the symptoms of typhoid fever;[1] but, as the above tables show, in one instance only were two patients, one of which laboured under typhoid, and the other under typhus fever, brought from the same house. The cases referred to are those of a man æt. 46, who was admitted October 10th, 1848, with well marked typhus fever, and his son, æt. 16, who had been received into the Hospital

[1] Although one could not have expected the cases of typhoid and typhus fevers, that is to say, supposing their cause was identical, admitted from any locality, to have borne to each other the exact proportion that the total numbers of the cases of either disease, admitted into the Hospital during the same period of time, did to each other, yet an approach to that proportion ought absolutely to have been present in a majority of instances; while if occasionally the cases of either disease were proportionally too few, they would on other occasions have been, if derived from the same cause, proportionally too many. I have, therefore, used the relative proportion of the cases of the two diseases admitted into the Hospital as a standard. It appears to me that correct conclusions are, in a question of the nature considered in the text, more likely to be arrived at by cumulative evidence, such as is above adduced, than by the application of the calculus of probabilities to any one number of cases, however large that number might be.

on September 19th, with equally well marked typhoid fever. The diagnosis of the latter case was made by my friend Mr. Humphrey. I verified the diagnosis in the case of the father. But in this apparent exception to the rule, the mother of the boy had visited him in the Hospital, and therefore might have carried the contagion of typhus fever to her husband. The father, moreover, had been little exposed to the contagion emanating from the son, because the latter, a vagabond, at variance with his father, was from home when he was taken sick.

From January 1st to November 26th, 1849, there were received into the Hospital two or more cases, of which one presented the symptoms of typhus fever, from each of eighteen separate localities. These eighteen localities afforded fifty-one cases. During the same eleven months nearly half the cases received into the Hospital were suffering from typhoid fever. The whole number of cases admitted was 262; of typhoid fever, 116—of typhus fever, 143—therefore nearly half, or exactly 22·6 of the 51 cases admitted from the eighteen localities ought to have had typhoid fever, i. e., if the cause of the two fevers is identical, while, as the above tables show, not one of the 51 presented the symptoms of that disease.

We see from these tables, moreover, that in 1848, two cases of fever, one of which presented the symptoms of typhoid fever, were admitted from each of five distinct localities; now, as rather more than three fourths of the cases admitted into the Hospital during the same year, laboured under typhus fever, the remaining five of the ten ought to have had typhus fever, but one only did so. To this apparently exceptional case I have before referred.

From January 1st to November 26th, 1849, two or more cases, one of which was suffering from typhoid fever, were received into the Hospital from four localities, in the whole ten cases. Seeing that rather more than half the cases admitted into the Hospital during the same period had

typhus fever, the remaining ten cases ought to have presented the symptoms of typhus fever, if, as I have before observed, the cause of the two diseases is identical. But in not a single instance was a case of typhoid fever and a case of typhus fever admitted into the Hospital, from the same house, during the eleven months of 1849.

The foregoing tables demonstrate, that in every month of 1848 and 1849, several cases of typhoid fever and typhus fever were admitted; that the epidemic constitution favorable to the spread of typhus fever had little influence in diminishing or increasing the absolute number of cases of typhoid fever;—thus, during the first eight months of 1848, 60 cases of typhoid fever, and 292 cases of typhus fever were admitted into the Hospital; and during the corresponding months of 1849, 70 cases of typhoid fever, and 121 only of typhus fever; so that, while the cases of the latter disease had diminished nearly three fifths, the cases of the former had increased only one sixth.

As some writers have asserted that there are certain transition cases to be observed, marking the passage of one epidemic constitution into another, I ought here to remark, that with reference to the characteristic peculiarities of typhoid fever, and the rose spots in particular, they were as well marked in the autumn of 1846, as during the epidemic of relapsing fever in 1847, or of typhus in the autumns of 1847 and 1848, or as they are at the present moment. The spots have undergone no change, have experienced no modification; although the epidemic constitution, on which the difference in the rash is said to depend, must have varied more than once. The same is true of the mulberry rash of typhus fever. It presented, in the few cases observed in 1846, the same characters as during the epidemic of 1847-48; the same characters during that epidemic as at the present moment, when the number of the cases of typhoid fever bear to that of typhus fever the proportion of three to one.

So with regard to the intestinal lesion. In all the fatal cases examined in the three years referred to, in which the mulberry rash existed during life, Peyer's patches, and the mesenteric glands were absolutely free from disease, and in every fatal case in which rose spots were noted during life, serious lesion of the agminated and mesenteric glands was discovered after death. The lesion, like the eruption, was quite unmodified by that epidemic constitution which favoured the spread of typhus fever in 1847 and 1848;—for example, although the constitution of the autumn of this year (1849) favoured the spread of typhoid fever, yet, when a man and his wife were admitted in August with typhus fever, the mulberry rash preserved its characteristics unmodified, and when they died, as both did, Peyer's patches and the mesenteric glands were found to possess their normal anatomical characters.

There are a few cases included in the preceding tables, which may here be more fully adverted to with advantage. In November and December 1848, forty-eight cases of typhus fever, and twenty of typhoid fever, were admitted into the Hospital, i. e., nearly one third of the patients were affected with typhoid fever. At the latter end of October, 1848, a boy, 14 years of age, went to reside with a family named Mitchell, in Adden Place, St. Pancras. The Mitchells were at that time in health. The boy left his own home because his brothers were "down with the fever." This lad was, early in November, admitted into the Hospital, suffering from typhus fever. Early, also, in the same month, the man Mitchell, aged 29 years, with whom the boy lodged, the man's daughter, aged 7 years, and a female lodger, aged 22, were also admitted with typhus fever. The other members of Mitchell's family, expelled from Adden Place, then removed to 21, Hertford Street, at least a mile from their former residence. At this time, so far as I could learn by personal inquiry, there was no fever in Hertford Street, and *certainly* none in the house in which they had taken up their

residence. On November 22d, the two sisters of Mitchell's wife, aged respectively 14 and 22, who had removed from Adden Place with Mrs. Mitchell and her infant, aged 4 years, were received into the Hospital, both suffering from typhus fever. On December 8th, the landlady of 21, Hertford Street, aged 60 years, was admitted with very severe typhus fever; and on December 20th, the son-in-law of the landlady was also admitted with the same disease. I subsequently saw Mitchell's infant, aged 4 years, at its own home; it was similarly but very slightly affected. The only member of the family that escaped was the woman Mitchell, and she had had "spotted typhus fever," according to her own voluntary statement, some few years before.[1] Here was a group of persons, whose ages varied from four to sixty years, and whose constitutional predispositions also must have varied infinitely, for there were several of them unconnected by blood, exposed to the poison of typhus fever, (introduced among them by the lad aged 14,) at a time when typhus fever was only twice as prevalent as typhoid fever. What was the result? Did one third of the eight have typhoid fever? No, not one.

In December, 1848, ten cases of typhus fever and seven of typhoid fever were admitted into the Hospital. Five cases came from one house; these five individuals varied in age from 7 to 52 years,—their degree of relationship was, grandfather, daughter, and three grand-children. All five

[1] I may here observe that I have never known the same individual to be affected twice with typhus fever. The same person has been admitted twice, or, indeed, oftener, into the London Fever Hospital. But on reference to the Hospital records, or to my own notes, I have invariably found that such persons had at the one time a different disease from that present at the other. Thus two boys were admitted, Sept. 1849, with typhus fever; they had been inmates of the Hospital in 1846. Reference to the Hospital records of that year proved that they then had relapsing fever. I know no evidence, I repeat, to prove that typhus fever attacks the same individual *twice* more frequently than typhoid fever.

had well marked mulberry rash, were unequivocally affected with typhus fever. It is evident that, as at this time, the number of those admitted with the two diseases was pretty nearly equal; two of these five ought, if the cause of the two diseases is identical, to have had typhoid fever, with rose spots.

In March and April, 1849, eight cases of typhoid fever, and thirty-one cases of typhus fever, were admitted into the Hospital. Between the 19th of March and the 10th of April, eight persons were brought to the Hospital from one room, suffering from fever. Did one fourth present the rose spots of typhoid fever? No, not one—all had well marked typhus fever.

In September, October, and November, 1849, eighteen cases of typhus fever, and forty-eight cases of typhoid fever, were received into the Hospital, *i. e.*, nearly three times as many cases of typhoid fever as of typhus fever. During the same three months, a mother and her two daughters, aged respectively 54, 16, and 13; a husband and wife, aged 40 and 47; a husband, wife, child, and lodger, aged severally 40, 39, 12, and 40—i. e., in all nine persons, were brought from three localities. At least five ought to have had typhoid fever, if that affection and typhus fever are due to the same specific cause. Was it so? No, in every case the persons secondarily affected, whatever their age or sex, had the same disease as the individual from whom they caught it.

In April, 1849, a girl, suffering from relapsing fever was brought from a house in Fulham — in a few days her brother and two sisters were admitted into the Hospital. Did either of the three latter have typhus fever, which was the prevailing disease, or typhoid fever, which was then also very much more widely-spread than relapsing fever? No; all had the same fever.

Although not absolutely necessary for the purpose of my argument, I may observe that I have visited, in a few in-

stances, the houses[1] from which more than one individual affected with typhoid fever, or typhus fever, were brought to the Hospital, without being able to detect any hygiènic differences in the condition of the people, or in the localities themselves to modify the exciting cause.

Before concluding, it will be well summarily to repeat, that in 1848, one fourth of the cases admitted into the Hospital had typhoid fever; while, from thirty-four foci of typhus fever, yielding 101 cases, there was brought to the Hospital once only a case of typhus fever and a case of typhoid fever from the same house; and during the same time, among five localities, affording nine cases of typhoid fever, one locality only, viz. the house from which the father and son before referred to were brought, yielded a case of typhoid and one of typhus fever. That in 1849, although eighteen foci of typhus fever yielded fifty-one cases, and four foci of typhoid fever afforded ten cases, not a single example of the two diseases being received into the Hospital from one house occurred. With reference to the exceptional case, I must observe, that for exceptional cases to be of any value in proving the identity of typhus fever and typhoid fever, they must be met with more frequently than similar exceptional cases are met with in diseases having a specific cause, universally acknowledged to be different.

Now, the following facts prove that, with respect to measles, scarlet fever, and typhus fever, such exceptional cases are as frequent as with respect to typhoid and typhus fevers. During the last three years I have seen a case of typhus fever brought into the Hospital from a house in which all the children were suffering from measles; another case of typhus fever brought from a house in which the children had scarlet fever; a girl admitted with scarlet fever, who

[1] These houses were situated in courts or streets in the City, Bethnal Green, St. Pancras, and Holborn. I visited too few and made too imperfect inquiries to draw any *strict* inferences, but my *general* impression is stated in the text.

had been on terms of intimacy with another girl admitted shortly before with typhoid fever. And in these cases no direct contagion for the diseases under which the patients laboured could be traced. It is also important to observe, that the cases of scarlet fever admitted during the time specified, were nothing like so numerous as the cases of typhus fever or of typhoid fever.

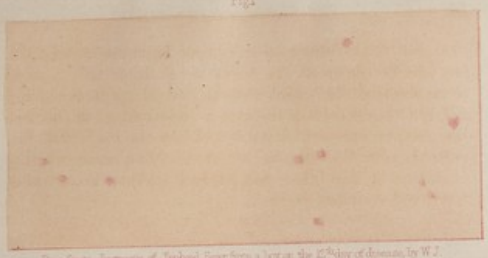
The facts contained in this paper appear to me to prove, incontestably, so far as induction can prove the point, that the specific causes of typhus and typhoid fevers are absolutely different from each other, and to render in the highest degree probable, that the specific cause of relapsing fever is different from that of either of the two former. I have elsewhere, as I stated at the opening of this paper, attempted to prove that the course, the symptoms, the lesion, and the sequelæ of typhoid and typhus are different, and as relapsing fever differs from both too widely, so far as symptoms and course are concerned, to be confounded with them, it follows that if smallpox be separated from measles, and both from scarlet fever, because their course, symptoms, lesions, and specific cause are different, so must, for like reasons, typhoid fever, typhus fever, and relapsing fever be separated from each other, and regarded as absolutely distinct diseases, not merely varieties of each other, as are scarlatina anginosa and scarlatina sine eruption, but distinct species of disease, as are scarlatina, rubeola, and variola.

I have, throughout this paper, expressed myself as if the specific cause respectively of typhoid fever, typhus fever, and relapsing fever, was an influence emanating from the bodies of those affected with either disease. With respect to the contagious nature of typhus fever, I know no one who entertains a doubt. If typhoid fever be contagious, it is infinitely less so than typhus fever. My experience leads me to regard it as contagious. Those who believe typhoid fever to be non-contagious while they admit the contagious nature

of typhus fever, cannot for a moment doubt the difference in the specific causes of the diseases. It would not, it ought to be observed, have weakened the force of the facts adduced if I had regarded these diseases as non-contagious, because the question here considered is not how the individuals respectively got the disease, but if the same cause, whether contagion or any other, can produce typhoid fever, typhus fever, and relapsing fever.

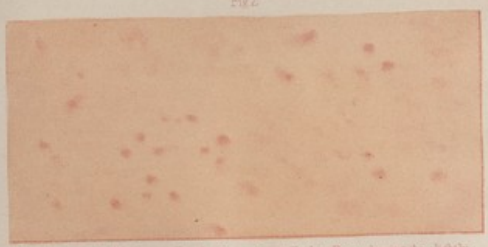
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Fig 1



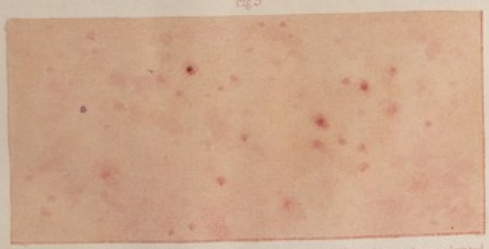
Rose Spots diagnostic of Typhoid Fever, from a boy on the 12th day of disease, by W. J.

Fig 2



1st Stage of the Mulberry Rash diagnostic of Typhus Fever shewing the slightly elevated spots and the subcuticular rash, from a man on the 6th day of disease. by W. J.

Fig 3



2nd & 3rd stages of the Mulberry Rash diagnostic of Typhus Fever shewing the more elevated spots and subcuticular rash, on the 11th day of disease. From nature by W. H. O. Sankey, M.D.

X.

On the Local Origin of the Yellow Fever Epidemic of British Guiana; in a letter from Daniel Blair, M.D., Surgeon General of British Guiana, to John Davy, M.D., F.R.S., Inspector General of Army Hospitals, &c., with Appended Documents.

Georgetown,
23d December, 1850.

My dear Sir,

I have not seen Dr. M'William's "Notes on the Boa Vista Fever;" but I have read the article in the "British and Foreign Medico-Chirurgical Review." I find by it, as well as a notice of our monograph in the "Lancet," that the subject of contagion of our late epidemic was not handled with sufficient detail. The fact is, that the idea of contagion in yellow-fever had become here so totally obsolete, that I felt reluctant to do more on that subject than make a declaratory statement of the universal opinion. When through you I was informed that a commission of military medical officers was sitting in London to investigate and decide on the subject of the alleged contagion of yellow-fever, I did not suppose it possible that there would have been the slightest difficulty in coming to a speedy conclusion in the negative. I find that I have mistaken the professional opinion at home, and that it will be necessary to supply some additional matter on the subject. For this purpose I enclose you copies of some papers, which, with the contents of this letter, be pleased to use in any way you think best. At the same time I must observe, that the fact of the opinion of contagion in yellow-fever here being abandoned by the unanimous consent of the entire community, professional and non-professional, who had countless opportunities of observing the disease, spread over

* [As indicated in the opening paragraph, this letter was written in consequence of some remarks contained in a critique on Dr. Blair's "Monograph on Yellow Fever," which appeared in the 6th vol. of the "British and Foreign Medico-Chirurgical Review." As an answer to these remarks, the Editor of that review has permitted it to appear in a late number (vol. vii. p. 554.) with the candid admission, — "That Dr. Fraser's communication to the 'Medical Gazette' is worthless as evidence on the question of contagion, and should not be used by any one." The additional and ample evidence now adduced, it is to be hoped, will have a more important issue, especially the appended documents, a selection from which was submitted to the reviewer, and noticed by him, but not published.] — Ed.

an eight years' epidemic, in all its phases, and from every point of view, seems to me testimony and proof as strong as the subject is susceptible of. There is certainly a moral certainty of the correctness of the conclusion so formed. This community is fully alive to the doctrine of contagion in disease; in fact, many diseases are reputed "catching" here, which are not considered so elsewhere. A short time since I incurred considerable obloquy by recommending the Governor to remove the Lazaretto for Lepers from the interior of Essequibo to the grounds of the Colonial Hospital, for the sake of superior superintendence. So they have not formed the opinion of the non-contagion of our yellow fever from any ignorance of the existence of such origin and mode of propagation of disease. And I submit that a universal conviction, founded on extensive experience, is very superior proof to that of any collection of facts, or process of reasoning, made by any individual after the lapse of some time from the date of the events, and without a searching cross-examination on the spot, and in which so much of the aspect of the whole question depends on the ingenuity of the advocate, and the omission, or suppression, or transposition of some facts. It might have occurred to the reviewer, in his notice of the opinion of the Medical Society, that the capability of arriving at a true and correct conclusion does not imply the power of communicating the mode by which the conclusion was obtained. Dr. Abercrombie, in his treatise on the Intellectual Powers, refers to a judge who was advised never to state his reasons, for they would be sure to be wrong, while in all probability his decision would be right. That rapid reasoning called *tact*, which the reviewer praises and recommends, is very frequently inexplicable to its most gifted possessor. And I imagine that a lover of truth in science would have been more impressed with the fact of the unanimous and perfectly disinterested conviction of the Medical Society, than any deficiency in their manner of enunciating it. The reviewer requires to know the number of practitioners in Georgetown or in the Colony, who were exposed to the quasi-contagion of the epidemic, and who escaped. It is difficult to give the entire number; but the "Local Guide" for 1843, contains the names of sixty-five, only two of whom suffered. The numbers, of course, fluctuated during the eight years of the epidemic, some dying or leaving the colony, and being replaced by others; and sixty-five therefore represents the number of only one year, who were exposed,—while three represents the number who suffered during the whole period. In the same authority — the "Local Guide" for 1843 — the names of the directors of the two hospitals are given. They number twenty-three, with the secretary. The duty of the directors was chiefly to check and pay the accounts, and visit

the wards to see that the rules and regulations were duly observed. None of these named suffered; nor do I recollect any others who suffered from an attack of the disease; in fact, it seemed as if those who were closest to the diseased, enjoyed the most perfect immunity. The number of resident surgeons of the hospitals during the course of the epidemic, whose term of service varied from six months to two years and upwards, was *six*. They also escaped. Dr. Smith, my predecessor as chief medical officer, he also escaped. All the dispensers (*three* in number, who did duty as such) escaped. He who died of the yellow fever, never did duty, but died the same evening he was gazetted to his office. All the stewards, eight in number, escaped. It is difficult even to guess at the number of nurses exposed, for they were frequently changed. They must have been very numerous. By a memorandum of Mr. W. B. Pollard, the financial accountant (and I prefer official and public documents to any others as vouchers), in 1842, the year of the second maximum of intensity of the epidemic, the average number of nurses (exclusive of other servants) in daily attendance in the Seaman's Hospital, was seven, and in the Colonial Hospital, fifteen. Washing is, and always has been, done for the Colonial Hospital by contract. The contractor is Mr. Friday Hopkinson. I do not insert any communication from him in the Appendix, as, although a man of very considerable property in houses and land, he can neither read nor write. Before 1840, Mr. Hopkinson contracted for the clothes-washing of both hospitals; and besides the assistance of his own family, required eight people constantly at work. After 1840, two women were employed separately for the Seaman's Hospital, and then only six assistants beside his own family were required by Mr. Hopkinson, and the same numbers respectively continue till now. The individual washers were often changed during the period of the epidemic, but none of them were ever affected with the prevalent disease. I hope I have now stated enough to rectify any looseness of manner with which my facts on the contagion-question have been charged by the reviewer.

The reviewer calls on me to substantiate the character which I gave to the letter of Dr. W. Fraser to Sir Andrew Halliday, which appeared in the "London Medical Gazette", and was replied to by the late Dr. Ferguson, in the same periodical, on the 24th March, 1838. It is easily done. I denounced his material statements in support of the importation and contagiousness of yellow fever, as *defective*, *hypothetical*, or *fictitious*.

His first material statement is that the disease commenced in Surinam, was transported thence to Barbados, and from Barbados was imported into Demerara. With Surinam, it little concerns us, although I have written to Baron Van Rader, the Governor, to obtain some information thereanent, in consequence of being informed by Mr. Van Oueneller of the B. G. Bank, that he was in Surinam in 1836 (the date given by Dr. Fraser), and that yellow fever did not exist there then. Dr. Fraser does not state the exact date of its introduction from Barbados. He does not state in what person, or bale of goods, or by what vessel it came. Where is the *corpus delicti?* Until the public and the profession here were startled by almost accidentally discovering Dr. Fraser's letter, twelve months after it was written, and twenty months after the epidemic had existed, no one person had ever heard of, or dreamed of, *importation*, as an imputed cause. Dr. Fraser had never trusted the secret to any one here, — neither the public, his professional friends, or the Government, whose health officer he was. About the time of the outbreak of the new pestilence, we find, in the Government Secretary's office, numerous quarantine communications regarding small pox,— but not one word about yellow fever, the danger of its being imported from Barbados, or that it already existed from neglect of quarantine regulations. Now these are fatal defects, unless they can be supplied or rectified by Dr. Fraser. Full explanations are the more necessary, as it happens that while our earliest cases in the epidemic could not in any likelihood have been earlier than the 1st of April, 1837, no yellow fever existed in Barbados during any part of the year. It is worthy of remark, in connection with this subject, that, although an immense intercourse existed between this colony and Barbados, and was not for a single day interrupted during the last lamentable devastations of yellow fever in 1847-48, of which you have had experience, we have been perfectly free from that scourge since 1845.

The second material statement of Dr. Fraser is, that the disease was propagated by contagion; and he illustrates this by the cases of Messrs. Wray and Howes, the first who died of the disease in Berbice, who evidently contracted it in Demerara, but who had had no known communication with the person or apparel of the sick; the family of whom did not become affected according to the testimony of the medical attendants, and whose cases, according to the same authority, had no relation with any of the subsequent cases in Berbice, except that of priority. In further illustration of its propagation by contagion, he asserts that Mr. (now Sir Robert) Schomburgk, the traveller, caught the disease by lying in a bed lately occupied by a young man who died of black vomit; that the Indian attendant caught the disease, and that Schomburgk and his retinue carried the epidemic into the "sylvan villages" of the interior. Now this is pure unmitigated fiction. Robert Schomburgk never had yellow fever,

although his brother Richard had, twelve or eighteen months afterwards. He never slept in the bed, or in the same room, in which the young man Cherrington had died. His Indian attendant never had the disease, and the disease never extended among his retinue, nor the other Indians of the interior, although a few Indians about the Sand-hills, who contracted it in town died of it there, and the disease died with them. This exquisite fiction about "poor Schomburgk," was retracted and apologised for by Dr. Fraser to Mr. Stutchbury, in presence of Schomburgk, when he next returned from the interior. What was Sir Robert Schomburgk's own opinion on the subject? In his "Description of British Guiana," published in 1840, he states that "immediate contact with the sick did not propagate the disease (yellow fever), nor did seclusion diminish it; and that there is no instance known in which it extended to the more elevated places of the interior." I cannot make out who the lady was, or what the mercantile firm which Dr. Fraser refers to, which was shut up for a couple of weeks, in consequence of the whole establishment being laid up by the prevailing malady. The case is very defective in being anonymous. It is singular that in the *beginning* of the epidemic they should all recover in that house.

We come now to the *hypothetical*. A ship, says the Dr. "arrives, and the captain uses every precaution to avoid having any communication with the shipping or shore. This precautionary sort of measure does very well for a week or a fortnight, but at the expiration of that period, the rigorous restrictions he has placed himself and crew under become relaxed, and they steal a visit to see some of their old messmates, who may perhaps be at the time very ill with fever. The visitor sits, has a long yarn with the visited; he returns to his ship," &c. &c. Now this is not only hypothesis, but impossible hypothesis, as any one who knows anything of this harbour could at once detect. The ships swing out in the river at cable length; the stream runs at the rate of three or four miles an hour; the river abounds with sharks; no approach to either ship or shore can be effected, except by the boats, which are entirely under the power of the officers of the ships. In fact, each ship is perfectly isolated, and remains so during the voyage; and whatever yarns the messmates may have with each other, they can have none with their neighbours, except through a speaking trumpet. Again, the Dr. says "an overseer of an estate comes to town with produce to be shipped; he visits some friend's house where the fever is raging; he returns to the estate, sickens and dies; and his fellow overseers go through a similar ordeal, having caught the infection from him: from this point the fever spreads itself to every family in the district." This is a mere fancy sketch, no real instance of which, I am sure, could be adduced. It is not

only not true as a generalisation, but the very reverse of truth. Dr. Fraser had no country practice during the time of the epidemic, and as, I am sure, no medical practitioner could have furnished him with materials for the description, he must have drawn entirely on his imagination. Many overseers and others who came to Water-street became affected on their return home; but the character of the cases was to have no issue, no lateral off-shoots. The epidemic seemed, in all cases, to have as it were dropped down into a family, and having struck its victim darted off to pounce down in the neighbourhood, or at a great distance. If Dr. Fraser, instead of speculating on fanciful sketches of the progress of the epidemic in the rural districts, where he had no personal experience, and, I feel confident, could have had no trustworthy correspondents, had observed the habits of the disease from his own house, he might, in a great measure, have steered clear of his gross errors. On the right hand house, there was a field for observation; on the left another; under his feet another; and before his eyes another. I, who was his left hand neighbour, took the disease. Besides having a nurse and my own servants constantly in the house, I had relays of bachelor friends, who every night watched by me and tended me. But no extension of the disease occurred in consequence. The next victim in the range was in the house to his right. Mr. McDougal, a young Scotch barrister, came up from Liguan, where he had been on a visit, caught the disease and died. Nobody else in that house (Sir Michael M'Turk's) became affected. Next, his (Dr. Fraser) turn came in the centre; he had had no intercourse with either parties, and was to windward of me. His wife was not aware of his doctrine of contagion, or did not regard it, and was unremittingly attentive; his son was there; his servants did not fly away; he recovered, and nobody else in his house sickened of the disease afterwards; and his neighbour opposite, Dr. Smith, who attended all the three cases, never was attacked. Dr. Fraser goes on to state something more tangible than his hypothetical cases. He states that "every member of the medical profession, who from their avocations were necessarily exposed to the infection, have been attacked and undergone a most severe fit of sickness; none of them, however, have died." This is fiction, except the last portion of the sentence. The medical men were extremely harrassed, but there was not much actual sickness of any kind among them; indeed, if we except Dr. McNish, who I believe was laid up for a time with hæmoptysis, and Dr. Smith, who was laid up for three months from dry belly ache, during the period referred to by Dr. Fraser, I question much if the medical men generally were ever freer from sickness. The two cases of yellow fever in town were Dr. Fraser and myself. I can

account for my own without the aid of contagion. The case in the country (Dr. Reid's) has been already referred to in the monograph. Dr. Fraser goes on to remark "the mortality has been great among the clergy." This is quite true; but the inference he would draw is not. Here again, those *most* exposed suffered *least* from the malady, and *vice versâ*. Those, the metropolitan clergy, whose avocation was among the stricken and the susceptible, passed through the ordeal least harmed; and those whose congregations were chiefly of the negro race, such as the London missionaries, and those of the rural parishes of the Scotch Church, suffered severely. This will be seen by an enumeration of the deaths within the period referred to in Dr. Fraser's and Sir A. Halliday's letters. They were

Messrs. Wray and Howes (London Mission) died.
Mrs. Rattray - - - „ - „
Mr. Murkland - - - „ recovered.
Mr. Kitley - - - „ - „
Mr. Williams (Plymouth Brother) - died.
Mr. Bryer (1st Minister of Christ Church) „
Dr. Hardy (Scotch Church) - - „
Mr. Stewart - „ - - - „
Mr. Haig - „ - - - „
Mr. McIntosh - „ - - - „
Mrs. Forbes - „ - - recovered.
Mr. Stevenson - „ - - - „
Mr. Anderson - „ - - - „
Mr. Menzies - „ - - - „

Now of all those cases, let Dr. Fraser point out one that had its origin in contagion, or, with a single exception, one that was even talked of as being suspected, in the remotest degree, of being concerned in the propagation of the disease. But turn we to those whose duties called them most to the bedside of the sick, and the funerals of the dead; to the clergyman of St. George's Church (now the Cathedral); to the clergyman of St. Andrew's Church, in Georgetown; and to him of Christ Church, also in Georgetown (whose predecessor lived only a few weeks after his arrival here). These clergymen minister to almost all the white inhabitants of the city. Mr. Fox, besides his other duties, had, during the whole epidemic, since the foundation of the Seaman's Hospital, solemnized the service of the Church of England every Sunday to the convalescent of the Seaman's Hospital. Mr. Lugar's labours had been unremitting, and he had his nephew, Mr. Austin (then lately arrived), ill of the gravior form of the disease in his house. Dr. Struthers was peculiarly exposed by his ministerial duties among the multitude of Scotch Presbyterians who inhabit the mercantile

part of the city. In addition to this exposure, he had at various times yellow fever cases introduced into his house. The Rev. Mr. Haig's case was one; the Rev. Mr. Stevenson's another. In a third instance, the captain of the "Duke of Gordon," Mr. Longmuir, was brought to his house, and there remained throughout a most severe illness and protracted convalescence. Dr. Struthers' family at that time consisted of thirteen persons. Now, neither of these clergymen, nor any of the inmates of their houses, ever contracted the disease. The Bishop of Guiana, not now in the colony, had a large family of children and a large retinue of servants. Two of the latter (whites recently arrived) died of the yellow fever, but the disease never spread in the house. During the presence of the disease in the colony, the bringing of the sick into the midst of families was of usual occurrence. No fear was felt. There was no difficulty in obtaining nurses; no danger was incurred. Several years after my own illness, I brought a favourite white servant who was very ill to the room next my own bedroom, that I might see him two or three times during the night. My brother-in-law, Dr. Manget, did the same with one of his servants. There was nothing remarkable or heroic in this; it was a mere matter of course, and practised by dozens without remark or laudation. I have not communicated, you will observe in the Appendix, with either the Roman Catholic pastors or those of the Wesleyan persuasion. The reason is, that none of either denomination, who saw the epidemic, are now in the colony. But, as far as I recollect, the Wesleyan clergymen had no deaths among them, and only one priest died of the yellow fever, although the services of their church brought them frequently among the Portuguese immigrants in the Colonial Hospital. The priest who died was considered at the time as having inhaled the malaria in crossing the Demerara River, in an open boat, at night.

I have now done with Dr. Fraser's most extraordinary letter. It may be thought strange, and it is so, that no one, save an anonymous correspondent of the "Guiana Chronicle," took the trouble to expose and refute a production so unfounded, at the time when it was discovered. That *exposé*, too, never saw the light, — no doubt for some personal reason of the editor, either of ill-will to the assailant or friendship for the assailed. Colonial apathy, which in many things disregards the opinions of "home," operated, likely, as a sedative. Where all disbelieved it here, where was the use of trouble merely to set the profession in England right! Compassion for the old gentleman who had so terribly committed himself, I know moved one individual to forbearance. Some despised the production, although endorsed by Sir Andrew Halliday. For there is no use in concealing the circumstance, that although Dr. Fraser was an old prac-

titioner in the colony, he never stood high, either with the public or the profession. Neither did Sir Andrew Halliday's character stand high in this community. Here he was celebrated chiefly for his credulity, on which some of the wags of the colony cruelly played, and for his patronage of quack medicines. However the neglect of the profession may be accounted for, Dr. Fraser's narrative remained without public contradiction from the faculty. No doubt many a successful fable has been similarly established. None of the chief practitioners seemed to regard the effect of Dr. Fraser's letter abroad, where the true circumstances were unknown, nor to care for it, except Staff-Surgeon Hackett, and his hands soon became too full of his own special duties to allow time for extraneous work.

I think the reviewer is hardly fair or correct in his estimate of the treatment described in the monograph. He says there is nothing new in it, and that it has been tried five hundred times, and that it is founded on a crazy hyphothesis long since exploded. I suppose he refers to Dr. Chisolm's mercurial treatment, which also was extensively tried and given up as useless here, when he inveighs against calomel. But the treatment recommended, I submit, is a very different affair. It is founded on no hypothesis whatever. It is purely empirical, and became established from the surprisingly beneficial effects found to follow its adoption. I should be very sorry indeed if the remarks of the reviewer were to deter any medical man, who has to encounter such fevers as are epidemic or endemic in this colony, from trying the plan recommended. The making of our medical men acquainted with the power of the combination of calomel and quinine *in large doses* in febrile diseases, I look on as a considerable compensation for the ravages of our late epidemic. The physician here now wields a power over the most formidable disease, such as he never before possessed, and with results as striking as the feats of the surgeon. On the day on which I got the October number of the "Medico-Chirurgical Review," while it was yet in my carriage, I had to see the daughter of Mr. W. Hick (a merchant, in Water Street) in consultation. I was told that on the Thursday previously, she had had apparently a paroxysm of intermittent fever; it left her on Friday, and she went out on Saturday morning. While out the fever returned. She had taken no medicine in the intermission. Notwithstanding active treatment, by purgatives, diaphoretics, &c., the hot stage never remitted up to Monday, at 3 P. M., when I saw her for the first time since the attack. She was then intensely hot; pulse rapid; headache intolerable; extreme restlessness and jactitation. We prescribed the large dose of calomel and quinine, and a blister to nape of neck; went back in four hours, and found a state of complete

apyrexia, and enjoyed a hearty laugh at the expense of the reviewer, whose criticism we then related to Mr. Hick. Calomel is very much disliked in this colony. It has hitherto been up-hill work for the practitioner to induce his patients to take it in any form, and it has frequently to be given in disguise. This repugnance to the mineral was inherited from our predecessors the Dutch, who considered mercury should never be given except in venereal disease, and existed long before the modern and improved views of the use of that medicine obtained in England. In some parts of Holland, I believe, it is considered disreputable to be known to have a mercurial prescribed for one. Influenced by this prevalent dislike to mercurials here; and from the ill effects that no doubt frequently follow the excessive use or abuse of them, I have sometimes been tempted to try a small dose of the compound, or a reduced proportion of the calomel, but I have seldom failed to regret the experiment. The formula became completely established for the treatment of yellow fever, and since the departure of that disease from among us, it has been most successfully extended to all the intensified forms of our endemic intermittent, except the congestive. I shall mention an instance, which has just now occurred, in illustration, and with the intention of enforcing my view, that the combination of calomel and quinine, given in large doses, is not the same thing as *with calomel alone*, as it is unfairly regarded and represented by the reviewer. It is a case now under my care,—Mr. R., a young Irish lawyer, only a few weeks in the colony. He came here on account of incipient phthisis, which commenced in July last, and which had proceeded to purulent expectoration, much emaciation, and some hæmoptysis. He has now no pectoriloquy, nor cavernous respiration, but humid rhonchus, and dulness under both clavicles, chiefly the right, as I found, in an examination at my own house. He had been a patient, he says, of Dr. Stokes, of Dublin, and, I believe, Dr. Townsend, of Cork. (?) He has been using, during all the voyage out, and while here till Saturday morning last, two or three tablespoonsfull of cod-liver oil daily, and keeping up a tartar emetic eruption below the right clavicle. Since he arrived in the colony, he says, he has gained five pounds in weight. I was called to him, for the first time, on the morning just mentioned. He was then suffering from fever, which had commenced the night before with a very slight coldness of the fingers. He had already taken purgative medicine, which had operated; I prescribed a draught of sol. acet. morph. and spt. æther. nitro., to be followed by six-grain doses of quinine when the fever subsided. Believing the case an ordinary intermittent, I did not return till the following morning. I found that although the fever had never subsided, he had taken the quinine, and was

cinchonized. Believing the fever kept up by bronchitic inflammation, I prescribed small doses of calomel and antimonial powder; the total amount of the former being four grains, and of the latter twelve grains. But the fever continued all Sunday and last night, and this morning I found it higher than ever. Still expecting it to subside, there having been free purgation during the night, and the skin, although burning hot, showing some moisture here and there, I left him without prescribing medicine. At half-past 1 o'clock I returned, found him lying on the sofa, as I had directed, his face and forehead deeply flushed, his eyes much injected, his pulse rapid, his skin burning hot, though moist; his tongue covered with a thick grey coating, but brown and dry at tip; no local pain complained of, *no headache*, even on shaking the head; *no irritability of stomach*. Except the injection of the eyes, there was no symptom in the slightest degree suspicious of yellow fever. I thought of bleeding him, to relieve the great vascular engorgement, but feared its after effects, and prescribed the twenty grains of calomel with twenty-four of quinine. At my visit this morning, I had the gratification to find a state of perfect apyrexia.* So much again for the resolvent effect of that combination on fever. I think that this case of intermittent fever, protracting its hot stage, so as to be of a continued type, was modified and exasperated by the previous course of cod-liver oil, but which he will resume when convalescent from the fever.

I see in the September number of the "Pharmaceutical Journal," a paper by Dr. Pereira, in which he speaks very disparagingly of musk as a medicine. I need only remark, that with it, as with every medicine I enumerated in the yellow fever "Materia Medica," I have seen its unequivocally beneficial effects over and over again. I don't know what chemists would say of it, but it seemed to invigorate and cheer, as if it had been a concentration of beef-tea. While at the Government Secretary's office, the other day, making sure that it contained no reports from Dr. Fraser on the importation-origin of our last epidemic, a conversation occurred between Mr. Austin, the assistant-secretary, Mr. Bent, secretary to the chief-justice, and myself. "As for my brother Hugh," said Mr. Austin spontaneously, and rather inconsequentially with the particular point of conversation, "he dated his recovery from his first dose of musk." His brother was Mr. Austin, who was ill in the archdeacon's family, and his case proceeded on to the snuff-like and clay-deposit vomit, at which stage the musk was prescribed.

* In a postscript of later date, it is mentioned, that his convalescence took place immediately, and without relapse.

I quite agree with the reviewer, that not to give the number of observations materially diminishes the value of the table, which proposes to show the influence of age on the mortality of yellow fever. I admit, also, that considerable improvements might be effected in the mode of grouping the *post mortem* appearances by the numerical method. But I don't think the candid inquirer can be at any loss to know how many livers were examined, when it is told that ninety-seven *post mortem* examinations were made. Louis's anatomical character of yellow fever was looked for on every occasion. I have not the review beside me, nor within easy reach; but, if I mistake not, the reviewer says that it is possible, according to the showing of the table, that only twenty livers were examined. Now, I deny this, unless it is possible for seven of that number to be "*enlarged*" and "unusually small" and "natural," at one and the same time. The obscurity of the table in reference to the liver, I think, arises chiefly from a redundancy; and the easiest remedy, I think, would be to strike out the first line, marked "natural 3," altogether. The table should record departures from the normal or healthy appearance only. I am of opinion, that the discrepancy which the reviewer affirms to exist between your description of the spleen and mine, in the fatal cases of yellow fever, is not discrepancy, but a very significant and harmonious coincidence, and helps to explain the difference of the results of treatment, and some difference in the constitution of the disease.

In regard to the severe remarks which the reviewer makes on the military authorities, about the site of the military hospital, it is possible they are undeserved. Might not the shifting of the fore-shores have materially altered the sanatory condition of the site? It is long since I read Sir Andrew Halliday's "Notes on the West Indies," but, if my memory serves me right, he then (1833 or 1834) states that the lately-built hospital was *within fifty yards of the sea*. If my reference be correct, then it is highly probable that its site was determined on when no such marsh as that described in 1840 existed, and that the authorities are not open to the measure of reprobation heaped on them by the reviewer.

Believe me to be
Ever yours faithfully,
(Signed) D. BLAIR.

Dr. John Davy, M.D., &c., &c.

APPENDED DOCUMENTS.*

No. 1.

To the Editor of the "Medical Gazette."

Demerara,
November 10th, 1838.

SIR,

A SHORT time ago I received from a friend in London a set of the "Medical Gazette," from the commencement of the present year to August last. In the first number I chanced to take up, was a letter from Sir A. Halliday, embodying an extract from one received by him from Dr. Fraser, Health Officer of this place, in which the latter endeavours to prove that the malignant fever that has unfortunately been so prevalent here for the last twenty months was propagated by contagion; he mentions three instances in which he states it was so, and which he says came under his own personal observation. Unfortunately *one* of his assumed facts is so contrary to the real, that I should deem myself wanting in my duty to the public, did I not take notice of it. The worthy Doctor says, "Mr. Schomburgk, the traveller sent out by the Royal Geographical Society of London, returned to us in June last, from his excursion up the Corentyne River, bringing with him, as his attendant, an Indian, indigenous to that part of the colony; Mr. S., be it remembered, had had repeated attacks of the colony fever and ague, and the colony bilious remittent fever, not only while engaged in his exploring duties in the interior, but while residing in Georgetown, after his return from his former expeditions. On his late return, however, he took up his quarters in the house of a druggist up town, who accommodated him with a bed and bed-chamber, in which, two or three weeks before, a young man belonging to the establishment had died of yellow fever and its terrific derivative, black vomit. Poor Schomburgk and his Indian attendant had no sooner come within the pernicious influence of the fomites of the bedding, &c., of the room, than first he was seized, and soon after his attendant, with the yellow fever, from which they both made a very narrow escape. Many of the Arrowaak and other Indians, who had come to town with their little commodities to traffic, caught the infection, returned to their sylvan villages, and not only fell victims to the disease, which they had casually caught, but communicated the infection to others of their tribe, who had not on that occasion left their homes, and of which some also died." The fact was, a young man died at our house during the prevalence of the fever, after about fourteen days' illness; however, amongst the symptoms of his disease he had NOT *Black Vomit*, nor was it (I have the authority of the medical gentleman who attended him for saying so) a decided case of yellow fever. Mr. Schomburgk, with whom I have the honor to be on terms of friendship, arrived in town about the time the Doctor mentions; he was invited to spend the short time he intended remaining in town at our house; he was not, however, accommodated with the same bed and bed-chamber in which two or three weeks before the young man had died (a decent manner, by the way, of accommodating a friend, even disbelieving as I do, the contagious nature of the disease); on the contrary, he never slept in the same room, much less upon the same bed, which indeed was never in the house during Mr. S.'s stay with us. Dr. Fraser must therefore have written upon very erroneous information, when he communicated his opinion to Sir A. Halliday. Of the nature of the disease, not being a medical man, I am not competent to speak, but I have frequently attended with impunity upon persons suffering under it. Another young man belonging to our establishment, newly arrived in the colony a short time since, was taken ill with the prevailing fever. I had him immediately brought into my own room, being more airy, and put upon my bed; he recovered, and as soon as he was convalescent, returned to his own room; I have in two other cases done the same, and have slept with impunity upon my bed afterwards without thought or fear of contagion. I have had frequent communication with Mr. Schomburgk since he departed upon his last expedition; in no one of his letters does he mention anything about the Indians, who accompanied him to town having died, or communicated the infection to others of their tribe; he certainly states that both himself and most of the Indians who accompanied him had suffered from fever, but he does not state it to have been of a malignant description, or that it in any one case terminated fatally. I should not have trespassed so long on your time, but when I reflect upon the dreadful consequences that might ensue from the spread of such a doctrine, I think it to be the duty of every person, as far as in his power lies, to controvert it.

Your obedient servant,

(Signed) I. S. STUTCHBURY,
Druggist, Georgetown, Demerara.

* The copies of documents in the Appendix are not numbered from any connection by which they should follow one another, but chiefly as they came to hand.

No. 2.

Georgetown,
28th December, 1838.

My dear Sir,

I regret that I cannot now enter into all the particulars that may be required in order to refute totally the opinion of Dr. William Fraser, in reference to the contagious nature of the yellow fever now prevalent in Georgetown. The reason is, the letters I expected from Berbice have not yet arrived, and therefore I am yet without *authentic* accounts as to the commencement and spread of the yellow fever in that county. As, however, I promised you to have some answer prepared to his strange epistle by to-day, I now proceed to notice some of the proofs of the contagious nature of the yellow fever as addressed by him in the "Medical Gazette" of the 20th January last. If you please to read over the ninth paragraph of Dr. Fraser's letter relative to Mr. Schomburgk and the Indians, you will find the particular propositions in reference to the point of dispute, viz., that Mr. Schomburgk and his Indians had yellow fever; that Mr. Schomburgk got it by sleeping in a bed, and residing in a bed-chamber, where a person had previously died of yellow fever and black vomit; and thirdly, that the Indians carried this disease into the interior where it spread. This is the most tangible of the cases adduced as proofs by Dr. Fraser; the rest of his cases are either anonymous or hypothetical, or placed beyond the reach of easy inquiry. On seeing this proof, I immediately went to Mr. Stutchbury's, a respectable chemist and druggist in America Street, and an intimate friend of Mr. Schomburgk, where Mr. Schomburgk had resided during his last visit to Georgetown from his expedition up the Corantyne River, and I made all the inquiries necessary, in order to obtain the true statement of facts. I also inquired of the medical gentleman* who attended the sick Indian in the retinue of Mr. Schomburgk, and read over the several letters which have been received from Mr. Schomburgk since he quitted Georgetown on his present expedition. What was my surprise to find that every assertion in the ninth paragraph of Dr. Fraser's letter is unfounded, first because Mr. Schomburgh *never had an attack of yellow fever;* that his only sickness while in town was a pulmonic disease, to which he is occasionally subject, and a slight attack of his usual fever, for which he took his usual remedy, quinine; secondly, that he came to reside at Mr. Stutchbury's a month after Cherrington's death, but never inhabited the room where the patient had died, nor even the same floor, nor

* Dr. Bonyun.

slept in anything but his own hammock while in town; that the patient (Mr. Cherrington) who died on the 23rd June, after eleven days' sickness in Mr. Stutchbury's house previously never had black vomit, and that his bed had never been used after, having been accidentally destroyed in consequence of being put out to sun, and forgotten till the rains had rotted it. As to the Indians (about six or seven in number) there happened no fever case among them. There were three deaths: the first was an Indian woman named Catherine, who died in Georgetown of dropsy, her husband named Solomon (who came from the Macousi country), was worn out by fatigue, suffered from nostalgia, was quite unfitted to accompany Mr. Schomburgk in his present expedition, was left behind, with his wife Catherine, in Georgetown; on the day of her death, 22nd September 1837, he left Georgetown for Berbice on his way home, but died shortly after his arrival on the Corantyne coast; he never was known to have had a day's fever all that time. The account of the third death I have from a letter of Mr. Schomburgk, now lying before me, dated "Mausi Village, Pirara, June 25th, 1838." I take the liberty of extracting the portion relative to this death. After lamenting for poor Catherine and Solomon, who died after he left the town, and of whose fate he had just then heard, he continues, "Their death has much concerned me. Solomon was a good Indian, and much attached to me. I have lately suffered another loss; my servant Van Meurs, a relative of theirs, died on the — instant. We were attending divine service; a missionary (the Reverend Mr. Youd) having lately arrived here under the direction of the Church Missionary Society, Van Meurs used that period, and broke in with his comrades upon the lately arrived brandy, and must have passed all bounds. We found him drunk; and I am much afraid he must have had another sup in the course of the night, having likely hid some brandy. Enough, we found him next morning in a most dangerous situation. All possible means in our power were resorted to, and towards evening we considered him past all danger; but about eight o'clock on Tuesday morning he breathed his last. With the exception of his immoderate love for spirits, he was a most useful and faithful servant."

This is all Mr. Schomburgh mentions of deaths among the Indians in any of his letters, up to the very last one received in Georgetown about a fortnight ago. He never mentions any epidemic or contagious disease at present or then among the Indians, nor of any deaths happening among his followers in the interior, except the case of Van Meurs; how, therefore, Dr. Fraser got his information, I cannot divine. I know of no other traveller lately in the interior

I had a case on board the barque "Highbury," whose second mate died with black vomit; and although the ship lay here for six weeks thereafter, no other case occurred. He was treated on board, and in the cabin where the mate and captain slept. I could add to the above a number of facts disproving the contagious nature of this obscure disease; and, if called upon, will do so in a more connected manner.

My dear Sir,

Yours, sincerely,

(Signed) E. P. CAMERON.

Dr. Blair.

Answers to Questions relative to Yellow Fever proposed by Dr. Blair.

Questions.	*Answers.*
Did you see anything to lead you to suppose the disease in question contagious?	Never; on the contrary, much to lead me to form the opposite opinion.
Who were the individuals attacked, and what the order and termination of the attacks?	1. Rev. —— Howe. Fatal. 2. Rev. J. Wray. Fatal. 3. ——— Recovered. 4. Rev. Joseph Ketley. Recovered. 5. Mr. Parish. Fatal. 1st. Two cases; one house at the Back Dam. 3rd. Clerk; store of Messrs. Foderingham & Co., Front Dam, or Water Street. 5th. Schoolmaster; residence, Middle Dam. 4th. Taken ill on plantation Blezendaal, six miles from New Amsterdam.
Did the disease spread immediately after the death of Rev. Messrs. Howe and Wray?	After July, the disease broke out among the ships in the river. The disease did not spread *immediately* after the death of any of the above fatal cases, there being a long interval between the last case on shore and the first among the seamen.
Did the yellow fever break out in the country, or town of Berbice first? If in town, what part of it?	The disease broke out first in town; the first two cases at the Back Dam, in Mission Chapel House; the next, Front Dam, store of Messrs. Foderingham & Co.; again at Blezendaal; and the fifth case on the Middle Dam of Town: very few genuine cases occurred in the country.
What signs, in reference to the tongue, eye, skin, pulse, and ejections of the stomach, do you consider necessary to indicate the presence of yellow fever?	Tongue at first dry and rough, gradually becoming brown, frequently red at the tip and edges; vessels of conjunctiva injected; pulse below the natural standard; skin dry and hot, becoming, sometimes *suddenly*, at other times *gradually*, yellow; in some cases minute petechiæ made their appearance; ejections from stomach *generally* at first

Questions.	*Answers.*
	dark coloured, but as the tongue, petechial skin, together with a hemorrhage from nose, commissures of the mouth, or even from the tongue itself, indicated the presence of a confirmed putrid disease, the black vomit speedily followed, unless the patient either rallied, or sunk from exhaustion.
What is the site of the headache generally complained of?	Across the temples and behind the orbits.
In cases of genuine yellow fever happening in Berbice, can you discover if the patients had been recently, before their illness (say fourteen days), in Georgetown?	Mr. Ketley came from Georgetown to Berbice, where he sickened; and the clerk at Messrs. Foderingham & Co.'s had recently arrived from Europe, *viâ* Demerara. The majority of my other patients were seamen.

(Signed) E. P. CAMERON, M.D.

Berbice, Dec. 1838.

No. 4.

1. Did the Rev. Messrs. Wray and Howe die of yellow fever, and with or without black vomit?	1. Mr. Howe died of yellow fever, and with decided black vomit. Mr. Wray had no black vomit, nor was his skin unusually yellow until after death.
2. How long after leaving Demerara did the attack commence?	2. Mr. Howe was attacked on the second day after leaving Demerara; Mr. Wray on the third day; and were ill five days: both died within twenty-four hours of each other.
3. Had these gentlemen one and the same, or a different domicilium?	3. They were both in the same house.
4. How many inmates were in the family, black, coloured, and white?	4. The family were nine in number; and during the illness of Messrs. Howe and Wray, four coloured nurses were added to the number.
5. Did Mrs. Wray or Mrs. Howe, the Misses Wray, or sick nurses, sicken with the same kind, or any other kind of fever, during, or shortly after, the illness of Messrs. Wray and Howe?	5. No person residing in the house, or any person in attendance, had any fever at that time, or shortly after, to my knowledge.
6. Did any person, doctor, or other, who might have been exposed to the exhalations of the sick chamber, sicken of the *same kind of fever*, then, or soon after such exposure?	6. Answered above.
7. Did the disease spread THEN; and who were the individuals attacked, the order of the attacks, and the termination?	7. Many cases occurred after, but the disease was principally amongst the sailors.
8. Did you see anything to lead you to suppose that the disease in question was contagious?	8. By no means.

Questions.	*Answers.*
9. In the early cases of *genuine* yellow fever, how many of your patients had been shortly before their illness (fourteen days) in Georgetown?	9. I had no patients with any kind of fever,-who had been in Georgetown fourteen days previous.
10. What signs, in reference to the tongue, eye, skin, pulse, and ejections of the stomach, do you consider necessary to indicate the presence of yellow fever?	10. Furred tongue; eyes at first red, soon after yellow; pulse strong; skin yellow; ejections from the stomach dark coloured.
11. What is the site of the headache generally complained of?	11. The headache, in almost every case, is in the forehead, and pain in the eyes.

The above questions were put by me on the one side of the page, and answered on the other by A. R. Hollingsworth, Esq. M.D., who was the medical attendant on the two rev. gentlemen.

(Signed) E. P. CAMERON, M.D.

No. 5.

Hoff Van Aurich House (Essequibo),
16th December, 1838.

My Dear Sir,

Your letter of the 4th did not reach its destination until the 14th. I have much pleasure in replying to your inquiries. With the exception to be mentioned, in all the cases of yellow fever that have occurred in my practice, the sufferers had been *recently* in *Georgetown*. The disease most commonly showed itself immediately on, or a few days after, their arrival on this coast. In a late fatal case of Mr. Howe's, the disease did not appear for about a fortnight after he left town. I know no other instance in which the attack was so long suspended. The exception to the remark that Demerara was the source of the disease to all my patients, occurred in *this house*. In August, 1837, there resided here Mr. and Mrs. Bratt, Mr. and Mrs. Fowler, and a white servant-boy of the latter. Mrs. Bratt, without having been in Demerara, was seized with yellow fever, had black vomit, and died. Mr. and Mrs. Fowler were much alarmed, feared contagion, and left the house just before the death of Mrs. Bratt. Their servant-boy had then been unwell for some days; but in the general alarm his case was overlooked, the more readily as he had managed to discharge his usual duties. When his master left this house, the boy was too ill to be moved, and I was called to him. I found him very ill; his

mouth filled with black vomit; he was delirious, and the pulse hardly perceptible. That I might appear to do something, I ordered a blister over the stomach, and hot brandy to be administered in teaspoonfuls. He recovered! owing very little to the Materia Medica. There was another victim; Mr. Bratt, after suffering excessive grief for the loss of his wife, at the end of a week, got likewise the disease, and died of black vomit in two days. The grounds around this house were last year, as now, ill drained. That the yellow fever is *not* contagious I am as sure as that I exist. I make a habit of smelling at the breath of those suffering under the disease, hoping to detect in a patient salivation; I am often bedaubed with the blood of the sick and with the matter they vomit; I sit for hours on the bedside, watching; I constantly observe the relations and attendants similarly exposed, and with absolute impunity. I shall be happy at all times to give you any information you may desire, and that my experience of eleven years in this colony furnishes.

I am, my dear Sir,
Yours truly,
(Signed) JAMES FRASER.

To Dr. Blair.

No. 6.

Georgetown,
November 15, 1850.

Dear Dr.

In reply to your note of yesterday, requesting me to state what prevented me in 1838 from sending the letter you have enclosed to me (which I gave to you some years ago), addressed to the Editor of the "London Medical Gazette," in reference to Dr. Fraser's communication with the late Sir. A. Halliday, as far as my recollection serves me, I think the circumstances were these; — that as my attention had not been directed to the article in question until so long after its publication, and never hearing an opinion expressed amongst my acquaintances other than that the disease was *not* contagious, and from the nature of my business, and residing in the part of the town where the fever was most rife, I was peculiarly favourably situated to hear what the opinion of the non-medical public was respecting it, — upon reflection, I thought the subject no longer one of public interest. I may add that, some months afterwards, I had an opportunity of re-

monstrating with Dr. Fraser for sending such a statement as he did without sufficient information. He admitted that he must have been misinformed; but that Sir. A. Halliday had made use of a private letter he had addressed to him which he had never intended for publication. This explanation confirmed me in my intention of not sending the letter.

I am,
Yours truly,
(Signed) I. S. STUTCHBURY.

Dr. Blair,
Surgeon General.

No. 7.

Georgetown,
12th November, 1850.

My Dear Sir,

I have only this moment received the enclosed from Mr. Forbes, which is a minute and I believe a correct statement of the cases detailed. Those with which I am acquainted are as follows : —

1. June, 1837.— Rev. Dr. McIntosh of St. Mary's, in the colony since 1829 ; ill about seven days.

2. July, 1837.—Rev. W. Haiys, formerly three years in the colony ; only three weeks in Georgetown from Granada.

3. Nov. 1837. — Rev. W. Stewart, about two years in the colony ; left Georgetown with me for Essequibo, and died on the fourth day at Cullen.

4. June, 1840. — Rev. R. Coltart, three years in the colony ; died after four days' illness.

5. June, 1841. —Rev. J. Train ; died in Georgetown, after upwards of two years' residence there and in St. Mark's.

6. In addition to these, there were the Rev. Mr. Gadstone, who died after two days' sickness in Georgetown, July, 1836; and

7. The Rev. Mr. McDonald, of St. Mary's, who died about the close of 1841 ; but I am not quite certain that theirs were cases of yellow fever. In none of the above list, however, did any contagion accompany or succeed the case ; and I knew most of the persons who attended them. In my own house Mrs. S. and the children, as well as

myself and the servants, were frequently in the sick room, and we neither feared nor experienced any sinister results.

I have the honour to be,
My dear Sir,
Yours very sincerely,
(Signed) J. STRUTHERS.

Dr. Blair, &c., &c.

Nos. 1, 2, 3, 4, 5, were well-established cases of yellow fever.—J. S.

No. 8.

I landed in Georgetown on the 13th July, 1837, with Mrs. Forbes and a white domestic, Anne Nicoll. None of us had ever been in the Tropics before. We went to Berbice on the 19th to the house of the Rev. J. Anderson : next morning I was attacked by what proved a long but not dangerous case of yellow fever ; Mrs. Forbes, Anne Nicoll, and the Rev. Messrs. Anderson and Menzies, attended on me constantly through the disease, as well as others ; none of them took it. Subsequently, about the beginning of October (the yellow fever having then become slightly epidemic in Berbice, for until then it was not, every case having been brought from Georgetown), Mr. Menzies was seized with it, and recovered : Mrs. Forbes saw him frequently during the disease. Mr. Anderson also was attacked on the 17th October, and was in very great danger ; Mrs. Forbes and Anne Nicoll, as well as others, were with him several days during the course of the disease, but did not take it. Mr. Anderson had been some three years and a half in the colony, Mr. Menzies about two. Rev. Dr. Hardy about one month in the colony, and fresh from Georgetown, was attacked in my house at Bohemia, eight miles from New Amsterdam, and died of it on the 24th October. Mrs. Forbes, Anne Nicoll, Mr. Heyland, and several others were often with him, as well as his wife, Mrs. Hardy, constantly ; but none of them took it. Rev. Mr. Buchanan landed in the colony in beginning of 1839, and died in Georgetown on the 28th September. He was said to have caught the disease from imprudently sleeping two nights on board a vessel in the river, in which vessel several of the men had died. Rev. Mr. Graham came to the colony in 1839, and died of yellow fever (black vomit) on August 28, 1842. Mrs. Graham, Mrs. Forbes, Rev. Stevenson, and

myself were with him constantly the last three days, and when he died: no one of those in attendance took it. Rev. Mr. Wray and Rev. Mr. Howe of the London Missionary Society were some days in Georgetown in May or June, 1837. Immediately on their return to Berbice the disease declared itself, and they died; but none of their families ever took it. I have never met any one in the colony who believed yellow fever to be contagious. A man seeing the disease and its sudden spread in a large community like Georgetown might be pardoned for believing the yellow fever to be contagious. But any one who has seen the disease, as I have done, in many isolated cases and scattered over a considerable period of time, and no one case of contagion even suspected, must be impervious to common sense if he still retained such a belief.

(Signed) FRANCIS FORBES,
Minister of St. Luke's.

November 11th, 1850.

No. 9.

My dear Sir,

I beg to apologise for not replying more promptly to your note of yesterday, but I have been much occupied with pressing business. With regard to the inquiry which you do me the favour of making as to my opinion, founded on my own experience and my knowledge of the sentiments of others, on the question whether yellow fever is contagious or not,—it is now a good many years since I have witnessed much of its ravages, and I cannot at this distance of time remember distinctly the observations of others; but my impression is, that it was not generally considered contagious, and this certainly was my opinion. As to your second inquiry, Have I myself suffered from proximity with those suffering from the disease? In 1838 and 1839 I attended, as a clergyman, very many who laboured under the disease, and I had no apprehension of contagion. Before I came to the West Indies I was for some years a curate in the north of Ireland, and there was in the habit at times of visiting persons suffering from typhus fever; and the feelings were very different with which I visited the two classes of patients. The typhus cases I attended with the grave consciousness that my taking the disease myself from the sick was a very possible contingency; but I do not recall to mind that in visiting persons suffering from yellow fever I had any apprehension

whatever of incurring any personal risk. No doubt, as I had not at that period been long resident in the colony, the possibility of myself taking the disease was often present to my mind; but the danger was connected in my thoughts, not with contagion, but with the primary causes of the epidemic, whatever those might be.

I remain, my dear Sir,
Very faithfully yours,
(Signed) WILLIAM FOX, A.M.,
Minister of Christ Church.

D. Blair, Esq., M.D.,
Surgeon General.

No. 10.

Georgetown,
14th November, 1850.

My dear Sir,

In consequence of the non-contagion of our late yellow fever epidemic having been questioned in high quarters at home, it becomes necessary even at this late period to obtain some further evidence on the subject; and from the many opportunities of observing, and from the many personal exposures which your sacred calling entails on you, I hope you will excuse this reference to you, and be good enough to favour me with answers to the following questions. Did you ever suffer by communication with those sick of yellow fever? Did you ever know or hear of a case which was imputed to contagion? When Mr. Austin was sick of the epidemic, did it appear to spread to any person in the house, to any visitor, to any nurse or washerwoman? With many apologies for the trouble I put you to in replying to the above queries,

I remain,
Very truly and respectfully yours,
(Signed) DANIEL BLAIR.

The Venerable Archdeacon Lugar.

The Parsonage, Georgetown,
14th November 1850.

My dear Sir,

With respect to the several questions contained in the above communication, I have no hesitation whatever in answering them by a

decided negative. Should you require this reply in any more formal manner, I shall be happy to give it you.

My dear Sir,
Yours very faithfully,
(Signed) JAMES LUGAR, A.M.

Daniel Blair, Esq., M. D.

No. 11.

6th November, 1850.

DEAR SIR,

IN answer to your note this morning, I beg leave to inform you that no other person in my family had yellow fever in 1837 but Dr. Fraser.

Dear Sir,
Yours very truly,
(Signed) M. FRASER.

Dr. Blair,
Surgeon-General, &c., Georgetown.

No. 12.

Prov. N. Chapel, Georgetown,
14th November, 1850.

DEAR SIR,

IN answer to your inquiry per note just received, I beg leave to state, that while I thought it always prudent, personally, to use every necessary precaution against contagion during the prevalence of the epidemic yellow fever, I remember but one instance in which the opinion of the disease being "possessed of contagious or infectious qualities" appeared to be confirmed; and even that seemed to me as likely to have arisen from predisposing circumstances as from infection. The case was that of the widow of her deceased husband, who, after his death and burial, gave herself up to excessive grief, and would not leave the very part of the bed on which he died. It was in the beginning of the epidemic in 1837, that the late Rev. John Wray of Berbice came to Demerara, and caught the infection by walking through Water Street; but his infection was not by contact with the diseased, but by the inhalation of the miasma occasioned by the rubbish used in the filling up or repairs of a certain part of the

street; for it was from that moment he began to complain, and believed himself to be ill with yellow fever, of which he died. It was thought that his son-in-law had caught the infection from him, as he died only a few days afterwards; but if it had been infectious, I should have expected that the other members of the family would have suffered, only one of whom was laid aside, but recovered. I went to Berbice on the occasion of their death, and after great exhaustion from excessive efforts in endeavouring to improve the death of my ministerial brethren at distant places, was myself taken dangerously ill of the same disease of which they died, though I never entertained the idea that I had caught the infection from visiting their abodes; — from personal contact I could not, as both were interred before my arrival at Berbice. As to "opinions," I have heard them variously expressed for and against, but without being satisfied on the point, I have ever considered it prudent to exercise due caution.*

I remain, my dear Sir,
Very respectfully yours,
(Signed) JOSEPH KITLEY.

Daniel Blair, Esq., M. D.,
Col. Surgeon General.

No. 13.

Tweedside,
20th November, 1850.

I HAVE delayed somewhat in answering your letter (31st Oct.), wishing not to depend entirely on my own conviction in regard to the two questions you wish for information on. From my own recollection, as well as from that of others, there was no yellow fever in Barbadoes in the year 1837. In 1836, from the month of June or July to December, we were visited by such an epidemic, but I cannot learn that a single case occurred in the year 1837; and I have never

* In my letters of inquiry to the clergy, I gave the utmost latitude for the most discursive reply, inviting not only their own opinions and observations, but also what they had heard expressed by others. I had no previous communication with the clergy on the subject; and I have taken their reply to my inquiries without re-examination or suggesting any correction. You will observe that Mr. Kitley revives an old rumour of one of the Berbice missionaries' wives having been affected by yellow fever from contagion. This subject, however, was long settled, and the report shown to be erroneous, by the testimony of the family medical practitioner, Dr. Hollingsworth. (See his replies to my inquiries.)

heard that it was even suspected at any period to have been brought from Surinam; and I am borne out also on this latter point by every one with whom I have conversed on the subject since receiving your letter.*

No. 14.

Examination of the Newspapers.—"Royal Gazette," 1837.

1st June.—A letter, signed "Medicus," appeared, in which the causes of the "dreadful fever which is carrying on such destruction in the town" are discussed; and therein no mention is made of importation, but local causes, such as filling up the old Dutch canals in the streets, and the entrance of miasmata from the interior by the lately made Lamaha canal, alone are referred to, and suggestions that the Board of Police should open the street canals.

12th June.—Mr. Cherrington died.

26th June.—"Humanity" recommends the burning of tar-barrels to remove the epidemic.

12th July.—"W." asks for public prayers to avert the pestilence, which is seconded by an editorial article in which the visitation is imputed to sabbath-breaking.

12th July (same date).—"A husband and a father" calls on those medical men who treated the disease successfully to make public their method, *pro bono publico.*

26th July.—Charles M^cDougall died.

1st August.—Rev. Charles Rattray's wife died.

15th August.—"A mortal" calls on a commission of chemists to analyse the filthy trenches and the dreadful "low water stench at the Stelling." The editor recommends the communication, and remarks on the stench around the news-room, which he calls "a cave of Avernus."

5th August.—"W. H." writes a letter; states that the disease prevalent *is not yellow fever, as* "IT IS NOT INFECTIOUS;" and recommends tartar emetic.

29th August.—The Rev. Mr. Bryer died.

5th September.—"Civis" disapproves of the site of the temporary

* This statement is in accordance with the return from official documents inserted by me, p. 46.—ED.

hospital for seamen as unhealthy. Says that the medical men with whom he has spoken consider the disease "only ordinary colony fever" under an unfavourable atmosphere.

4th January, 1838.—The editor of the "Royal Gazette," in his retrospect of the year, refers to the epidemic yellow fever as extinct.

19th April.—Dr. Hackett, staff surgeon, writes to the governor about the wretched state of the barracks; and the editor, in a comment, calls on the authorities to look after the drains, &c., as we are now approaching the time of year when the yellow fever broke out.

17th September.—A public meeting was held in the Court of Justice, his Honour the high sheriff in the chair, to consider the propriety of establishing a seaman's hospital. An editorial article appeared on the 15th recommending the object. The public meeting was attended by all the influential merchants of Water Street, many ship-masters, &c., four of the city medical men. Committees were appointed; correspondence with the governor and Court of Policy took place. At an adjourned public meeting a report was adopted which the colonial government acted on, and established the present seaman's hospital. An angry discussion between two or three parties continued to occupy the newspapers for some time after the first meeting. One party suggested a floating hospital; but that was overruled, as, unless in a very inconvenient situation, the hospital would be moored in the disease-bringing locality. Another party wished the colonial hospital to be enlarged for the admission of seamen; but this was also overruled, as it *would be exposing the seamen to the contagious diseases of the colony;* and, to *prevent any such risk*, a separate establishment was almost unanimously determined on.

22nd November.—The letter of Dr. Fraser having become known in the colony, Dr. Hackett, staff surgeon, sent as an antidote and reply a paper of Dr. Ferguson on yellow fever.

3rd August.—There appeared in the "Guiana Chronicle" a letter, signed Thomas K. M'Lennan, M. R. C. S. L., on the yellow fever. After stating the imputed local origin of other epidemics of that disease, he remarks, "Now it is from sources of the same nature as some of those described above, that the fatal fever of last year, which is not yet extinct in this town and harbour, is propagated." "The bad drainage of the town; the full and dirty small drains and privies, situated in improper and disgraceful places, which in case of heavy rains are but very partially swept down to the front of Water Street, where a horrid and infectious swamp is formed all the way from Dr. Rodie's Lots in Cumingsburgh to M'Inroy, Landbach, and Co.'s (with the exception of the improvements made by Messrs. Glen and Co. and their spirited neighbours in their division of the town), together

with the filling up of so many mud lots and stellings with all kinds of decomposing materials;—the combination of these create sufficient material to account for the sickness we have experienced." Not a word is said of contagion or of importation.

26*th November*.—A letter signed "W." appeared in the "Guiana Chronicle," referring to the now discovered letter of Dr. Fraser and Sir A. Halliday, insinuating that Dr. Fraser obtained the office of health officer through Sir A. H., and asserting that they shared the emoluments, which of course would be largely increased by the indiscriminate quarantine which the two recommend.

21*st December*. — Appeared, in same paper, an editorial article, acknowledging the receipt of a communication from "W." of thirteen pages of foolscap, but which he, the editor, does not publish, as four of the pages consist of vituperation against Dr. Fraser. The object of "W.'s" letter was to refute the statements of Dr. F. in his letter to Sir A. H. The editor publishes the letter of Dr. F. as it appeared in the "London Medical Gazette"—its first publication in the colony; and after stating, "We can only express our surprise that all our medical practitioners in this colony should so long have been uninformed of the existence of such a letter, and should betray such indifference on the subject after its publication in so generally circulated a periodical, and under the sanction of so respectable a medical recommendation as that of Sir Andrew Halliday," he goes on to make his own non-professional observations on it. He asserts, but in friendly tone, that Dr. F.'s description is overdrawn; that the epidemic, instead of causing so much sickness, alarm, and destruction of human life *throughout* the colony, as represented by Dr. F., was only *partial*, and the representation of Dr. F. was "greatly exaggerated;" that the statement now made, of the disease having prevailed in Surinam and Barbadoes prior to our epidemic, and that it found its way from thence to this colony, should be carefully inquired into. The editor proceeds: "These statements, we are told, have decided the scepticism of Dr. Fraser as to the contagious nature of yellow fever. We own that further evidence as to the correctness of these statements will be required before we shall feel induced to change our opinion, founded on eighteen years' experience and observation, tending to an opposite conclusion." . . . "That the mortality among our shipping, and with new comers, especially in Water Street, was very great, will not be denied; but the subsequent statement, that its pestiferous force from thence emanated, and spread its infection to every quarter and corner of the colony, we wish to see disproved by higher authority than our mere assertion." . . . "The assertion that seven or eight funerals formed the daily average of this awful and

calamitous period of the destroying angel's visitation, would be best answered by a publication from authority of the obituary of the time. We have seen it, and venture to say that it will not support this highly-wrought picture, although occasionally sufficiently melancholy. We believe we may assert that the daily average of deaths was one and a fraction." . . . "The remainder of this long section, where it is stated that no shade or caste of colour made the least difference; that it at last carried off its victims from every description of the human race inhabiting this province, will probably surprise our readers, who, we venture to say, have scarcely heard of any instance of death by yellow fever except among the shipping or new comers from Europe residing in this town, chiefly along the river side." . . . "In fact, we have not heard of any estate in the colony losing a single negro by yellow fever for many years past." . . . "We shall not proceed any further in our notices on this letter, as it is less our object fully to reply to it ourselves than to induce others better qualified so to do."

The editor concludes by, "We think it especially behoves the medical faculty of this colony to animadvert on this letter of one of their body, unless they wish to have it understood that they sanction all the statements and theories it contains."

22*d December*.—There appears in the "Royal Gazette" a short letter, signed "W.," complaining of the injustice of the editor of the "Chronicle" in suppressing his communication regarding Dr. Fraser's letter, although he criticises that of "W." It is probable that "W." is the *nom de guerre* of Dr. Bascome.

1839 contains no communication on the subject of the epidemic, but many attacks on the mode of conducting the New Seaman's Hospital; all, however, successfully repelled.

No. 15.

List of Physicians, Surgeons, and Licensed Practitioners in British Guiana. — From the "Local Guide," 1842.

Georgetown. — J. H. Alleyne, Joseph Bankhead (Col. Hospital), E. Bascome, D. Blair, G. R. Bonyun, F. Clancey, C. Clifton, H. G. Dalton (Seaman's Hospital), J. M. Fraser Dodgson, J. R. F. Hutson, J. M. Johnstone, H. A. Kock, E. J. M'Nish, E. A. Manget, Sir M. M'Turk, J. H. Pollard, T. Pollard, M. C. Van Well, H. Hutson.

East Coast. — J. Scott, J. Merry, N. Martin, David Spence, Thomas Reid.
Mahaica. — T. M'Creath.
Mahaicony. — T. K. M'Lennan, T. Gilbert, sen.
East Bank, River Demerara. — — Houston, Wm. Bruce, Hon. W. Rankin, W. M'William.
West Bank. — S. S. Forte, A. S. M'Farlane, J. G. Clifton, — Black.
Hobaboe Creek. — H. Rodie.
West Coast. — P. F. Watt, J. Paton, J. M'Lagan.
Leguan. — E. G. Boughton, W. Miller.
Wakenaam. — H. A. Dobridge, P. Croal.
West Bank, Essequibo River. — —Cooper, W. C. Stack, H. S. Vandamzorg.
Arabian Coast. — James Fraser, E. Cullen, D. Brydges.
Pomeroon. — W. Paton, T. Gilbert, jun.
Penal Settlement. — S. Cramer.
Berbice. — J. J. Dare, J. B. Beresford, John M'Kenzie, W. H. Tucker, E. P. Cameron, John Cameron, John Fairman, R. Smith Dupré, J. C. Jamieson, W. Richardson, A. R. Hollingsworth, jun.

No. 16.

Canal, No. 1.
29th November, 1850.

Dear Sir,

I duly received yours of the 14th instant; and I will now supply, so far as I can, the information required on the subject referred to.* I believe the disease of which Mrs. Rattray died was yellow fever. . . . I have no reason whatever to believe that the disease was communicated to her through any afflicted person or by contact with any infected thing. I am not aware that any other case of yellow fever, or supposed yellow fever, occurred in this district in 1837 except one, that of an overseer, a *new comer*, who died on the second day of Mrs. R.'s illness: between him and her there could have been no possible communication; and neither myself, nor children, nor any person employed about the house, nor any visitor, ever had any thing like yellow fever at the time of Mrs. R.'s illness or after her death. I had just completed my third year in the colony; and when she was taken ill, I was suffering from the *first* attack of intermittent fever that ever I had. . . . . Our house had been finished in May;

it was then painted; and we went to live in it in the early part of June. The weather was close and sultry, and wet, though the rains were not very heavy. Our new house was situated on the south-west corner of an abandoned coffee estate, where two acres of land had about seven months before been cleared, leaving the high bush on the east and north sides. By the time that our house was finished a thick crop of wild starch had grown up; and when we came to reside there it was cut down. The new house recently painted, in short not yet dry — the mass of recently-cut vegetable matter lying around during the close weather, with now and then a few hours of very hot sunshine — the high bush so close to our residence on the windward — may have combined to make our situation there unhealthy at that particular time.

I remain, dear Sir,
Yours very truly,
(Signed) CHARLES RATTRAY.

Daniel Blair, M. D.,
Col. Surgeon General.

P. S. I recollect the Rev. Mr. Murkland had the yellow fever in 1837. He was taken ill in town, and lay at Mr. Ketley's. He was very ill, but recovered. I know Mrs. Murkland had no symptoms of it after waiting on him; and I never heard of any of Mrs. K.'s family having any such symptoms. Dr. Clifton attended him.

C. R.

No. 17.

Names of the several resident surgeons, dispensers, and stewards who served, varying from a period of six months to two years and upwards, in the Seaman's and the Colonial Hospitals, during the yellow fever epidemic.

Drs. Johnston, Rowe, Bonyun, Koch, Dalton, and Bankhead.
Dispensers. — Harris, Hessener, Driver, and Allen. Mr. Bill, who died the same night he was gazetted to the office, never served.
Stewards. — Gillis, Myers, Jones, Burke, Stewart, Wicham, Rice, King.

No. 18.

Names of Board of Directors of Colonial and Seaman's Hospital, extracted from "Local Guide," 1843.

COLONIAL HOSPITAL.

President. — Honourable U. J. F. Bach.

Members.—Venerable Archdeacon Lugar, Rev. Jas. Struthers, D.D. (the minister of the Dutch Reformed Church), Rev. E. Macnamara, Colin Simson, Thos. Naghtan, Adam Vinfhin, Peter Miller Watson, John Cooper, and Alex. Wishart. W. E. Peirce, Secretary.

SEAMAN'S HOSPITAL.

Sir Michael M'Turk, Hon. H. E. F. Young, William Davison, William Brand, George Blacklock, Thomas Kirkpatrick, P. Gilbert, Geo. H. Loxdale, J. C. Preston, Westaway Howes, and Alex. Duff.

Note. — None of the above named gentlemen suffered from an attack of yellow fever.

Extract from the Hospital Rules and Regulations.

"Two of the directors shall visit the hospital at least twice every week, in such order and rotation as may be from time to time appointed, and shall investigate any complaints that patients may have to make regarding their treatment in hospital."

No. 19.

Copy of tariff of fees paid by the schooner "John," of about 30 tons, every time she cleared from this harbour, in her trade with Surinam, contained in letter of complaint from Captain Forbes in the "Guiana Chronicle," 1st March, 1836.

	Guilders.	Stivers.	Charges in Surinam.
"Harbour-master's fee -	f 15		f 3
Boat with health officer -	6		f 3
Health officer - - -	12		f 3
Bill of health - - -	11		f 3
Receiver's office - - -	20	10	
Gov. sec. office - - -	20		f 5
High sheriff's office - -	12		
Pilot's office - - -	22		
	f 118	10	

Note. — The health officer is paid extra for visits when a vessel is in quarantine.

No. 20.

Certificate of parties put in quarantine on the return trip of the schooner "John," 19th March, 1836. Quarantine imposed for three days, viz, till date of certificate.

"We, the undersigned, passengers on board the schooner 'John' from Surinam, do declare that there was no contagious disease prevailing in that colony, and that the colony is generally healthy.

(Signed) "HUGH M'LEOD.
"M. G. VANDERPANT.
"WIDOW N. RIGUBOUT."

Quarantine Grounds,
21st March, 1836.

No. 21.

Minute of financial accountant, showing daily average number of NURSES (exclusive of cooks, &c.) in attendance in Colonial and Seaman's Hospitals, for 1842.

Office of Financial Accounts,
23rd December, 1850.

SIR,

IN accordance with your request for information as to what was the average number of nurses employed in the Seaman's and Colonial Hospitals during the year 1842, I have made up a statement from the pay-lists of both hospitals, and the result you will find at the foot of this communication.

I have the honour to be, Sir,
Your most obedient servant,
(Signed) W. B. POLLARD, A.F.A.

To Daniel Blair, M. D.,
Colonial Surgeon General.

Average daily number of nurses in the Seaman's Hospital during 1842, seven (7). Average daily number of nurses in the Colonial Hospital during 1842, fifteen (15).

W. B. POLLARD, A.F.A.

Note. — As the search involved considerable trouble and time, I did not ask from Mr. Pollard more than the average of one year, viz. 1842, the year of the second culmination of the epidemic.

THE END.

LONDON:
SPOTTISWOODES and SHAW,
New-street-Square.

SOME ACCOUNT

OF THE

YELLOW FEVER EPIDEMY

BY WHICH

BRAZIL WAS INVADED IN THE LATTER PART OF THE YEAR 1849.

BY J. O. M'WILLIAM, M.D., F.R.S., R.N.
SURGEON TO THE HONOURABLE THE BOARD OF CUSTOMS.

[*Extracted from the* "MEDICAL TIMES" *of April 19th and 26th.*]

LONDON:
PRINTED BY WILLIAM TYLER, BOLT-COURT.

1851.

Dr Carpenter
with the compliments
of the Author.

Seems impossible to overrate the importance of this Paper. Feb 1849 Y. F. had never appeared in Brazil though such position & all the circumstances would have rendered it infinitely probable that it would have been prevalent there at all times.

PREFACE.

In drawing up the following Paper for the Epidemiological Society, my object was to eliminate from the voluminous documents to which I had access, just as much of their substance as was required for a brief, but fair and impartial account of the Yellow Fever Epidemy which has lately invaded Brazil.

And although the paper is now relieved (so to speak) from the compression to which it was subjected, in order that the time occupied in reading it should not exceed one hour, I have not considered it as yet necessary to extend the subject beyond the addition of some new facts which have been recently supplied me by Dr. Alexander Paterson, of Bahia, and Dr. May, of Pernambuco.

With these exceptions the paper is, in every respect, the same as when I had the honour of submitting it to the President and Members of the Society.

J. O. M'W.

14, *Trinity-square.*
April 28th, 1851.

SOME ACCOUNT

OF THE

YELLOW FEVER EPIDEMY IN BRAZIL.

Brazil occupies a great portion of South America, extending from 5° north to 32° 30 south latitude, and from 35° to 70° west longitude.

Viewed upon the map, Uruguay, La Plata, Bolivia, Peru, Columbia, and the other countries of this great continent, seem to form but a margin of variable width to the southern, western, and northern limits of the Brazilian territory.

The central and eastern portions of Brazil are occupied by high table lands, traversed by serrated mountain ranges. The lowlands to the northward comprehend the great plain of the Amazon, and the flat lands extending from it far to the eastward; a country among the richest in the world, and containing immense tracts of impenetrable forest. The lowlands to the southward include the greater part of that country lying south of a line drawn from eastward in a north-western direction, in the parallel of 29° south. Here there is a vast extent of pasture land, cut up in all directions by rivers, lakes, and marshes, and abounding with herds of cattle and horses.

The lowlands on the eastern side or seaboard of Brazil, vary in extent from 120 to 15 or 20 miles from the sea.

Following the course of its various indentations, the coast itself comprehends a distance of 4000 miles, which may be thus rapidly described. From its southern extremity, as far as 29° south, it is low and sandy; it then becomes rocky as far as the bay "Espirito Santo" in 20½° south; and from this to Bahia the coast is again low and level. Proceeding northward, and then bending to the westward, the coast is first of moderate height and then slopes away, low and marshy, as far as the Amazon.

The climate is cool and temperate between 28° and 33° south, and moderate within the southern tropic even as far as 15° south.

At Rio de Janeiro, in about 23° south, the medium heat is 74°. In summer, that is to say, during December, January, and February, the average heat at noon is 86° Fahr., the minimum 72°. During winter, or the months of June, July, and August, the temperature at noon is 72°, minimum 59°. There are heavy falls of rain, with thunder

and lightning, during the summer. As we proceed towards Bahia and Pernambuco, and further northward, the temperature increases; but, as these ports are more open to the winds, the heat at them is said to be less oppressive than at Rio de Janeiro. The seasons are of course earlier, and less distinctly marked, proportionately to their proximity to the equator.

Throughout the whole line of coast, the climate is of the most beautiful description, its general tranquillity being seldom disturbed by any more violent winds than the trades, or the daily land and sea breezes.

Mr. Cowper, the Consul at Pernambuco, a gentleman of great observation, who was not only present, but most actively and usefully engaged, during the prevalence of the recent yellow fever epidemy in that Province, says:—"The climate of the whole Brazilian tropic is moderate and varies very little. At Para, upon the line, the thermometer scarcely ever changes. During the day, in summer, it invariably stands at 86° Fahr., and during the night it falls, with the land breeze, to 65°, and sometimes to 60° Fahr. In the winter, the temperature during the day is 84° Fahr., at night 72°, owing to the absence of the land breeze in winter. This state of climate will apply to the whole coast, as far as Cape St. Roque, (lat. 5° south.) From thence to the southward of Bahia (lat. 13° south) the temperature is about two degrees lower; and Rio de Janeiro, although far south, (lat. 22°53 south,) owing to its geographical position, is said to be equally warm."

Notwithstanding the geographical position of Brazil, its great variety of climate, and its abounding in those elements which theoretically are supposed to induce the more aggravated forms of tropical disease, endemic disease, except in a mild form, is little known; from sweeping epidemic disease, of any kind, with the exception of small-pox—introduced by slavers(a)—the country has, until lately, been generally considered as wholly exempt.

It was, therefore, with astonishment not unmixed with doubt, that information of the existence of yellow fever in the city of Bahia, in the month of November, 1849, was received in the other parts of the empire; and it was not until the disease had extended to Pernambuco and Rio de Janeiro, that the people in general could be led to believe that so formidable an invader had arrived among them.

(a) Mr. Cowper doubts that the introduction of small-pox is due to the slavers, for from the earliest times the Indians are said to have suffered from this disease. Mr. Cowper informs me of the curious fact, that hydrophobia was unknown in Brazil until within the last few years; but that it is now as common and as fatal as in Europe, and is attributed by the Brazilians to the importation of foreign dogs.

I shall now endeavour to trace the origin and progress of the yellow fever epidemy of Brazil, commencing with Bahia, and then taking up the other ports of the empire in the order, so far as my information enables me, according to which they were respectively invaded by the disease, prefacing the account of the fever at each place by some brief topographical remarks.

Bahia.—The town is situated in lat. 13° south, within the north-eastern boundary of the magnificent Bay of All Saints. The shore rises high, and is richly wooded. The lower town, the resort of the shipping and the great mart of trade, consists of long narrow streets, filthy, and badly paved. The upper town rises abruptly behind, is beautifully situated, and contains some fine buildings and private residences.

The population of Bahia is about 140,000; of whom one-third are whites; the same number are mulattoes, and the rest negroes.

The rocks belong to the granitic series. Darwin, in his "Narrative of the Voyages of the Adventure and Beagle," says, "throughout the coast of Brazil, and certainly for a considerable space inland, from the Rio Plata to Cape St. Roque, lat. 5° south, a distance of 2000 geographical miles, wherever solid rock appears, it belongs to the granitic formation."

I may also mention, that at a point near the city Darwin observed an appearance in the granitic rocks which has been discussed by Humboldt, who states that "at the cataracts of the great rivers Oroonoko, Nile, and Congo, the syenitic rocks are coated by a black substance, and look as polished with plumbago." It is a curious fact, that the Indians, and even the Missionaries of the Oroonoko, connect the unhealthiness of the villages round the cataracts with those rocks which they describe as carbonized or burned by the rays of the sun. They carefully avoid sleeping near these black rocks, which they imagine are fertile sources of the "calentura" or fever. Their insalubrity is so proverbial, that villages are not built in such localities, if space elsewhere can be found.

The insalubrity of Hong-kong has been by some connected with the granite rocks which abound in that colony.

Bahia had for several months enjoyed an immunity from all diseases except those common ailments incident to every such community, when, on the 30th September, 1849, the American brig Brazil arrived at that port, her papers said from New Orleans, but, as was afterwards ascertained, actually last from the Havannah. She had lost two men on the passage from fever, attended with black vomit; but, as their deaths were not reported to the authorities, the vessel was not placed in quarantine. The circumstance of admit-

ting to immediate pratique a vessel from New Orleans, the very focus, as it was said, of yellow fever, while quarantine was being rigorously enforced upon all vessels coming from European ports where cholera prevailed, was much discussed by the people in the town, and even became the subject of comment in the public prints.

I have already said that the Brazil arrived on the 30th of September. It is of importance also to state, that we have no account of any case of yellow fever occurring, either in the town or in the harbour, earlier than the 3rd of November. On that day, Dr. Paterson, one of the chief medical men of the place, was called to his first two cases of yellow fever. "One of them was a Brazilian boy, living over the American store, frequented by the captain of the Brazil. He had been ill," says the Doctor, "for some days prior to my visit, and on the afternoon of the 3rd he had black vomit, and died within a few hours." On the same day, Dr. Paterson was also called to an Englishman who had long resided in the country, and who had had frequent communication with the captain of the Brazil. During the convalescence of this, the second patient, fell ill the third case,—a young man, recently from Europe, living in the same house, and still more intimate with the captain. At the end of nine days he died of black vomit. Dr. Paterson adds: "One of the first fatal cases, and immediately succeeding those of my own, was the American Consul, also not long in the Brazils, and who constantly associated with the captain of the Brazil, and with him frequented the house of my second and third patients." He continues: "I could thus go on through a considerable number of cases, in the early part of the epidemy, tracing out the transmission of the disease from one person to another; as the number of foci of infection increases, the task would of course become the more difficult."

I have just invited attention to the importance of there being no account of any case of yellow fever on shore before that of the Brazilian boy, seen by Dr. John Paterson on the third of November. But since this paper was written, I have heard from Dr. Paterson's brother, Dr. Alexander Paterson, surgeon of the British Hospital, Bahia, and now in this country, that the case of the Brazilian boy, although his brother's first case, was not the first case in the town. Dr. Alexander Paterson "ascertained beyond doubt" that the Brazil took in several Spanish passengers at the Havannah, which, on landing, took up their abode chiefly in that part of the town of Bahia called "Santa Barbara;" and that in this very district yellow fever broke out within three weeks after the Brazil's arrival: but its occurrence was

carefully and, for a long time, successfully concealed by parties connected with the slave-trade.

Up to the 18th of November, no recorded case had occurred afloat. In a day or two, however, two deaths were reported in a Swedish brig, which was the next vessel to the Brazil, and, being consigned to the same house, had frequent intercourse with that ship, And two other deaths, as it afterwards appeared, had already occurred on board the Swede without any medical man having been called. In a few days, every man on board the Swedish vessel had caught the disease, and the greater number of them died. It was not until the 3rd of December that there was any case among the English shipping. From this time, however, there was scarcely a vessel in port that escaped. Once the disease had established itself on board of a vessel, says Dr. Paterson, "it was the rarest thing possible for the fever again to quit it until every one capable of catching it had caught it." Meanwhile the disease had extended over the town and immediate suburbs, and ceased not to rage until it had "burnt itself out for want of fuel." "It spread," says Dr. Paterson, "to the suburbs and the adjoining country in radiating lines, in a direct ratio with the amount of traffic in any given line." By the end of February, it had attacked not less than 96 per cent. of the inhabitants. Out of 700 seamen (English) in port, between the 1st December, 1849, and the 28th February, 1850, 223 were attacked, and 72 died. Among the native Brazilians, "and those long resident in a tropical climate, the disease was comparatively mild; not fatal, certainly, in 1 per cent. Among the African blacks it was still milder. The native coloured population suffered as numerously, but, perhaps, less severely than the whites." In those recently arrived from extra-tropical climates, the fever assumed a more intense and malignant character, the mortality amongst such having exceeded 25 per cent.

Pernambuco, in 8° south, which was invaded by yellow fever next after Bahia, is built upon a low, swampy land, surrounded by an undulating range of hills, it may be 200 feet above the level of the sea. On one extremity of this range, and five miles to the northward of the city, the old town of Olinda is situated. The city is divided into the three districts of Recife, of San Antonio, and of Boa Vista, which are connected with each other by wooden bridges. Boa Vista, where the British Hospital is situated, is the cleanest and airiest part of the town, and that furthest removed from the harbour, near to which the streets are narrow, rather filthy, and in bad order. As the river is influenced by the tide higher up than the town, the water there is unfit for use, but a good supply of wholesome water is obtained

rom the Rio da Prata, about two leagues from the city. The banks of the river are studded with the mangrove. A secure natural harbour is formed by a reef of hard sandstone, which runs parallel to, and not far distant from, the shore.

The city contains 120,000 persons, viz.:—

1. Whites, native	34,000	
,, foreign	6,000	
		40,000
2. Blacks, native	30,000	
,, African	20,000	
		50,000
3. Mulattoes		30,000
		120,000

On the 17th day of December, 1849, the French bark Alcyon arrived at Pernambuco from Bahia, having lost two men on the passage from yellow fever. (a) Notwithstanding this she was admitted to pratique, and anchored among the other vessels in the harbour. Some fresh cases of fever occurring on board, they were sent to the French hospital (situated in San Antonio;) but one of them having terminated fatally on the 19th, the authorities ordered the rest to be re-embarked, and interdicted all communication with the Alcyon. The Consul, writing on the 21st December, says, "No cases have as yet to my knowledge occurred within the city. The authorities have ordered all vessels from Bahia to be placed under quarantine for eleven days." In a postscript to the same letter, he adds, "Two cases have since been reported on shore." (b) On the 25th December, Dr. Paton, an English medical man, young, healthy, and robust, and but a short time in the country, was attacked with fever in the British Hospital at Boa Vista, and died on the 27th. The immediate cause of death was at first supposed to have been epilepsy. He laboured for some days under low fever, and was found upon the floor of the room, and almost instantly died, the body turning bright yellow.

(a) Dr. Paterson, of Bahia, states, that four men were sent on shore ill with fever just before the Alcyon sailed from that port, although she was granted a clean bill of health. A correspondent at Bahia, writing on Jan. 29, 1850, says, "Accounts have been received, that the epidemic has made its appearance at Rio and Pernambuco, which is not surprising, as the authorities here, until now, denied its contagiousness, and continued to grant clean bills of health to all vessels."

(b) Dr. May, from whom I have heard since this paper was read at the Epidemiological Society, says, "One or two cases appeared in the town, and were speedily fatal, but nothing was said about them, in order not to create alarm. In the last days of December a seaman came into the British hospital with broken ribs from a Brazilian steamer, in which he had arrived direct from Bahia, where he had been on shore for some time. A few days after the admission of this man Dr. Paton was attacked. The seaman had no fever on admission, but he was emaciated and sallow."

On the day of Dr. Paton's death, another gentleman, the house apothecary, Mr. Pitt, also residing at the British Hospital, was seized with fever, and died on the fourth day with suppression of urine and black vomit. At this time there were only three other patients in the hospital—one with phthisis, one with ulcer, and the other with paronychia. On the evening of the day on which Mr. Pitt was attacked, the seaman with paronychia, was seized with similar symptoms. Next morning his countenance was anxious, and his extremities were cold, and he died on the following day with black vomit. Dr. May and his housekeeper, who attended Mr. Pitt, were next attacked. Both recovered, but the housekeeper's escape was a narrow one. During Dr. May's illness, the seaman with ulcer was taken ill, and, although sedulously attended by a friend, he also died. After this, all Dr. May's black servants in the house were attacked, but none of them died.

The man with phthisis escaped.

Dr. May and Dr. Arckbuckle, the two chief practitioners of the place, both declared that Mr. Pitt died of yellow fever; but it was resolved, at a consultation held by the chief men in the profession, with the view of preventing alarm, to conceal the existence of the disease in the city.

The Consul, writing at this time, says, "It is hoped that the general purity and breadth of the streets, intersected as they are in all directions by the rivers, the perpetual sea breeze, and the clearness of the atmosphere, will at once check the disease, although for the present the city may be looked upon as threatened by invasion from without, and by pestilence within."(a)

The disease, however, radiated in all directions from the Boa Vista Hospital, so that, by the 14th of January, most of the inhabitants in that part of the city had been attacked; and its course, say the best authorities, could be traced step by step, from one district to another, all over the city, and even throughout the province.

So early as the 6th of January, the disease had spread to the shipping anchored in the harbour near the Alcyon, proving extremely fatal to those newly arrived in the country.

On board a Prussian vessel lying in the same tier with the Alcyon, a man was attacked shortly after the Alcyon's arrival with excruciating pains in the loins and legs, and died on the third day with black vomit. Although the Prussian vessel was quite new and remarkably clean, the captain had her fumigated, and used chloride of lime daily. Still in a day or two after another man was attacked, and he also

(a) Civil war was raging at this time.

died in a few days with black vomit. Others followed in rapid succession, until this vessel had lost eight men out of a crew of twelve.

The next vessel in which the fever broke out was an English barque, called the Esther Anne, a perfect model of cleanliness. She was also in the same tier with the Alcyon, and she lost her mate and four or five of her crew all from yellow fever. During the whole of this time, not a single case occurred in any other ship in the port, except among those in the same tier with the Alcyon.

The fever gradually extended from ship to ship, until all, with very few exceptions, were infected.

One English ship, the Columbus, anchored very high up the harbour, far from every other vessel, escaped entirely.

Another vessel, laden with guano, escaped for a long time, and the captain attributed his good fortune to the nature of his cargo. A day or two, however, before this vessel left Pernambuco, one of her sailors went on a visit to an infected ship. He was soon seized with fever and died, as did also the mate who attended him. Hundreds of similar instances might be narrated.

By the 20th of February, not less than 30,000 persons had been attacked on shore by the fever, and it had also extended to English, Portuguese, and Imperial men-of-war in the harbour. On board the Imperial corvette there were twenty cases of fever. At this time the business of many of the public departments could scarcely be carried on, from the number of *employés* affected.

The weather was at this time very hot. "On the 27th," says the Consul, "the sun will be vertical, and it is to be hoped that shortly afterwards we may have a change for the better."

These hopes were not, however, realised, for early in March the fever had increased in intensity, "no longer confining itself to persons newly arrived in the country, but proving fatal to natives as well as foreigners. By this time eleven of the British community, and probably two thousand residents, had perished. The Consul had already established an hospital on one of the islands in the bay, to which, during the month of February, seventy seamen, American, Hamburghian, and Swedish, were admitted, of whom forty-one died. Of forty-one British seamen, twenty-seven died.

This increased mortality of the disease was considered the more remarkable, as the Consul at this period observes, "the arrangements are all improved, the weather latterly uncommonly cool for the season; the medical men have increased experience, and the people invariably adopt habits of precaution; but nevertheless the plague increases both on board and on shore."

Nothing could be better than the judicious arrangements suggested by Mr. Cowper, the English Consul, for the accommodation of the sick. Finding that the hospital was crowded, he, with the permission of the President of the Province, caused tents to be erected on Cocoa Nut Island, and afterwards had the sick treated on board their own ships, lying on deck, under awnings, exposed to a free circulation of air.

Mr. Cowper, to whom I am indebted for so much valuable information regarding this epidemy, thus classifies those that were attacked by fever at Pernambuco:—"1. New comers; 2. Seamen; 3. Residents of less than two years, not perfectly acclimated; 4. Natives of the first class from the southern provinces and from the interior, including North and South Americans, and even Brazilians. Of the second class, during the first month, the deaths were 1 in 3; during the second month, 1 in 2; during the third month, 2 in 3; during the fourth month, 4 in 5; and the mortality increased in proportion as the number attacked decreased. Of the third class very few escaped attack, and certainly not more than half death. Of the fourth class scarcely one escaped attack, but the disease was in a mild form; whilst nearly 100 per cent. of the two first classes, and 50 per cent. of the third, fell victims, not more than 3 per cent. of this class died."

Without reference to race, 16 per cent. of all foreigners attacked died, and 3 per cent. only of the natives.

The Sardinians, from some unknown cause, suffered more severely, both afloat and on shore, than other foreigners. According to Dr. May, the mortality afloat was 60 per cent. of those attacked.

Rio de Janeiro, the capital of Brazil, lies in 22 53 south latitude, and 43°12 west longitude, on the western side of a magnificent bay, not less than sixty miles in circumference, containing numerous islands, and surrounded by a scenery of surpassing beauty. Dr. Lallemant, one of the reporters on the yellow fever of Brazil, considers this bay to represent a "microscopic daguerreotype" of the Gulf of Mexico. "At Rio," he says, "we have a regular but almost land-locked bay, little subject to the influence of the ebb and flow of the tide, and into which a series of rivers discharge themselves, at whose marshy mouths, Paullinieæ and Avicennieæ, and the wondrous forms of the Rhizoporeæ determine the zone of vegetation."

The city is built on an undulating plain, which is said to have been a swamp, and is scarcely elevated above the sea level. There still exists a considerable marsh in the immediate vicinity of the city, to the westward. The salt water passes freely through a small creek, and mingles with the

waters of this marsh. Although this source of malarial exhalation is, according to Dr. Leonard, surgeon of Melville Hospital, Chatham, not less than fifteen hundred paces long, and nearly five hundred paces wide, the inhabitants of its neighbourhood are said to be not more liable to fever than those in other parts of the city.

The mountains which encircle Rio consist of granitic rocks, and the soil is composed of disintegrated granite, with a variety of clays in beds of considerable depth.

The city itself occupies about three miles along the shore. There are some fine squares, but the streets in general are long and narrow. Sewerage and public cleanliness are matters of little concern. There are a few drains, but they seem to be little attended to, and, from the flat situation of the city, there is no run of water through them. The town is well supplied with good fresh water. The large aqueduct which conveys it from the Corcovado Mountain, is one of the most remarkable buildings in the city.

Situated just within the southern tropic, and environed by hills which partially intercept both the sea and the land breezes, Rio possesses an excessively sultry atmosphere, especially when the sun is south of the equator. From November to April, (the most sultry period,) a good deal of rain falls; the air is generally moist, and the heat is oppressive. From May until October the weather is drier.

In 1847, according to Dr. Leonard's observations, the thermometrical indications were as follow :—

	Maximum.	Medium.	Minimum.
August	78°	69°	61° Fahr.
September	81°	75°	69°
October	83°	72°	68°
November	83°	74°	69°
December	86°	77°	71°

The population may be estimated at about 100,000 whites, and 200,000 negroes and mulattoes.

The information regarding the invasion of Rio de Janeiro by yellow fever, and the subsequent course of that disease through the city and other parts of the province, is both voluminous and clear. The documents to which I have had access consist of :—

1. A Full Account of the Rise and Progress of the Epidemy at Rio de Janeiro, embracing the Histories of the first twenty Cases on Shore. By Dr. Lallemant, a German Physician in practice at Rio.

2. Replies to Queries sent to Rio de Janeiro. By Sir William Pym, Superintendent-General of Quarantine; with Observations by Mr. Lee, Surgeon, Rio de Janeiro.

3. Replies to Queries, with Observations; and a small

Pamphlet on the Fever. By Dr. Croker Pennell, Rio de Janeiro.

4. Replies to Queries; with a Description of the Yellow Fever, and Remarks on its Successful Treatment on the Homœopathic Principle. By Mr. Cockran, a surgeon and homœopathic practitioner at Rio de Janeiro.

Besides these, there are several highly important official papers.

By the concurrent testimony of all these authorities, Rio de Janeiro was considered to be in a state of general good health prior to the latter end of December, 1849.

Intelligence that a malignant fever was raging at Bahia reached Rio by the Portuguese steam-vessel, Don Alphonso, on the 13th December, 1849. On the following day, the Portuguese ship Don João arrived from Bahia, five of her crew having sickened, and two having died, during the passage. This vessel was accordingly placed in quarantine. The English packet Petrel, also from Bahia, arrived on the 23rd or 24th December,(a) with two cases of yellow fever on board—a passenger and the steward of the vessel. One of them died on the 25th, and the other on the 27th, while the vessel was in the harbour of Rio.(b)

The first known case of yellow fever in the city of Rio de Janeiro was seen on the 28th December;(c) on which day, says Dr. Lallemant, "On entering Fergusson Ward in the large city hospital, 'Misericordia,' to which I have been physician for seven years, I found two new patients whose appearance greatly struck me, and who presented a complication of morbid phenomena, especially a 'yellow colour of the skin, black vomitings, suppression of urine, hæmorrhage from the mouth and bowels;' and, in the case of one of them, great disturbance in the mental faculties, which led me to suppose something peculiar in these cases."

The cases were—

1. Enquist, from Finland; who had arrived about a fortnight before, direct from Russia, by the Russian brig Volga, and had been ill some days.

2. T. Andersson, a native of Sweden, who had arrived as sailor on board the barque Navarre, and had also been ill some days.

(a) Dr. Lee, December 23rd, 1849.

(b) They had both been on shore at Bahia, and had visited a house where a person was lying ill of yellow fever, and who afterwards died.—Dr. Lee's "Replies to Queries."

(c) Dr. Lee; "The first case of yellow fever in Rio was at the Brazilian Hospital, on December 28th."

Dr. Croker Pennell; "The first known case of yellow fever occurred in the instance of a seaman, who was attacked and taken to the public hospital on December 28th."

The patient No. 1 died the following night. No. 2 also died after forty hours, and on the 30th December.

Keeping these, the first known cases of yellow fever in the city of Rio de Janeiro,(a) stedfastly in view, it now becomes necessary to bring into prominent light the proceedings of the barque Navarre and her crew, already alluded to, during the months of November and December, as this vessel occupies an important place in the history of the epidemy.

The Navarre sailed from Bahia, in ballast, on the 24th November, 1849, and arrived at Rio de Janeiro on the 3rd December.(b) As we have already seen, yellow fever was raging at Bahia at the time the Navarre left that port. The Navarre's crew were, to appearance, in good health; the vessel was sold, and the men were paid off. Some of them went into other vessels, and were no more heard of; others went on shore at Rio, and took up their abode in the Rua da Misericordia, at a lodging-house kept by a man named Frank. The sleeping apartment on the ground-floor of this house was at this time very dirty, and many sailors were congregated in it. Close to Frank's house, on the opposite side of the way, were two other lodging-houses, the one kept by an Englishman named Wood, and the other by a Frenchman called Auguste Hourde. The lodgers of these houses very frequently visited each other.

I have already said, that some of the sailors of the Navarre went to lodge with Frank. When Dr. Lallemant visited Frank's house early in January, in consequence of the arrival of three fresh yellow fever cases from it into the Misericordia Hospital, he then learnt that Andersen, the subject of case No. 2, and Josiah Baker, another seaman of the Navarre, by this time also in the hospital, were the first that sickened at Frank's house; and that Enquist, the subject of Case No. 1, had lodged in an apartment behind Frank's house, where he was a frequent visiter. Dr. Lallemant says, with reference to these cases,

(a) These were the first cases *in the city*. Dr. Lallemant, however, observes: "When, on the 11th of January, 1850, at a sitting of the Medical Academy of this place, I submitted the cases of yellow fever which had come under my own observation, Dr. Noronha Feitel stated a case which had occurred at the Naval Hospital on the 29th December. On that day, a man who had come from Bahia in the steamer Dom Pedro, was admitted into the Naval Hospital on the Ilha das Cobras; when he died soon after of black vomit and diarrhœa. Now, although this case occurred almost close to the city, no further consequences ensued; nor did the physician of the hospital attach any importance to it, for he did not speak of it until a fortnight afterwards, in consequence of my statement at the Academy; and even then he merely mentioned it casually. So that it loses all importance which might otherwise attach to it."

(b) Dr. Lee and Dr. Pennell, 2nd December, 1849. Mr. Cockran, end of November or beginning of December.

"Enquist was admitted on the same day as Andersson, and I am perfectly satisfied he caught the fever by frequenting Frank's house."

By the 10th of January ten cases of yellow fever, of which seven proved fatal, had been admitted into the Misericordia Hospital; all of them were either from the lodging-house of Frank or Wood. One man, who refused to go into the hospital, was treated at Frank's house.

On the 10th of January, Robert Luff, an Englishman, was admitted to the hospital with yellow fever. He had been frequently under Dr. Lallemant's care for wounds in his legs. He was a drunkard and a vagabond, and had been lodging at Wood's, where he sickened. He died in forty-eight hours. About the same time another Englishman, called Thomas Fox, also from Wood's, came into the hospital with symptoms of yellow fever, but he recovered.

Dr. Lallemant's attention being thus called to Wood's house, he proceeded thither, and found Wood himself, his wife, and a German waiter, all suffering from yellow fever in a comparatively mild form.

Auguste Hourde, the landlord of the French lodging-house, had meanwhile been admitted into the hospital on the 3rd of January. He left it on the 5th; but, suffering a relapse, he returned, and died of malignant yellow fever after five days. Hourde's wife had also a slight attack on the 17th January, but she recovered.

By the 19th of January, Dr. Lallemant had seen and treated twenty cases of yellow fever, all them belonging to the houses of Frank, of Wood, or of Hourde. Of these, fourteen were admitted to the hospital in a hopeless state, and ten died.

The histories of the whole of these cases are detailed with great clearness and circumstantiality by Dr. Lallemant. That they were the first cases of yellow fever on shore in Rio de Janeiro, there seems no reason to doubt; for, besides Dr. Lallemand, Dr. Croker Pennell, Dr. Lee, and others, distinctly refer the outbreak of the disease in the city to the lodging-house in the "Rua da Misericordia."

The patient Enquist, whose name stands first in the hospital list, had frequently visited the Russian and Finland ships in the harbour; in fact, he sickened while actually on board one of them. Suddenly, a captain, a steersman, and a sailor, died from an acute attack of yellow fever on board the Finland vessels Niord and Norna, on the 9th January, 1850; and, on the following morning, several patients from these vessels, and from the Swedish ship Scandia, were received into the Misericordia Hospital.

The yellow fever consequently prevailed in the harbour so early as the 9th of January.

With reference to the appearance of the fever on board another vessel, Dr. Lallemant has the following remarks :—

"In the Rua da Misericordia, between the lodging houses of Hourde and Wood, separated from each by a few intervening dwellings, and exactly opposite Frank's, (the street being about twenty-four feet wide,) is the mercantile house of a German coffee merchant, whose eldest daughter had arrived from Hamburg in the ship Maria Christina, in the middle of December, bringing with her a strong healthy girl. This girl went frequently to the merchant's house, was taken ill on the 8th of January, and died on the 13th, at the house in the Rua da Misericordia, of fully developed yellow fever."

The sailors of the Maria Christina frequented the merchant's house, and in the course of a few days the fever broke out on board that vessel. The crew of the now infected ship purchased their fresh meat at a Danish butcher's in the Praga da Don Manoel. The apprentice, a German, who sold them the meat, was seized with a violent attack of yellow fever on the 8th; but he recovered in three weeks.

In consequence of the great influx of patients into the hospital from the vessels, on or about the 18th January Dr. Lallemant was ordered to take all his yellow fever patients in the hospital to an uninhabited convent on the "Ilha da Bon Jesus," distant about six miles from the city. It was not until now that Dr. Lallemant's diagnosis, given on the 30th of December, and hitherto severely combated, that the disease was yellow fever, was considered incontestably true.

"From this period," says Dr. Lallemant, "it was impossible for me to take cognizance of all the cases of yellow fever that occurred. The disease fixed itself in the Rua da Misericordia, proceeding from the house above mentioned, running almost without interruption from house to house, though with milder symptoms, and even passed into the narrow or cross streets, until that entire quarter had sickened, while, during that and the following days, not a single suspicious case occurred in any other part of the city."

Towards the end of January the disease seems to have manifested itself in many streets along the river, running down to the banks, and at length surrounded the whole city.

The account of the rise and spread of the fever at Rio de Janeiro, given by Dr. Lee, Dr. Croker Pennell, and other reporters, differs in no important particular from the elaborate report of Dr. Lallemant. Dr. Pennell says, in his pamphlet, that the disease for the first fortnight was confined to the Rua da Misericordia; and that the locality next

infected was the "Saude," situated at the opposite end of the city, about a mile and a half distant from the Rua da Misericordia. This district is only a little elevated from the high-water level, with a clayey soil interposed with open gutters, affording a ready lodgment to stagnant water.

According to Dr. Lee, also, the first cases appeared in a sailors' lodging-house, in the "Rua da Misericordia," and then spread along the shore, "attacking vast numbers who inhabited the 'Prainha' and 'Sande,' both localities immediately contiguous to the loading-ground of the merchant shipping. From these spots it quickly spread over the city, attacking all classes."

"In the harbour," continues Dr. Lallemant, "the disease did not take any sudden leaps, but went slowly from ship to ship. There then were lying alongside each other, the Norna, Niord, Scandia, Alfhild, Elizabeth, Maria, Helsinfors, Louisa, Vestalinden, Adam, Brave, Frode, and also another Niord, all carrying different amicable flags, so that the captains and crews visited each other; whereas the American and English vessels, which had less intercourse with the above-named ships, were not attacked by the fever till afterwards."

Subsequently the crews of the Brazilian men-of-war were affected by the fever; and now the far-spreading epidemic raged in and around Rio de Janeiro and in the harbour. "Thus," continues Dr. Lallemant, "the yellow fever was brought into the 'Rua da Misericordia' by the crew of the American vessel coming from Bahia. Here it propagated itself, and was from thence carried into the harbour. And we see the first cases developing themselves, linked together as unquestionably as the closely united links of an iron chain."

While the fever was raging along the flat coasts of Rio, the mountain districts of Petropolis, Nieufreiburg, and Constantia continued perfectly healthy. Accordingly, in February and March, the coast, and numbers of the residents of the city, and of the newly arrived Europeans, fled for safety to those elevated regions. Several were attacked within three or four days after their arrival in the mountains, and some died; but in no case was the disease communicated to the mountain residents. If, however, the German or other colonists of Petropolis, or of any of the other mountain districts ventured down to the flats of Rio, they were almost sure to contract the fever.

This is in perfect accordance with what Humboldt states regarding yellow fever in Mexico. The great "Oracle of Nature" says, "The whites and the Mestizos who inhabit the interior table-land of Mexico, of which the mean temperature is 60° and 62° Fahr., and where the thermometer

sometimes falls below the freezing point, are more liable to contract the vomito, when they descend to the Plan del Rio, and from thence to la Antigua and the Port of Vera Cruz, than the Europeans or inhabitants of the United States who come by sea." The farm of L'Encero, which Humboldt found to be 3043 feet above the level of the sea, is the highest limit of the vomito. "Here the inferior limit of the oaks warns the colonist who inhabits the central table-land, how far he may descend towards the coast without dread of the mortal disease of the vomito."—*Political Essay*, &c. Vol. II. P. 251.

Petropolis, according to Dr. Croker Pennell, is situated at a height of nearly three thousand feet above the level of the sea.(a) In the month of February, (summer,) the thermometer ranges, from the hottest hour of the day to the coldest hour at night, from about 68° to 80° Fahr. In March, its range is from 62° to 75° Fahr.; in April, from 56° to 72° Fahr. In the coldest night in the depth of winter, (July,) the thermometer occasionally falls as low as 36° Fahr.

The thermometric maxima and minima are here too loosely stated to warrant the application of any of the usual methods of educing from them the true mean temperature with rigorous accuracy; still I think it may be safely inferred, that the mean temperature of the hottest month mentioned, barely reaches the point at which, according to Humboldt and others, yellow fever can be maintained, at all events for any length of time; and, moreover, the indispensable condition of a certain duration of high temperature is wanting. Dr. Croker Pennell states in his report, that the absence of infectious property in the disease at Petropolis cannot be explained by the difference of temperature; for the temperature of the atmosphere at Petropolis was higher at the time the fever patients were there than it was in Rio de Janeiro, in July, 1850, where there were still some bad cases of yellow fever. Dr. Pennell has here overlooked the necessity for a continuance of high temperature to keep the disease alive; for instance, May is a warmer month at Vera Cruz than September or October; but it is in the two last months that the "vomito" commits the greatest ravages, showing that a certain time is required for the germ of the disease to be developed in full energy. At Vera Cruz the yellow fever does not appear before the medium heat is 75° Fahr., and it never passes the limit of an elevation of 3000 feet above the sea level. As was the case at Petropolis, people frequently carry the disease from the flats near Vera Cruz to the town of Jalapa, situated at a height of 4335 feet, on one of the terraces which occur in the steep ascent to the

(a) According to Dr. Lallemant, about 2000 feet.

high table-lands of Mexico; but they have never been known to communicate it to others there.

I have not been able to ascertain with any degree of accuracy the number of deaths from the epidemy at Rio Janeiro and the other provinces of the empire; but it is probable that at Rio Janeiro alone not less than 10,000 or 12,000 persons perished.

At Rio Janeiro, and indeed everywhere else, the disease dealt its fury upon the natives of extra-tropical climates, and more especially upon new comers from the more northern latitudes. Hence, says Dr. Lallemant, "Norwegians, Swedes, and Finlanders, constituted the melancholy trio which furnished the greatest number of victims. The crews of many Swedish and Finland vessels died almost systematically one after the other, and almost invariably after suffering from three to five days." The men of the most colossal stature died most rapidly. Of 299 northern patients treated at the hospital of Bon Jesus, 154 died, and only 145 recovered.

The mortality among the newly-arrived Portuguese was also very remarkable. "In the literal sense of the word, whole families were swept off by this fever." Next to the Portuguese, the Italians suffered most. Of the company composing the Italian opera, seventeen died; as did also nearly every member of an equestrian company. For a long time not a single image-vender, rag-merchant, or umbrella-seller, (who are almost without exception Italians,) was to be seen in the streets of Rio. In many instances, half the passengers who arrived by vessels from Havre de Grace,—nay, sometimes even three-fourths of them, died within three weeks after their arrival.

The three foreign embassies of Paris, Rome, and Washington, each lost its Secretary of Legation.

Dr. Lallemant found the greatest powers of resistance among the English and Americans, and the least among those before named, viz., the Finlanders, Swedes, and Norwegians; and next to these the Italian seamen from the west coast of Italy.

Having thus given some account of the epidemy in the chief ports of Brazil, it now becomes necessary, in order to clear the ground for the more important points of the inquiry, to determine—

1st. The state of health in Brazil prior to the invasion of that country by the recent epidemy; and

2nd. The precise nature of the epidemy, and whether it manifested itself everywhere in Brazil as the same disease.

The general salubrity of the whole coast of Brazil, south of the equator, in spite of the presence of those conditions of soil and of atmosphere usually considered favourable to the production of disease, has been so remarkable as to

cause men engaged in climatorial inquiries to pause before they adopted the commonly-recognised theories as to the origin of fevers and those other forms of disease which are so rife within and in the neighbourhood of the tropics.

And certainly it was not easy for an inquisitive mind hastily to admit the cause-and-effect relation of marshes and rich rank soils, acted upon by heat, and other meteoric agencies, with the worst form of fevers, when it was seen that so great an extent of territory as Brazil, situated in chief part within the tropics, and abounding with every supposed pathogenetic influence, with a population containing Europeans, the mixed races, and negroes, enjoying a high and singularly uniform degree of health.

Dr. John Wilson, in his Statistical Reports on the Health of the Navy, inquires, "Why is it that, in a land-locked harbour in this part of the world, under a powerful sun, surrounded by marshes and rich vegetation, ships lie for months or years without a single case of concentrated fever; while in Asia, in Africa, in North America, and more especially in the West Indian Islands, things, which to superficial observers appear the same, are productive of so much disease and death?"

Dr. Bryson, the successor of Dr. Wilson in the office of medical statistics at the Admiralty, also observes with reference to these shores:—

"Irrespective of geographical position and the almost universal presence of terrestrial sources whence fevers and agues are supposed to arise, they are, from the equator to Cape Horn, regions of surpassing beauty and salubrity."

Examining the whole of the evidence furnished by the various reporters on the late epidemy, it does not appear that, throughout the whole line of coast, there was, with the exception of Rio Janeiro (where, according to Dr. Lallemand, there were gastric and other affections) the slightest deviation from the usual high standard of health, previous to the outbreak of the fever at Bahia in the latter part of the year 1849.(a)

Dr. Lallemant, in his able Report, states that, at Rio Janeiro of late years, during the hot season, thunder-storms,

(a) It may be proper to mention that at Rio de Janeiro, at Pernambuco, at Bahia, and probably all along the Coast of Brazil, the inhabitants had, during each summer of four years preceding the late epidemy, suffered from a mild form of fever, which in no case proved fatal; and was therefore jocularly called the "polka," from the fact that its appearance in Brazil was nearly coincident with the introduction of the well-known dance of that name into that country. No one was twice attacked by the "polka;" but the fact of having had it gave no protection against an attack of its fatal successor, the yellow fever. I recollect that somewhere about the year 1830 a mild fever of this kind ran through nearly the whole of the West India Islands. Like the polka in Brazil, this fever obtained a whimsical appellation, "The Dandy Fever."

which had the effect of clearing the atmosphere, and heavy rains which cleansed the streets, are now much less common than they were some years back; and that four years since, and during the two following hot seasons, the city was visited by a disease, *sui generis*, called by him "insolation fever," and by others the "polka," which he considered to resemble the first stage or stadium of yellow fever, but never developed any of the grave symptoms, such as black vomitings, suppression of urine, and yellowness of skin, which characterized the late epidemy. Dr. Lallemant also says that the summer of 1849 was indisputably hotter than during former years, and that, although there was a good deal of lightning, there was little thunder or rain, and that gastric fevers were common. All the other medical men who replied to the question, "Was Rio healthy before the epidemy began?" distinctly declare that "the city was remarkably healthy." The same may be said of Bahia, Pernambuco, Para, and other ports from which information has been obtained. Mr. Cowper, the Consul at Pernambuco, informed me that the population, before the appearance of the fever, was very healthy, that the temperature of the atmosphere was 2° less than during some seasons, and 3° lower than it was two years ago.

I think, then, we have reason to believe that there was nothing remarkable in the weather along the Coast of Brazil during the summer of 1849, and that the inhabitants generally were in good health before the outbreak of the recent epidemy.

Regarding the nature of the epidemy: time will not allow me to go over the history of symptoms described by each individual reporter; but no one who will take the trouble to read those accounts, can for a moment doubt that the disease was yellow fever. The red, bleary eye, the black vomitings, the yellowness of the skin, the suppression of urine, and the rapid progress of the disease, sufficiently determine its nature.(a)

It may then be allowed, that the epidemy by which various provinces of Brazil were invaded during the latter part of the year 1849, was yellow fever.

The next and most important point to be ascertained is, whether this epidemy was of endemial origin, or a foreign introduction.

Ferreyra da Rosa, a physician, of Olinda, in Brazil, describes a disease similar to yellow fever as having prevailed there in 1687, immediately after the conquest of Pernambuco. It was at this time that the ship Oriflamme, on her

(a) Suppression of urine was a very usual symptom during the yellow-fever epidemy at Boa Vista in the Cape de Verde during the years 1843-46. Louis, in his account of the yellow-fever epidemy at Gibraltar in 1828, speaks of suppression of urine as only an occasional and rare symptom.

voyage from Siam to the West Indies, touched at Olinda, and, as it is said, became there infected with yellow fever, which she afterwards introduced into the island of Martinique, where Père Labat, on his landing, found the disease committing great ravages. Sir Gilbert Blane says that the yellow fever was "introduced into Lisbon in 1723, probably from Brazil." Dr. Gomes dos Santos, a physician still at Rio de Janeiro, states, that a fever, attended with "yellowness of skin and vomitings," but altogether of a milder form than the late disease, prevailed in that city during the years 1808-1809, but that it excited no interest beyond the limits of the province, Europe being at that time too much occupied with political events to take any notice of a fever in Brazil.

Attaching to these statements all the importance that belongs to them, we must, I think, admit, that nowhere in Brazil has there existed, within the memory of man, an epidemy similar to that which has recently ravaged that country.(b)

Yellow fever, from time immemorial, has occasionally raged in the adjoining countries, the Guyanas,(c) and in the West Indian Islands; but in Brazil, until 1849, the equator had always been considered the barrier against the progress of this pestilence southward.

In 1849, however, the yellow fever passed this boundary, and, according to all accounts, Bahia was the place where it first appeared. The situation and some of the leading features of Bahia have been already alluded to, as well as the immunity of that city, in common with the whole of the Brazilian coast, from disease, previous to the arrival of a vessel infected with yellow fever. And it now remains for me to inquire how far the supposition of the disease having been imported into Bahia is borne out by the circumstances of the case.

Here I must observe, that, besides the Brazil, another vessel, called the Alcyon, (by which the yellow fever is supposed to have been afterwards introduced into Pernambuco,) a French slaver, has been blamed for carrying the

(b) Mr. Cowper informed me that some old inhabitants of the province of Pernambuco affirm, that it was visited by a fatal epidemy in 1790, and for some years previous to that year.

There was also a report at Rio de Janeiro that a fever prevailed there at the close of the last century. Nothing certain concerning either of these epidemics has been obtained, either from oral tradition or literary sources. Garcia, in his "Memorias Historicas de Pernambuco," mentions an epidemic disease as having prevailed at Pernambuco between 1712 and 1728.

(c) Yellow fever prevails at the present time in French Guiana, the country adjoining Brazil. It is worthy of remark, that its first victims were among those of the higher ranks. The Governor, the Bishop, and the President of the Court of Appeal all died in the early period of the epidemy.

yellow fever to Bahia. Dr. Paterson, however, whose statements are altogether more circumstantial than any of the others I have seen, speaks only of the Brazil. Therefore I am inclined to think that, if the origin of the disease at Bahia is to be ascribed to importation by a vessel, that vessel was the "Brazil."

In the examination of this question I may be allowed to adopt the principle that guided me while investigating the Boa Vista epidemy, in which I considered that, although much light may be thrown upon the nature of the disorder during its ulterior progress, it is to the first cases of the epidemy that we look for the commencing link in the chain of that process by which we may trace out its true history, and ascertain the properties with which it is endowed.

Two deaths had already occurred on board the Brazil before her arrival at Bahia;(a) and from the concealment of them, as well as of two others that afterwards took place on board, it is probable she had the disease on board at the time of her arrival, on the 30th September, 1849. No case, however, was seen until the 3rd November, or before 34 days after the "Brazil's" arrival.

The first case, a Brazilian boy, who lived over a store frequented by the captain of the Brazil,(b) had been ill for several days, and, as death with black vomit ensued on the afternoon of the day on which he was first seen (3rd November,) the probability is, he had been laid up for four, five, or it may have been seven or nine days, thus reducing the interval between the arrival of the vessel and the occurrence of the first case to 30, 29, 27, or even 25 days. The second case, an Englishman, long resident in the country, who had been often visited at his own house by the captain of the Brazil, was also seen by Dr. Paterson on the 3rd November. The Englishman recovered; but, during his convalescence, a young man who lived with him was seized with the disease, and died on the ninth day with black vomit.

The occurrence of the first case being, at the lowest reckoning, 25 days after the arrival of the vessel suspected of having brought the disease, would leave a long period of inactivity on the part of the disease, between the ship and the shore, were we obliged to believe that the fever poison, if imbibed at all from the captain, could only have been imbibed on the first day. But we are by no means driven to adopt such a supposition. The captain, for a consider-

(a) The Brazil arrived on the 30th of September, having landed a cargo of slaves at Havannah. "There is no doubt of some of the crew having died on the passage."—Consul Porter's Letter.

(b) The first victim was in the house frequented by the captain of the Brazil, and among the first was the American Consul, who, it is believed, was on board the Brazil. Four or five others, living in the same hotel with the American Consul, died among the first.—Consul Porter's Letter.

able time after the arrival of the vessel,—in fact, until the death of the boy,—had been in the daily habit of visiting the American store and the house of the Englishman;(c) therefore, supposing that the boy and the Englishman were infected by the captain, infection may have taken place after an interval sufficiently long from the arrival of the vessel to bring the incubative period within a very reasonable limit.

But then it nowhere appears that the captain was at all sick; therefore, if he infected the boy and the Englishman, he must have done so through the medium of "fomites," a means of communicating disease the existence of which many will be disposed to question, even more than that which is said to result from contact with a sick person, and concerning which all must admit our knowledge is still in a very imperfect state. The information from Dr. Alexander Paterson, which, as has been already mentioned, was not received until some time after this paper was written, renders the introduction of the fever into Bahia by the Brazil even still more probable. Dr. Paterson, to use his own words, "on the strongest possible evidence," ascertained that the disease first appeared in that part of the city (Santa Barbara) where the passenger by the Brazil went to lodge, and within three weeks after that vessel's arrival.

But, independently of all reasoning regarding the period of incubation, or the exact period of the outbreak of the epidemy, I would ask the candid and unprejudiced, whether it be not a remarkable fact that Bahia was the port of Brazil first invaded by yellow fever, and that at that port a vessel had a short time before arrived, which, if not actually at the time suffering, had suffered during the passage from this terrible scourge?

If, as some allege, the yellow fever of Brazil was due to an endemial cause, it must I think be admitted, as indeed singular, that throughout this vast empire, comprehending a territory at least twelve times as large as the whole of France, the very first spot that cause should select for the development of its influence was that at which a vessel infected with the same disease had just before arrived.

That the outbreak of the fever at Bahia, and the arrival of a vessel infected with the same disease were mere coincident events, is certainly possible. But those who contend for such a coincidence must allow that it was, under all circumstances, one of a most extraordinary nature.

At Pernambuco the first case occurred in the cleanest and

(c) The captain of the Brazil slept in the Englishman's house, and the American Consul, whose house he frequented on duty, were among the first victims.—Speech of the President of the Province, at the opening of the Provincial Assembly at Bahia, 1st March, 1850.

airiest part of the town, and that furthest removed from the harbour, within a week after the arrival of a vessel infected with yellow fever. Some cases had been received into the French hospital (San Antonio) from this vessel. But there is no evidence to show that Dr. Paton, the subject of the first case, was ever near them or the infected ship.(a) The next case was Dr. May's apothecary, who also lived in the British hospital (Boa Vista.) From this centre the disease radiated, first over the whole city and afterwards throughout the entire province. Mr. Cowper, the Consul, informed me that during the progress of the epidemy many positive instances of contagious propagation came under his own notice, of which the following are examples:—

In the early part of the epidemy a wagoner was sent into the city from Engenhio Caruana, an estate five leagues in the country, belonging to Dr. Domingos de Souza. Having finished his business in the city, where he remained many hours, the wagoner sickened on his way home, and shortly after his arrival there showed unequivocal symptoms of yellow fever, of which he soon died. Those who attended him were next attacked, and the disease speedily spread over the whole estate. So great was its mortality, that Dr. Domingos was obliged to beg aid from the neighbouring estates, which was refused, upon the ground that the appearance of the disease on his own estate afforded so palpable an evidence of contagion. Mr. Cowper is certain there was no disease on the estate before the return of the sick wagoner from the city.

Mr. Cowper furnishes another case of this kind. He says: "When all communication with the Alcyon was forbidden, M. Lassere, her consignee, offered this measure all the opposition in his power. To show his contempt for infection, he went on board the Alcyon daily for several days until he was laid up with fever, and died upon the third day. His death created the greatest panic on shore. Up to this period, the fatal cases had been confined to seamen and new comers; but M. Lassere was a man of robust constitution, had been resident in the country twenty-five years, and his wealth enabled him to obtain the first medical aid; notwithstanding all this he died, and from that moment no one considered himself safe. He died at his country house in the village, or rather district, of Capunga, where my cottage adjoined

(a) In a former note, written after this paper was read at the Epidemiological Society, I have said that the only known connexion between Dr. Paton and an infected place, was a seaman from Bahia, who entered the British Hospital with fractured ribs. I learn from Dr. May, now in this country, that, before Dr. Paton was attacked, one or two other cases had occurred in the town immediately after the arrival of the Alcyon; but that their occurrence was kept secret. Dr. May says, from this circumstance he could not discover whether they could be traced to contact with the Alcyon or her crew, although this very probably was the case, as they lived in that part of the town much frequented by seamen.

his place. Capunga had been perfectly healthy up to this period; but, within a week, twenty cases of fever, and five deaths, had occurred."

At Rio de Janeiro a considerable time elapsed between the arrival of the vessel alleged to have introduced the yellow fever, and the appearance of that disease on shore.

Dr. Lallemand infers from the disproportionately small number of her crew (nine) for a bark, and from the suspiciously hurried manner in which the captain disposed of his vessel, that she had already before arriving at Rio lost some men from fever. But in a case of this kind mere conjectural evidence cannot be taken. Granting that there were suspicious circumstances attending the arrival and selling off of the vessel, there is no proof that her crew were at all sick at that period, and no one of them was found sick earlier than the 28th of December, 1849. It is true, the man Andersson, one of the Navarre's sailors, had been laid up some days at Frank's house, before his admission into the "Rua da Misericordia" Hospital. Now, making the same allowance for the probable period he was ill at Frank's house, as was made in the cases at Bahia; and deducting accordingly four, five, seven, or nine days from the time of the Navarre's sailing from Bahia, and the same number of days from that of her arrival at Rio de Janeiro, the interval between the outbreak of the disease among her crew, and those periods respectively, will be reduced to twenty-nine, twenty-eight, twenty-six, or twenty-four days; and twenty-one, twenty, eighteen, or sixteen days.

Dr. Croker Pennell states that there were many cases where no source of infection could be traced. He also says that several masters of vessels declared to him that, though coming direct from Europe, fever made its appearance on board of their ships as soon as they approached the coast of Brazil, and that, from the accounts the masters gave of the disease, it appeared to be the same as that prevailing at Rio, but slighter. The same was said regarding vessels arriving at Bahia; but Dr. Paterson soon found out that these cases were merely from attacks of the mildest nature, and not yellow fever.

The argument derived from the long absence of yellow fever from Brazil may alike be taken hold of, although differently applied, by those who favour and those who oppose the doctrine of the contagious nature of this disease.

The former may, with propriety, ask, How is it that, for nearly a century, yellow fever has not appeared in Brazil, if the recent epidemy depended upon endemial causes, seeing that, at the period in question, there was no meteorological or other influence likely to induce any unusual operation of such causes?

The latter may, with equal reason, inquire, why, if the yellow fever was imported into Brazil, was it not introduced until lately, seeing that there has been, for a long period, constant and unrestricted intercourse between the ports of that country and the coast of Africa, the Havana, New Orleans, and other places notorious for the prevalence of yellow fever?

As it would be difficult, if not impossible, in the present state of our knowledge, to answer the question of either party, the value to each of any argument deduced from the long immunity of Brazil from yellow fever, is thus, to a certain extent, neutralized; nevertheless, I think, it must be owned, that the general healthy condition of that country immediately before the outbreak of the late epidemy, is a fact of considerable importance in the present investigation, inasmuch as it has enabled us to determine with accuracy the periods at which the various ports were invaded by the fever, and, moreover, will assist us materially in endeavouring to ascertain why some intermediate as well as distant ports continued wholly exempt.

The following Table shows, at a glance, the chief ports that were invaded by the fever, and some of those that escaped, during the epidemy of 1849-50.

Place.	Latitude.	Longitude.	When Fever Appeared.	Remarks.
Bahia	13° S.	38° 42' W.	Nov. 3, 1849.	Ship "Brazil" arrived 30 Sep., 1849, sickly, from Havana.
Pernambuco ...	8° S.	34° 50' W.	Dec. 25, 1849.	Ship "Aleyon" arrived from Bahia, having been sickly on the voyage, 17th Dec. 1849.
Rio de Janeiro	22° 53' S.	43° 12' W.	Dec. 29th, Island in Bay, Dec. 31st, City.	Ship "Navarre," Dec. 3, 1849, arrived.
Paraiba	... ...	... ...	January, 1850.	... ...
Para	1° 18' S.	... ...	March, 1850.	... ...
Porto Allegre, Rio Grande do Sul.	30° S.	... ...	April, 1850.	One case only in the whole province.
Maranham ...	2° 30' S.	43° 50' W.	Escaped.	Rigorous Quarantine.
Ciara	Between 3rd and 4th degree South Latitude.	... ...	Do.	Little or no communication with infected ports.
Aracati ...		... ...	Do.	Do.
Parnahyba ...		... ...	Do.	Do.
Monte Video ...	34° 55' S.	56° 10' W.	Escaped.	Quarantine.
Buenos Ayres in Rio de la Plata.	34° 37' S.	58°16'11" W.	Do.	Do.

Thus it is evident that the Brazil epidemy first broke out at Bahia, next at Pernambuco and Rio de Janeiro, the ports having the greatest intercourse with it, and afterwards at the other ports not in the order of proximity; but in the order of the frequency of their communication with an infected place.

There was fever at "Maceio," a port between three and four degrees to the northward of Bahia, before it was in any of the intermediate towns along the coast; and at Pernambuco, five degrees to the northward of Bahia, before it was at any of the many intervening towns. Passing Maceio, it broke out at Pernambuco, and was thence imported into Maceio by an infected vessel from Pernambuco.(a) It was at Para in 1° 18 south, in March, 1850; but all the while the ports of Maranham, of Parnahyba, of Aracati, and of Ciara, all lying between the second and fourth degree of south latitude, continued free from the disease. The escape of Maranham is ascribed to the rigorous quarantine enforced there upon all vessels from infected ports; that of the other three places just mentioned, to the almost total absence of any communication with those places where fever existed.

In the same manner the yellow fever was at Rio de Janeiro, ten degrees to the southward of Bahia, before it was anywhere, even within 100 miles, in the same direction. At Port Alegre, in Rio Grande do Sul, about eight degrees south from Rio Janeiro, there was one case in April. Monte Video and Buenos Ayres, in the Rio de la Plata, each enforced strict quarantine; and, although both were visited by infected vessels, they entirely escaped.

Had the disease depended upon a general morbific agency in the atmosphere, its spread certainly could not have been so obedient to a law regulated by the greater or lesser intercourse of places with infected ports; nor could its visitations along a line of coast of more than 4000 miles have been so partial, irregular, and capricious as they seem to have been. As little can the presence or absence of endemial localizing influences explain the invasion of one place by the fever, or the immunity of another. Witness the opposite results at Para and at Maranham ports, nearly similar in regard of climate and general physical condition.

Again, places very dissimilar as to situation, soil, and

(a) The late Dr. M'Hardy, of Maceio, thus wrote to Dr. A. Paterson:—
"The English ship Emperor arrived from Bahia on the 23rd December, with two of her crew convalescent, two having died in your hospital and another here, on the 27th, with black vomit; but I believe the disease was communicated here, not by her, but by a passenger who came from Bahia in a steamer, about the 25th, lived on shore in the house of a Portuguese merchant, where I was called to attend him the night he arrived. Some days after, a clerk in the same house was taken ill; and one of the partners died with black vomit, on the 12th Jan., 1850. All these parties had their meals in a room where the passenger slept on a sofa."

climate, equally continued free from the disease. Monte Video and Buenos Ayres, both lying beyond the southern extremity of Brazil, with a moderate climate, the one standing on a promontory on the northern bank, and the other on the southern bank of the river Plate, escaped throughout; but so did also Maranham, burning under an average annual mean temperature of 80° Fahrenheit, and within two degrees of the Equator.

I consider, then, that the whole tenor of the evidence adduced bears out the following conclusions:—

1. That the Brazilian yellow fever epidemy did not arise from endemial causes.

a. Because, notwithstanding the presence of supposed endemial causes, yellow fever has been for nearly a century unknown in that country.

b. Because places where endemial influences were abundant escaped altogether.

2. That the epidemy did not depend upon any general morbific agency, either stagnant in or travelling through the atmosphere.

a. Because while some places were affected by the fever, others near to them, and under the same conditions of soil and climate, remained free.

b. Because the disease sometimes followed the direction of the south-east trade wind, and at other times travelled in the teeth of that wind.

3. That the evidence of the importation of yellow fever into Bahia, Pernambuco, and Rio de Janeiro, although not of so absolute and positive a nature as there was in the case of Boa Vista, in the Cape de Verds, is still sufficiently presumptive to warrant the belief that that disease was a foreign introduction into these and other ports of Brazil.

a. Because each of these ports was healthy before the arrival of a vessel infected by yellow fever.

b. Because, after the appearance of the disease on shore, its general spread was in accordance with the law of contagious diseases, and because positive evidence of contagious propagation was manifested in particular cases.

c. Because ports, having little or no communication with infected places, and ports at which quarantine measures were adopted, entirely escaped the disease.

Lastly. Yellow fever, unlike cholera and typhus fever, which may prevail at ordinary and even at low temperatures, requires a high temperature, and that of some duration, for its maintenance.(a) It is even driven from the flats on the

(a) Rush is the only author, so far as I am aware, who considers that yellow fever can be maintained under a low temperature. Sir Gilbert Blane was of opinion that Leghorn, 43° north latitude, was "the most northerly place it could reach." Caillot says yellow fever was imported into Brest (in

coast of intertropical Mexico between the months of November and March, when the mean heat does not exceed 71° Fahr. Wherever this disease has visited in the old world, it has always been preceded by a season of unusually high temperature. The mean heat of the hottest month in England does not exceed 61°3 Fahr.(b) Hence, experience justifies us in concluding that yellow fever, even if introduced into this country, can never find an abiding-place here.

45°23 north) and communicated to a custom-house officer of that place. His words are :—"La ville de Brest, située beaucoup plus au nord que Cadix, a pourtant eu de preuves non equivoques du caractère contagieux de la fievre jaune. Lorsque cette maladie était dans toute sa force au Cap (San Domingo et qui revenait beaucoup de vaisseaux à Brest un employés des douanes fut placé abord de l'un de ceux qui avaient perdu du monde pendant la traversée, pendant le temps de son sejour abord, cet homme contracta un fievre jaune, dont il mourut dans moins de trois jours." Two other custom-house officers were afterwards attacked, and one of them died. Cases similar to these occured in the Eclair, while on her passage from the Motherbank to Standgate Creek, and while at Standgate Creek, in October, 1845. There were also two men sent on shore to Woolwich Infirmary, from the Growler, (which, like the Eclair, had just arrived from the west coast of Africa,) who died of unequivocal yellow fever. Neither the cases at Brest, nor those in the Eclair, can be considered to furnish absolute evidence of contagious propagation; for some will say that they were caused by the agency of a local cause in the ship herself, independent of the crew. They merely show that yellow fever can exist, for a very brief period, in countries situated considerably beyond the tropics.

(b) Mr. Glaisher, of the Royal Observatory, Greenwich, has kindly given me the following mean temperature of every month, as deduced from 79 years' observations:—

	Fahr.		Fahr.
January	39°7	July	61°3
February	38°2	August	60°5
March	40°9	September	56°3
April	45°7	October	49°3
May	52°6	November	4[illegible]
June	58°9	December	38°2

REPORT

OF THE

PROCEEDINGS OF THE SANATORY COMMITTEE

OF THE

BOARD OF HEALTH,

IN RELATION TO THE

CHOLERA,

AS IT PREVAILED IN NEW YORK IN 1849.

NEW YORK:
McSpedon & Baker, Printers, 25 Pine Street.
1849.

Members of the Sanatory Committee.

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CONTENTS.

PAGE.

Report of the Sanatory Committee to the Board of Health, 5

APPENDIX.

1. Report of Seth Geer, M. D., Resident Physician,..... 45
2. Report of Professor Ellet on the analysis of the atmosphere during the Cholera,..... 59
3. Report from the Centre and Thirty-fifth street Hospitals, 65
4 Report from the William and Centre street Hospitals,.. 82
5. Report from the Thirteenth street Hospital,..... 90
6. Report from the Stanton street Hospital,..... 99

REPORT.

THE CHOLERA having finally left our city, the Sanatory Committee in bringing their labors to a close, deem it their duty to make a full report of their proceedings to the Board of Health, by whom they were appointed, and whose views and wishes they have endeavored to fulfill according to the best of their ability. The Committee consider such a report to be necessary, not merely as a record for future reference, but as a justification of some of the measures which they have felt themselves obliged to take in opposition to the remonstrances of many of their most respected and valued fellow-citizens. In making their report, it is not their intention to enter into discussions in relation to the nature, causes, or the modes of treating the epidemic which has recently afflicted our city. These are subjects not within their province, and they shall leave them where

they properly belong, to the medical profession, feeling assured that if the members of that profession acknowledge themselves embarrassed by numerous problems connected with this mysterious disease, it is hardly to be expected that a body of laymen can enter the field of controversy with profit to themselves or advantage to the public. Their design is simply to give an account of the various measures which they adopted with the view of guarding the public health, and of protecting, as far as lay in their power, the interests of our great metropolis.

The Sanatory Committee were appointed by the Board of Health on the 16th day of May, 1849, and were invested with the full powers of the Board. Feeling the great responsibility resting upon them, and knowing that many questions would have to be decided by them involving medical facts and principles, their first business was to associate with them a medical counsel, for the purposes of advice and consultation. Three gentlemen of high standing, and in whom the profession and the public reposed full confidence,

were accordingly selected, and these, together with the Resident Physician and Health Commissioner, have acted as the medical advisers of the Committee during the whole prevalence of the epidemic.

The first case of cholera was announced on the 14th of May, and as this and several of the succeeding cases occurred at the "Five Points," a locality pre-eminent for its filth, vice and destitution, and where appeared to exist every requisite for the spread of the disease, it became evident at once, that some public place should be immediately provided, to which the sick might be removed, with the view not merely of separating them from the offensive and foul region in which they were lying, but to give them the best possible chance of recovery. A small two story house, No. 127 Anthony street, was accordingly taken at once as a temporary hospital, and Dr. William P. Buel appointed as the physician to take charge of it. To this place seven patients were carried. As it was apparent that should the cases of cholera increase, a larger and permanent hospital would

be required, the question at once presented itself where shall this hospital be located? Shall it be in the city, and in the neighborhood where the cases are at present occurring, or shall it be remote from the city, and so far secluded as to prevent any communication with the neighboring population? As the decision of this question depended entirely upon that of another question, i. e. the contagious or non-contagious character of the disease, it may readily be conceived that the Committee felt themselves not a little embarrassed. They found that medical men of the highest eminence differed on this subject, and that not merely theoretical opinions, but facts were arrayed in favor of the opposing doctrines. After obtaining all the light they possibly could from the testimony of medical men both at home and abroad, on mature consideration, they came to the conclusion that however certain isolated facts seemed to favor the doctrine of contagion, yet as a general rule, the disease did not appear to be propagated in this way—that the cause of it appeared to exist in the atmosphere, and that its spread was entirely independent of any communi-

cation between the well and the sick. The Committee confess that they were happy to come to this conclusion; it relieved them from the difficulties attending a selection of hospitals at a distance—it obviated the inconvenience and danger (sometimes fatal) of a long transportation of the sick—while it enabled the Committee to make more efficient provisions for the comfort and treatment of those attacked with the disease. Having come to this decision, a large three story building, called "Monroe Hall," at the corner of Pearl and Centre streets, was immediately secured, and possession taken on the 18th day of May. This building was in the neighborhood of the "Five Points," but combined the advantages of air and elevation. The two upper stories consisted of large halls, well calculated for the wards of a hospital. This building has since been known as the "Centre street Hospital." To this hospital, all the patients were immediately transferred from the temporary house in Anthony street, and the whole placed under the care of Dr. Buel.

On the 21st day of May, the Sanatory Commit-

tee in company with his Honor the Mayor and the Medical Counsel, made a personal visit to the "Five Points," with the view of satisfying themselves more completely in relation to the condition of this part of the city. It is not the intention of the Committee to describe what they witnessed. To be appreciated, the place must be seen. Suffice it to say, that the exhibition of human degradation and wretchedness which presented itself was truly appalling. To those of the Committee who visited this place for the first time, it seemed almost impossible that such a state of things could exist in any portion of our city. In addition to what had been done previously, renewed orders were now given, and efficient measures adopted to cleanse the place.—The truth, however, cannot be concealed—the place itself is incapable of proper purification—and will continue to remain so until it is razed to the ground, filled up and suitably rebuilt.—To accomplish this, will require Herculean energy; our city, however, is equal to any thing, and the Committee do not know of any object

to which its united interest and philanthropy could more legitimately and advantageously be directed.

As it now seemed evident that we were about to be visited by a severe and long continued assault of the epidemic, the Committee felt it to be their duty at once to enter upon the consideration of those general measures which might be deemed necessary to meet, in the best possible manner, the impending danger. In doing this, they found several important facts, which appeared to be well established, and which might serve as guides in their operations. These facts are the following:

1st—That the general cause of the disease appears to exist in the atmosphere.

2d—That in attacking individuals, the disease generally gives notice of its approach by some preliminary symptoms.

3d—That these preliminary symptoms are usually under the control of medicine, and being arrested, the further development of the disease is prevented.

4th—That the agency of various exciting causes is generally necessary to develope the disease. Among these causes the principal are the existence of filth and imperfect ventilation, irregularities and imprudencies in the mode of living, and mental disturbance.

With these facts in view, the duties of the Committee were obvious, and they conceived them to be the following:

In the first place, to provide public hospitals for the reception and treatment of those actually attacked with the disease, and who, from poverty or other causes, might be deprived of the means of being attended to properly, at their own homes.

In the second place, to adopt such measures as might counteract the development and extension of the disease by obviating, as far as possible, all the exciting causes of it, and also, by prompt medical assistance arresting the disease in its preliminary stages.

With these objects and purposes in view, the Committee commenced and continued their labors throughout the whole course of the cholera. They established hospitals in such succession and in such parts of the city, as the spreading of the disease required, until the whole number amounted to five. They commenced and accomplished a thorough purification of the city, such a one as the city probably never had before. Through repeated publications from the Medical Counsel, they kept their fellow citizens constantly alive to the important fact that the progress of the disease depended, in a great measure, upon their own discretion in their modes of living. They endeavored to inspire them with moral courage, as one of the best preservatives against disease—and finally, they supplied the city with such an abundance of medical attendance as that every person might have the benefits of immediate advice and attention.

Such in a very few words, is a sketch of the general plan of operations pursued by the Committee, and with which, on reflection, since the

cholera has passed by, they have every reason to be satisfied. With the details entering into the carrying out of this plan, it is unnecessary to trouble the Board. ' There are some points, however, which the Committee deem of sufficient consequence to notice, and may be of practical importance hereafter, should the cholera again unfortunately assail our metropolis.

On the 24th day of May, the following address, prepared by the Medical Counsel, was ordered to be published in all the city newspapers, and a suitable number in pamphlet form, for distribution:

New York, May 24, 1849.

The undersigned having been requested by the Sanatory Committee of the Board of Health, to prepare a communication to the public, in relation to the cholera, respectfully

REPORT:

That as yet, only a few cases of the disease have occurred, and from the peculiar locality where they have appeared,* amid destitution, filth and intemperance, the hope is cherished that under the protection of a kind Providence, this city may yet be preserved from its ravages. It is evident, however, that the cause

* The Five Points and its vicinity.

of the disease is among us, and it therefore becomes a duty to adopt all such precautionary measures as may tend to mitigate its evils, should it unfortunately become epidemic. Under this impression, they beg leave to make the following suggestions:

As the whole history of the cholera shows that its diffusion is promoted by all those causes which have a tendency to render the air impure, the first and most important concern relates to the cleanliness of the city. To accomplish this, the undersigned are satisfied that the Board of Health are taking such measures, and will continue to do so, as they trust will be efficient. The Board of Health, however, it is evident, cannot do every thing, and the Committee would respectfully call upon their fellow citizens to co-operate with them, in every possible way in their power. Sources of filth and impurity may exist without the knowledge of the public authorities, and every citizen should feel the necessity of keeping a watchful supervision over his own premises, and when individual efforts are inadequate, to call upon the Board for aid and assistance.

Believing that much may be done in the way of prevention, if not cure, of the disease, in case it should unfortunately extend among us, the undersigned would offer a few suggestions of a general nature, founded on experience, leaving the application of them to the good sense and discretion of the community at large.

In the first place, they would advise particular attention to clothing. From the nature of the complaint, it is evident that much depends upon keeping the body protected from sudden alterations of temperature and moisture. Flannel next the skin should be a universal article of apparel, and they would respectfully suggest to those charitable associations which are in the habit of supplying the poor with clothing, to make this an object of special attention.

In the second place, the diet should be particularly attended to. The undersigned would not recommend any sudden or great change in the ordinary modes of living, where those modes are temperate, and have been found to agree. Such changes, they believe, would do more harm than good. From the peculiar nature of the disease, however, it is well known that certain kinds of foods are injurious, and, without going into particulars, they would merely state that all such articles as have a tendency to relax the bowels, ought to be avoided. All crude and raw vegetables, unripe fruit, as well as violent purgative medicines, are calculated to do mischief. Excesses, either in eating or drinking, cannot be too religiously abstained from.

In the third place, attention to personal cleanliness, by the frequent use of the tepid bath, is particularly recommended.

In the fourth place, the preservation of a calm and composed state of mind is all important, and may do more than is generally supposed in preventing the onset of this disease. It is the result of experience that all epidemics are aggravated more or less by mental disturbance, whether in the shape of active panic or low despondency. To the cholera this is particularly applicable. While our citizens, therefore, use every prudential and precautionary measure, let them keep a good heart and dispel all fear.

In the fifth place, with regard to the treatment of cholera, it may be observed that, as a general rule, the disease does not attack so suddenly as to preclude the possibility of calling in medical assistance. A relaxed state of the bowels for a longer or shorter period, gives notice of its approach. In all cases, therefore, when any disorder of this kind exists, common prudence will suggest the necessity of resorting to medical aid. If this be done in season, the disease may generally be promptly arrested. When professional aid cannot be immediately obtained, and

where simple relaxation exists, twenty or twenty-five drops of laudanum may be taken; to be repeated in one or two hours, according to circumstances. For young persons and children, the dose must be reduced, according to the age—at the age say of ten years, eight drops; at the age of two or three years, two or three drops.

Where the symptoms are more severe, and the patient is cold, in addition to the laudanum, he should be put immediately to bed, between blankets, and every appliance in the shape of bottles of hot water, bags of hot salt or sand, frictions, &c., be diligently resorted to. A strong mustard poultice, too, should be applied over the region of the stomach, to remain on till it produces smarting of the skin. In addition to this, a little brandy and water should be given, with a view of restoring warmth. As in this city no difficulty in obtaining the speedy assistance of a physician can exist, any directions in relation to the after treatment are deemed unnecessary.

JOHN B. BECK, M. D.,
JOSEPH M. SMITH, M. D.,
SAMUEL W. MOORE, M. D.,
} *Medical Counsel.*

SETH GEER, M. D., *Resident Physician.*

RICHARD L. MORRIS, M. D., *Health Commissioner.*

At the same time the following hand-bill was prepared and directed to be distributed throughout the city, and posted up in all public places:

NOTICE.—Preventives of Cholera, published by order of the Sanatory Committee, under the sanction of the Medical Counsel.

Be temperate in eating and drinking. Avoid raw vegetables and unripe fruit. Abstain from cold water, when heated, and

above all from ardent spirits, and if habit have rendered them indispensable, take much less than usual. Sleep and clothe warm. Do not sleep or sit in a draught of air. Avoid getting wet. Attend immediately to all disorders of the bowels. Take no medicine without advice. Medicine and medical advice can be had by the poor, at all hours of the day and night, by applying at the Station House in each Ward.

CALEB S. WOODHULL, *Mayor.*
JAMES KELLY, *Chairman of Sanatory Committee.*

As it soon became evident that should the cholera continue to increase, a number of hospitals would be required in different parts of the city, a sub-committee was appointed to report on the subject. After a laborious investigation, the following was presented:

The Committee on Hospitals, to whom it properly belongs to recommend suitable buildings to be used as cholera hospitals, and to whom was referred a resolution passed by the Board of Education at its last meeting, which resolution reads as follows:

"*Resolved*, That this Board respectfully remonstrate against the appropriation of public school houses, by the Board of Health or other Corporation authorities, for cholera hospitals, and urge the Common Council to take prompt measures to revoke any such appropriation that may have been already made, and to give some public expression against such appropriation in future."

Beg leave respectfully to REPORT: That they have a high respect for the honorable gentleman who proposed the resolution, as also for the very intelligent Board which unanimously adopted

the same; that they concede the right of petition or remonstrance to the Board of Education, as they do to any and all other of their fellow citizens; yet they are not prepared to admit that the Board of Education is the best judge as to the measures proper to be pursued by the Board of Health. From the nature of the circumstances it cannot be expected. The Board of Health consists of the Mayor, eighteen Aldermen, eighteen Assistant Aldermen, Resident Physician and Health Commissioner; and as that number of persons was thought too large to transact the daily executive duties with efficiency which devolve on said Body, during the prevalence of an epidemic, seven persons were chosen from the two Boards, subsequently two more were chosen, to whom the whole power of the Board of Health was transferred, and which Committee are of necessity compelled to meet daily in the discharge of the responsible trusts committed to them, exercising their best judgment in adopting measures to preserve the health of a city of four hundred and fifty thousand inhabitants, among which duties is that of locating hospitals. And here we need not apprize the Sanatory Committee of the trial of our patience, the anxiety and the numerous difficulties experienced in every step of our progress. Anxious to avoid the necessity of sending children from any of the schools, your Committee have made unwearied efforts to secure other suitable buildings, and have been entirely unable to do so in a single case, unless at the most extravagant and enormous prices. And while at this time only one private building has been converted into a hospital, partly by agreement and partly by the necessity of our requirements, we think when all the expenses of that building are ascertained, the Committee will feel satisfied that the words "extravagant and enormous" were not wrongly applied. In short, whenever and wherever your Committee have made efforts to procure private property, that moment the alarm has been sounded, and they have been either entirely refused, or a price named so exorbitant that it could not be entertained. The expenses, ordinary and extraordinary, of our

city, have been for several years largely on the increase, and have indeed become burdensome to the people, and the augmentation of the same in consequence of the present epidemic, and the measures which have been taken to prevent its spread, (in the opinion of your Committee, wisely taken,) are already very heavy and likely to increase largely, and as faithful servants of the people, while we hold office, we consider it our duty not to lose sight of this circumstance, and as far as in our power consistent with a due regard for the best interests of all our fellow-citizens, carefully to guard the public treasury, and scrutinize every point and direction where disbursements are being made to prevent, if possible, too free a use of the public funds. The sickness in our city has not been rapid in its progress, and the Committee did not deem it advisable to erect buildings for hospitals, or to appropriate them any faster than they were actually needed, so that if the epidemic did not increase, more hospital accommodations would not be required. Modern school houses are, in the opinion of your Committee, well adapted for the purposes of cholera hospitals. They are large, well ventilated buildings, so arranged as to give one story to males, another to females, and a third to convalescents. The recitation rooms are convenient for nurses and attendants, having passages for egress and ingress, separate and distinct, so that the unfortunate sick can be taken in at one door, and the dead taken out at another, without the sick and convalescent being compelled to witness the painful ceremony of transporting the dead in their sight. These buildings are also the property of the city, and in that sense appropriate, for it is the city's unfortunate fellow-beings for whom your Committee are providing. In taking these buildings, the Corporation will avoid paying exorbitant rents, and (what is always very desirable,) litigation in any of its forms.

Again, the schools are to be closed on the 1st of August, for a vacation of a month or more, and in taking possession now, near the 1st of July, the children are deprived of, at the farthest, but

one month's tuition in the hottest summer weather. It is a matter of consideration whether they are not better off *out* than *in* school. In some instances your Committee have found them so congregated as to render the air very impure and unwholesome, so much as to have led to the enquiry whether their duty as a Sanatory Committee did not require some steps to be taken for lessening the numbers of many of these schools during the excessive heat of summer, which subject, in the opinion of your Committee, demands immediate consideration by the Sanatory Board.

Your Committee are the friends of education, whether in the elementary or higher branches. They believe the laws adopted within the last few years for improving the character of the schools in this City, opening them alike to the children of the rich and the poor, and in which all are equally well instructed, most benevolent and creditable to their authors and supporters, and not less to be prized as a real and enduring measure of public benefit than any on your statute book. They do not believe, however, that the temporary appropriation of two or four school houses, will be any serious detriment to the cause of education in this city. Admitting it an evil, it is the one of *lesser* magnitude, and should give way to the *greater*. The building selected in William street, was deemed a very suitable one in all respects, and your Committee have not yet seen any good and sufficient reason to change the appropriation thus made of Public School No. 1.

In reply to that portion of the resolution of the Board of Education, which requests the Common Council or other Corporation authorities, to give some expression against the appropriation of school houses in future, they desire distinctly, but respectfully to state, that, for the reasons herein given, they not only cannot give *any such public or private expression*, but in justice to them-

selves, to their fellow citizens who sent them here, and to those poor unfortunate beings without home, without shelter, daily and hourly needing our sympathy and aid, they cannot fail to recommend as they hereby do from this time forth, the appropriation for cholera hospitals, when other suitable buildings cannot be procured, public or ward school houses, selecting as nearly as may be, an equal number from each organization, in the belief that humanity and compassion for the destitute and dying, require the very best temporary accommodations which can be obtained, and that no others bear any favorable comparison in their adaptation to the purpose.

EDWIN D. MORGAN,
ALEX. H. SCHULTZ,
ROBERT A. SANDS,
CHARLES WEBB,
Committee on Hospitals.

New York, June 28, 1849.

The above report was accepted by the Sanatory Committee, and they determined to act according to the recommendations of the sub-committee. In carrying their plans into execution, however, they found themselves constantly embarrassed by difficulties. Public meetings of citizens were held, protesting against the establishment of hospitals in certain localities, while the friends of education made the most vigorous opposition to the selection of the school houses for that purpose. After a full and mature consideration of the subject, the Sanatory Committee determined that every thing ought to give way to the public good, and such of

the public schools as were deemed necessary, were successively occupied as cholera hospitals. As is generally the case, decision was followed by acquiescence, and the Committee believe that no one now denies the wisdom or propriety of the measure. The first school house selected as a cholera hospital, was in William street, under the professional charge of Dr. ALEXANDER F. VACHE; the second in Thirteenth street, under the supervision of Dr. OVID P. WELLS; the third in Stanton street, under the control of Dr. ISAAC GREENE, and the fourth in Thirty-fifth street, under the care of Dr. WILLIAM P. BUEL, who was transferred from the Centre street Hospital, on the closing of that institution, which was subsequently re-opened, under the medical direction of Dr. VACHE, and continued for the reception of patients until the disappearance of the epidemic in the city.

On the 14th day of June, a petition was sent in to the Board of Health, requesting the establishment of a cholera hospital, in which patients might be treated on the homœopathic plan. This petition having been referred to the Sanatory

Committee, was by them again referred to the medical counsel, by whom the following report was made :

NEW YORK, JUNE 19th, 1849.

A resolution having been offered to the Board of Health, ' that a hospital for the reception of cholera patients, be established in this city, in which the practice of homœopathic physicians shall be pursued,' and the same having been referred by the Sanatory Committee to the Medical Counsel, the undersigned state that should the above resolution be adopted, they see no satisfactory reason why the same courtesy should not be extended to the Hydropathists—the Thompsonians—the Chrono-thermalists, and indeed all others claiming to have specific modes of treating the cholera.

By intelligent and well educated physicians generally, homœopathy is looked upon as a species of empiricism. It is neither practised by them, nor countenanced by them Concurring entirely with their professional brethren on this subject, the undersigned conceive that the public authorities of our city would not consult either their own dignity or the public good, by lending the sanction of their name or influence to homœopathy or any other irregular mode of practice.

JOHN B. BECK, M. D.,
JOSEPH M. SMITH, M. D., } *Medical Counsel.*
SAMUEL W. MOORE, M. D.,

SETH GEER, M. D., *Resident Physician.*
RICHARD L. MORRIS, M. D., *Health Commissioner.*

In adopting this report, the Sanatory Committee did not wish to be considered as expressing any opinion either in favor or against what is commonly denominated homœopathy. This they viewed as a subject entirely beyond their province. In executing a great public trust, however, which had been committed to their hands, they did not feel that they had the right of arraying themselves against any of the existing institutions of the community for whom they were acting. However defective these institutions might be, it was not for them to set about the work of reform. In looking round, accordingly, they found the medical profession existing in a certain form, exercising certain rights and privileges conceded by law, and recognized by long usage and general suffrage. The medical profession was, in fact, one of the legitimate divisions into which society had resolved itself, and for the performance of the most important functions subservient to the public good. Taking this view of the subject, the Committee felt it to be their duty to have nothing to do with medicine, except as they found it embodied in what is understood and known both by the public, as well as physicians, as the regular profession. While in this way they paid all suita ble respect to so honorable a profession as that of

medicine, the Committee felt that they did no injustice to those who suppose themselves in advance of the age, and profess themselves gifted with superior knowledge and wisdom.

In providing the poorer classes of the city with all necessary medical advice and attendance, the Committee adopted two modes, which proved to be exceedingly efficient and useful. One of them was the appointment of a number of physicians, whose business it should be to be in constant attendance at the "Police Station houses," of which there is one in each Ward. The object of this, was to have some convenient and well known place in each of the Wards, where prompt assistance might be obtained by all those who felt disposed to avail themselves of it. Medicines accordingly were provided, and two physicians were appointed to each Station house, and public notice of the fact was given in the newspapers.

Beside this, the parts of the city where the cholera prevailed was divided into districts, and physicians were appointed to each district for the purpose of visiting the houses of the poor, with the view of ascertaining the existence of disease

and affording immediately the necessary relief. The number of physicians appointed to the discharge of this duty was twelve.

The amount of good accomplished by these measures, especially the last, in the way of preventing the developement of disease, the Committee believe to have been great.

On the 5th of June, the following communication, presented by the Medical Counsel, was directed to be published in each of the daily and Sunday papers:

New York, June 5, 1849.

In reporting to the Sanatory Committee in relation to the cholera at present prevailing in our city, the undersigned are happy to state, that although the disease is gradually extending among us, yet in its general character it is much milder than it was when it visited this city in 1832; that it seems more under the control of remedies, and that the proportion of deaths is smaller. These facts are consoling, and should excite in the hearts of our citizens the liveliest feelings of gratitute to a kind Providence.

The undersigned would also state, that whatever opinions may be entertained in relation to the abstract question as to whether the cholera is or is not a contagious disease, no evidence has appeared, thus far, that the disease at present prevailing in our city, has in a single case been produced by contagion; on the contrary, every thing connected with its history proves

that its origin and diffusion have been dependent upon causes entirely different. The simultaneous occurrence of cases in different and distant parts of the city, between whom no sort of communication could be traced, and the immunity enjoyed by those holding constant intercourse with the sick in our cholera hospitals, are facts which establish this point beyond the possibility of dispute. In not a single case, as yet, have either physician, nurse, or visitor of our cholera hospitals been known to contract the disease. These facts should dispel entirely from the minds of our citizens all fear in attending their friends and relations who may be attacked with the disease, and at the same time dissipate whatever apprehensions may hitherto have existed in the public mind in relation to the establishment of cholera hospitals in any part of our city, when called for by the demands of necessity and humanity.

The undersigned believe that the cause of the disease exists in the *atmosphere*, and that the whole community are at present more or less under the influence of this peculiar condition of the atmosphere, and in this way predisposed to the disease. To develope the disease, however, *exciting causes* are necessary, and these are to be found in all those things which have a tendency to disorder the bowels. With regard to the peculiar condition of the atmosphere which predisposes to the disease, we know nothing. Human skill and human agency, therefore, can do nothing in meeting this difficulty. The exciting causes, on the contrary, are in a great measure under our control, and by properly guarding against these, much, very much, may be done in obviating the developement and extension of the disease.

John B. Beck, M. D., } *Medical*
Joseph M. Smith, M. D., } *Counsel.*
Samuel W. Moore, M. D., }

Seth Geer, M. D., *Resident Physician.*
Richard L. Morris, M. D., *Health Commissioner.*

On the 13th day of June, a communication was received from Professor Ellet, expressing his intention to analyze the atmosphere during the prevalence of the cholera, and requesting the cooperation of the Sanatory Committee. On being referred to the Medical Counsel, they reported as follows:

New York, June 15, 1849.

A communication having been addressed to the Sanatory Committee, from Professor Ellet, in relation to analyzing the atmosphere during the prevalence of the cholera in this city, and the same having been referred to the Medical Counsel, the undersigned state, that in their opinion the subject is one of the highest importance and eminently deserving of investigation. It involves interests common to science and humanity. Whatever may be the result of the investigation, it cannot fail to attract general attention, and should any discovery fortunately be made which throws light upon the cause of the cholera, it will reflect the highest credit upon the authorities under whose sanction and patronage it originated. With regard to the qualifications of Professor Ellet, as a chemist, the undersigned deem them of the highest order, and they are of opinion that the whole subject may be safely entrusted to his hands.

John B. Beck, M. D., } *Medical*
Joseph M. Smith, M. D., } *Counsel.*
Samuel W. Moore, M. D., }

Seth Geer, M. D., *Resident Physician.*
Richard L. Morris, M. D., *Health Commissioner.*

Convinced of its importance, the Committee

felt a deep interest in this subject, and were prepared to make an appropriation for the purpose of aiding Prof. ELLET in the prosecution of his researches; but on consultation, they found that he preferred going on upon his own responsibility, under the sanction of the Board. A copy of the above report, certified by the Sanatory Committee, was accordingly transmitted to Professor ELLET. The results of these investigations will be found in a communication from Professor ELLET.*

On the 23d of June, the following communication from the Medical Counsel, was directed to be published in the editorial columns, three times in each of the daily papers, and once in each of the Sunday papers:

NEW YORK, JUNE 23, 1849.

The undersigned, in the discharge of their duty as the Medical Counsel to the Sanatory Committee, are happy to state that although the cholera is still prevailing in our city, yet it does not appear to be on the increase. They are confidently of opinion that this is owing to the observance on the part of our citizens generally, of those prudential measures which have been so strenuously recommended by the Sanatory Committee. With regard

* See Appendix, No 2.

to the cases which have occurred, the undersigned state that so far as their observation has extended, they have been mainly referable to three causes, viz: the neglect of a relaxed state of the bowels—the taking of cathartic medicines—and imprudencies in diet. They would therefore, take the liberty of urging upon their fellow citizens the necessity of the most rigid and continued caution in relation to all these circumstances. Let not a diarrhœa be neglected for a single moment—let no cathartic medicine be taken except by the direction of some regular physician—and with regard to diet, let the utmost prudence be observed both in the quantity as well as the quality of the food which is taken. During the excessive heat, at present prevailing in our city, the digestive organs are necessarily much enfeebled, and the overloading the stomach, even with good food, is frequently as injurious as taking those things which are known to be indigestable.

JOHN B. BECK, M. D.,
JOSEPH M. SMITH, M. D.,
SAMUEL W. MOORE, M. D., } *Medical Counsel.*

SETH GEER, M. D., *Resident Physician.*
RICHARD L. MORRIS, M. D., *Health Commissioner.*

Believing that moral influences exercised no inconsiderable control over the cholera, the Committee thought that they might advantageously call upon the clergy of our city to co-operate with them in their endeavors to arrest, if possible, its progress. They accordingly prepared the following circular, a copy of which was addressed to every clergyman in our city:

New York, City Hall, July 6, 1849.

Rev. Sir:

The Sanatory Committee of the Board of Health, in their endeavors to mitigate the evils, and if possible, limit the extension of the cholera in our city, deem it their duty to call upon the clergy for their aid and co-operation. The Committee believe that the clergy may do much good, by addressing their congregations on the subject of the present epidemic, if not in a formal, at least in an incidental manner. Without presuming to dictate, the Committee believe that the points which might be most advantageously alluded to, besides the ordinary imprudencies in the mode of living, are the injurious effects of fear and of excitement, whether mental or physical. A composed state of mind, and a humble but cheerful reliance on Providence, are all important in times like the present.

As congregations differ so widely in their general character and habits, the mode of addressing them is best determined by the particular clergyman attached to each, and to his discretion and judgment, the whole matter is respectfully submitted.

James Kelly, *Chairman*,
Robert T. Haws,
Alex. H. Schultz,
Charles Webb,
George H. Franklin,
Edwin D. Morgan,
Robert A. Sands,
Jacob F. Oakley,
Oscar W. Sturtevant,
} *Sanatory Committee of the Board of Health.*

John B. Beck, M. D.,
Joseph M. Smith, M. D.,
Samuel W. Moore, M. D.,
} *Medical Counsel.*

Seth Geer, M. D., *Resident Physician*,
Richard L. Morris, M. D., *Health Commissioner*.

The Committee are happy to state that this circular was favorably received, and they have no doubt that the exhortations made both in private and from the pulpit were of eminent service.

They also acknowledge, with gratitude, the liberal contributions which were made in some of the churches, for the benefit of the poor.*

Many thanks are due to Messrs. Clarke & Co., for their kind offer to furnish the sick of the hospitals, with "Congress Spring" water, and for the abundant supply of it, without remuneration.

One of the subjects which attracted a large share of the attention of the Committee was that of *nuisances*. As might naturally be supposed in so large a city as New York, and containing a

* The following distribution was made of these moneys:

By amount of collection in Grace Church		$200 00	
" " Brick "		76 80	
" " Mercer st. "		51 40	
" " Trinity "		83 09	
" " St. Paul's "		84 82	
" " St. John's "		92 72	
" " Church of the Mediator		114 65	
			$703 48
To amount paid 11 Visiting Physicians to relieve the poor.	$275 00		
Less amount returned by them unexpended	37 25		
		237 75	
To amt. paid Society for Amelioration of the Condition of the Poor,		465 73	
			$703 48

population of so heterogeneous a character, almost daily complaints were made of their existence, and urgent petitions sent in requesting their immediate removal. Wherever it was in the power of the Committee to do so, they promptly applied the proper corrective. In many cases, however, difficulties presented themselves not appreciated by the public, and which were not so readily overcome. It is not every thing that is offensive to the sight or smell that is really a nuisance. What is looked upon as such by one class of persons, is not so by another. It is not always so easy therefore to decide at once what is a nuisance, and how far the authorities are justified in removing or suppressing it. Private rights too, are not to be encroached upon, unless evidently required by the public good. In some of the large manufacturing establishments too, considered as nuisances, hundreds of hands are employed, and upon which they depend for daily subsistence, and which ought not to be interfered with unless for the most imperative reasons of general and public security. The Committee make these remarks, in reply to the constant cen-

sures cast upon them by a certain portion of the public press, in relation to this matter. The Committee have only to say, that they acted in all cases according to the best of their judgment and discretion. While they endeavored to respect private rights as much as possible, they did not hesitate to suppress establishments however large, which were manifestly injurious to the health of the surrounding population.*

The Committee cannot dismiss this part of their report without bearing the testimony of their approbation to the indefatigable labors of the Captains of Police and their Health Wardens, in

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* The Committee were vested with full power over these matters, by the following resolution, passed by the Board of Health:

Board of Health.—At a meeting of the Board of Health, held July 14, 1849, the following preamble and resolution were adopted, and directed to be published.

D. T. Valentine, *Clerk.*

Whereas, Numerous complaints of an aggravated character, have been made to the Sanatory Committee of this Board, of the existence in this city of extensive manufactories of starch, soap and bone-boiling establishments, slaughter houses and other nuisances, the miasmas and exhalations of which are intolerable and injurious to public health; and

attending to the cleansing of the city. They take the liberty, however, of adding that the department which has the cognizance of nuisances is one of the most onerous, as well as important, connected with our city government. As at present constituted, it is wholly inadequate to the wants of our city. It requires an entire re-organization, and for this purpose, no period seems more appropriate than the present. The labors of your Committee, during the past appalling season of sickness

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Whereas, Upon examination, the neighborhoods of such places have been visited with unusual sickness and deaths by cholera, and

Whereas, The parties owning such establishments have refused to cause an abatement of said evils, after being duly notified, therefore, be it

Resolved, That this Board, in the exercise of the authority conferred upon it by the laws of this State, for the suppression and abatement of all nuisances that may be deemed and declared to be deleterious to public health and promotive of epidemical disease, do hereby empower and direct the Sanatory Committee of this Board to cause all such places mentioned in the foregoing preamble, and all other places of the like character, to be immediately shut up, and the business thereof discontinued during the prevalence of the said epidemic, and until the further order of the Board of Health.

and death, and the awful scenes of degradation, misery and filth developed to them by their researches, have brought into full view the fact, that we have *no Sanatory Police* worthy of the name; that we are unprotected by that watchful regard over the public health which common sense dictates to be necessary for the security of our lives, the maintenance of the city's reputation, and the preservation of the interests of its inhabitants. Cholera may again assail us before we know it, and it is the dictate of true policy to be prepared in season to meet it; and not cholera alone, but any and every other malady which may be produced or aggravated by local causes. To no other work should the authorities address themselves more earnestly than the establishment of a thoroughly organized medical police, at whose head should be an active and experienced medical man. The advantages of such a measure would be incalculable.

On the 6th of August, the following communication from the Medical Counsel, was ordered to be published in all the daily papers of the city:

August 6th, 1849.

The Medical Counsel of the Sanatory Committee, in the discharge of their duty, feel themselves called upon again to address a few words of advice and precaution to their fellow citizens.

In the first place, as the season has arrived when our markets are usually flooded with fruits of all descriptions, many of which are unripe, and some in a state of decay, our citizens cannot be too cautious how they indulge in the use of them. Even in ordinary seasons they are known to be the prolific source of serious and fatal bowel complaints. During the present season they are doubly pernicious. With regard to children especially, of all ages, every effort should be made to prevent them from either touching or tasting them. A single indiscretion in this respect, has been known to be fatal to life.

In the second place, during the hot season the greatest caution should be observed in incurring unnecessary exposure and fatigue. Some of our most valued citizens, it is to be feared, have, by such imprudence, sacrificed their lives. Let the pursuits of gain and the excitements of business be suspended, or at least moderated, for a time, and let every person who feels himself indisposed rest quietly at home for a few days, until the exhausted energies of the system are rallied. The undersigned are convinced that many a useful life might be saved by this simple precaution.

In the third place, the undersigned would again urge upon their fellow citizens the necessity of endeavoring, by every means in their power, to preserve a calm and composed state of mind. Melancholy experience has shown that among all the exciting causes of the cholera, none is so likely to bring it on, as a perturbed state of mind. Let our citizens rest assured that a manly courage, with an humble reliance on a beneficent Providence for protection, is one of the best preservatives against the assaults of this disease. Judging from past experience, as well as from present appearances, the undersigned believe that we have seen the worst of the cholera, and that not many weeks will elapse before returning health will bless our city

JOHN B. BECK, M. D.,
JOSEPH M. SMITH, M. D.,
SAMUEL W. MOORE, M. D.,
} *Medical Counsel.*

SETH GEER, M. D., *Resident Physician.*

RICHARD L. MORRIS, M. D., *Health Commissioner.*

In the progress of the cholera, it became manifest that the daily reports of the Sanatory Committee, and the weekly reports of the City Inspector did not correspond. The discrepancy, indeed, became so great as to attract general attention. The cause of this discrepancy was self-evident—many physicians not considering themselves compelled to report their cases, neglected to do so altogether—others merely reported cases without afterwards feeling themselves obliged to report the result. In this state of things, it was manifest that neither cases, nor deaths, especially the latter, could be reported with any degree of accuracy. On the other hand, as no person could be buried without its being reported to the City Inspector, his list of deaths would be much larger than that of the Committee. In all

this, the Committee hold themselves blameless. They could do no more than they did; they could only report the cases handed in to them by the physicians, precisely what the City Inspector did. The fault lay entirely with certain physicians, who, through mistaken views, resisted the execution of the law. The Committee regretted this exceedingly; it threw discredit upon the accuracy of their daily reports, while it betrayed a spirit on the part of the physicians unworthy of them as good citizens and members of a liberal profession. For the purpose of enforcing the requisitions of the law, the Committee issued the following:

BOARD OF HEALTH.

The Board of Health of the City of New York do ordain as follows:

§ 1. Each and every practising physician in the city of New York, shall, daily, at ten o'clock, report to the Board of Health of the said city, the number of persons attacked with Asiatic cholera in said city, or any other malignant diseases attended by such physician for the twenty-four hours preceding, and the number of persons attended by such physician who shall have died during the twenty-four hours preceding such report of Asiatic cholera, or other malignant diseases. Such report shall particularly specify the disease of each person mentioned therein. And such report shall also contain the birth place, residence, sex and

occupation of the person so sick or dead, if the same can be ascertained, and the age of the person so sick or dead.

§ 2. Such daily report shall be signed with the name of the physician making the same, and shall be left daily, and before the hour of eleven o'clock each day, at one of the Station houses of the several Wards in this city, with the Captains of Police, or some person in charge thereof, directed to the President of the Board of Health of the city of New York: and the persons so receiving the same shall immediately cause to be delivered the several reports so received, at the office of the Board of Health, in the City Hall of said city.

§ 3. Each and every physician who shall neglect or refuse to make such report, and deliver the same as aforesaid, shall be proceeded against as provided in and by the sixth section of the act of June 22d, 1832.

By order of the Board of Health.

JAMES KELLY, President *pro tem.*

N. B.—The Board of Health, for the information of physicians and others, publish the sixth section of the act of June 22d, 1832, now in force, and which is as follows:—

§ 6. Every person who shall wilfully violate any regulations so to be made and published by such Board of Health, shall be deemed guilty of a misdemeanor, and on conviction thereof, shall be subject to fine and imprisonment, or both, at the discretion of the Court; such fine not to exceed one thousand dollars, nor such imprisonment two years.

On the 5th September, 1849, the following report was presented by the Medical Counsel, which was accepted; and, on motion, it was

Resolved, That after this day the Board discontinue making daily reports to the public, but that physicians will be required to make reports as heretofore of such cases as may occur.

New York, September 5, 1849.

The undersigned, Medical Counsel of the Board of Health, are happy to state, as their opinion, that the cholera, in its epidemic form, may be considered as having, in a great measure, ceased to exist in our city. In a short time, it is hoped and believed, that it will be entirely extinct. In expressing the foregoing opinion, the undersigned deem it to be their duty to add that the long continuance of the cholera atmosphere has created predispositions to disease, which cannot of course, be at once eradicated. They would therefore, exhort their fellow-citizens not to abandon, prematurely, those prudential measures with regard to their modes of living, which have been so frequently urged upon their attention, and which, it is believed, have had a most salutary influence in limiting the extent of the disease. With due attention to these, the undersigned are of opinion that our citizens may return to their homes in perfect security, and that our city may be visited by strangers with as much security as any portion of the country.

John B. Beck, M. D.,
Joseph M. Smith, M. D.,
Samuel W. Moore, M. D., } *Medical Counsel.*

Seth Geer, M. D., *Resident Physician.*

Richard L. Morris, M. D., *Health Commissioner.*

The Committee have thus endeavored to give a full account of their proceedings during the prevalence of the epidemic cholera in our city. Under Providence, they believe they have accomplished

a great amount of good, and they trust their proceedings will receive the sanction of the Board of Health.

The Committee herewith submit reports from the Resident Physician and the Hospital Physicians, containing all the necessary information, in relation to the statistics and treatment of the recent epidemic.

JAMES KELLY, *Chairman*,
ROBERT T. HAWS,
ALEX. H. SCHULTZ,
CHARLES WEBB,
GEORGE H. FRANKLIN,
EDWIN D. MORGAN,
ROBERT A. SANDS,
JACOB F. OAKLEY,
OSCAR W. STURTEVANT, } *Sanatory Committee.*

APPENDIX.

NO. I.

REPORT OF SETH GEER, M. D.,

Resident Physician of the City of New York.

THE Resident Physician of the city of New York, in compliance with a resolution of the Sanatory Committee of the Board of Health, to furnish a report on the general history of the epidemic which has recently visited us, together with such statistics as may have been furnished by the various Medical Departments, herewith submits the following

REPORT:

On the 15th of last May, I received a communication from his Honor Mayor WOODHULL, informing me that through Dr. F. HARRIOT, he had learned that four cases of a disease resembling cholera, had occurred at No. 20 Orange street, (a part of the "Five Points") accompanied with a request that I would repair to the above place, and discover, if possible, the nature of the disease prevailing in that locality, and report the results of my investigations.

In accordance with these instructions, I visited the place designated, and in a rear basement, surrounded with filth and wretchedness, I found the body of a woman, who a few minutes before had expired. There was no mistaking the cause of death—the pinched and sharpened visage—the corrugated, blue, and attenuated appearance of the body showed too plainly the nature of the disease which had marked its victim—Asiatic cholera was manifested in every expression. While there, I endeavored to obtain what information I could from the persons by whom she was surrounded; yet I found it almost impossible to get a reply to the most simple interrogatory. In truth the idiotic condition which rum, debauchery and extreme wretchedness had reduced the intellects of these loathsome objects of humanity, completely incapacitated them from comprehending the meaning of the most simple question. From Dr. F. Harriot, the following particulars of the first seven cases have been obtained. On Friday, May 11th, Dr. H. was called upon to attend James Gilligan, a laborer, residing in the rear basement of No. 20 Orange street, who had sickened the night previous with vomiting and purging, attended with cramps. Having indulged freely in intoxicating drinks, &c., the night previous, his sickness was attributed to this cause, and he was treated accordingly. On the following day, he was better, and hopes were entertained of his recovery. On Monday, the 14th, Dr. H. learned that two women, lying in the same cellar, had sickened on the evening of the 12th, in a similar manner. On again visiting the place, he found the two women and his previous patient (Gilligan), presenting all the appearance of the stage of collapse of cholera. Their situation was truly deplorable—lying on a few filthy

rags on the half decayed floor of a miserably damp and dark basement, with clothing hardly sufficient to cover their nakedness—the odor on entering the place was disgusting in the extreme. Two of these cases died in a few hours. On the afternoon of the 14th, another sickened; this case died early the next morning. On the 16th, three more cases occurred in the same place, thus making a total of seven cases of cholera occurring already at No. 20 Orange street. On this day, the 16th, and the one following my visit, the Mayor convened the Board of Health, and made known to them the fact that the cholera had made its appearance at the "Five Points."

Although many of the members composing the Board of Health were but recently sworn into office, and consequently, as might be supposed, would not act with that promptness and energy which such an occasion required, yet they at once directed the proper authorities to repair to the locality where the disease had appeared, and not only employ every sanatory measure calculated to cleanse and purify the place, but to use every human effort to effect a thorough expulsion of all matters throughout the "Points," which might serve as a *pabulum* upon which the disease could feed. These, together with the establishment of a temporary hospital, at 127 Anthony street, were the work of a very short period of time. The hospital was placed under the charge of Dr. W. P. Buel, as physician to the same, and to which all seized with the disease were at once conveyed.

The Committee had innumerable difficulties to encounter while the disease lasted, one of the greatest was to obtain hospitals for the reception of the sick, and they did

not gain possession of the first in Anthony street, (although intended only for temporary use), until the chairman of the Sanatory Committee and some other of its members, after an unsuccessful negotiation, were obliged to force open the door of the building and take possession.

Notwithstanding the precautionary measures which were so promptly adopted and carried out in the locality where the cholera first appeared, it gradually spread itself over the "Points" and continued prevalent for a brief period of time; and there is no doubt but that the sanatory measures which were here adopted, did much to lessen the extent and malignancy of the disease.

On the 20th of May, his Honor Mayor Woodhull, the Sanatory Committee and the Medical Counsel visited the temporary hospital in Anthony street, and afterwards made a thorough inspection of the "Five Points." The streets were found perfectly clean, although some of the houses, alley-ways, yards and pig-styes in this locality required cleansing. Orders were accordingly left with the Health Warden and Captain of the Police, to superintend the work of purifying the same. In accordance with which, over three thousand hogs were removed to the outskirts of the city.

On the 26th of May, the Sanatory Committee reported two cases and one death as having occurred by cholera in a new locality (No. 10 Mulberry street), and about four hundred yards from the "Five Points." The first case which occurred in this place, was that of a woman, who was immediately conveyed to the Centre street hospital, corner of Centre and Pearl streets (which in the meantime had

been established by the Sanatory Committee), in a state of collapse, and who died in a few hours after admission. Several cases followed, and soon after the disease prevailed here as an epidemic. This locality, upon examination, was found in a filthy condition, yet through the active agency of the Health Warden, Mr. Francis Owens, who was as efficient an officer as any in the employ of the Sanatory Committee, the place soon bore a very different appearance. From this time, cases began to occur in different parts of the city, apparently without any connection with the previous cases, and the disease was now considered as having fairly assumed an epidemic character. It became necessary at this time, that some provision should be made for the poor and destitute, so that at any time they were stricken down by the epidemic, they could at once be provided with medical attendance and medicines from the different Station houses. To effect this object, a resolution was passed by the Sanatory Committee, to appoint two physicians for each Ward, whose duty it should be to visit and prescribe for all such as were attacked and unable to pay for attendance, and furnish for their use such medicines as were required.

The following named gentlemen were accordingly appointed as Ward Physicians:

Edward McGregor,.....48 Liberty street,
Jarvis M. Andrews,.....46 Pearl street,
P. W. Macdonnell,.....74 Beekman street,
Thomas W. Henry,.....75 Beekman street,
A. S. Doane,.....cor. Warren and Church,
Alexis Smith, (since deceased.)
A. H. Jones,.....148 Chambers street,

Joseph Hilton,.....44 Oak street,
William O'Donnell,.....41 Oliver street,
James Kennedy,.....186 Duane street,
J. Sage Kilbourne,.....126 Franklin street,
Owen Sweeney,.....97 Elm street,
C. Conning, ...30 Bowery,
R. S. Carpenter,.....3 Mulberry street,
William H. Clussman,.....11 Market street,
Saamuel Rotton,.....78 Jefferson street,
A. K. Gardiner,.....153 Wooster street,
Samuel S. Purple,.....183 Hudson street,
Edward R. Chapin,.....3 Eighth avenue,
William J. Johnson,
Samuel B. Phillips,.....415 Hudson street,
Daniel Wells,.....98 Eldridge street,
W. S. Tompkins,.....161 Bowery,
N. H. Cheseborough,.....639 Fourth street,
Elias S. Nichols,.....Columbia street,
John Ives,.....Forty-eighth street,
William Cecil, ...Eighty-sixth street,
G. A. Hills,.....126th street,
J. C. Hepburn,.....Forty-eighth street,
G. Prince,.....18 Columbia street,
W. Churchill,.....173 Clinton street,
Cornelius B. Archer,.....210 Mulberry street,
H. W. Thayer,
Samuel L. Griswold,.....328 Fourth street,
John W. Hubbell,.....201 West 18th street,
John S. Carter,.....187 West 19th street,
Bern W. Budd,.....22 Third street,
Thomas M. Franklin,.....11 East 17th street,
John Busteed,.....East 27th, bet. 2d & 3d av.
John G. Sewall,.....372 Fourth avenue,

At the same time two sets of men were appointed to each Ward, whose duty it was to convey the poor and helpless, who sickened with cholera, to the Centre street and other hospitals, which were soon after established.

On the 8th of June, the public school house No. 1, in William street, near Duane, was appropriated by the Sanatory Committee, and opened on the 9th for the reception of patients, under the care of Dr. Alexander F. Vache. This hospital, like that of Centre street, and those that were subsequently opened, were well supplied with physicians, nurses and orderly men.

On the 8th of June, a case of cholera occurred at Nos. 7 and 9 Washington street, in the First Ward. The person seized was a woman named Mary M'Annally, a servant in the employ of Michael Burke, keeper of an emigrant boarding-house. She was at once conveyed to the Centre street hospital, where she died on the 10th of June. The disease here for a period of two weeks, continued with considerable violence. The localities were it was most fatal, was in Washington, West, Morris, Cedar and Thames streets. Soon after it broke out in Trinity place, and in a short time disappeared almost entirely from this part of the city. During the existence of the cholera in these localities, a number of very severe cases occurred. Many proved fatal. Those who sickened were immediately conveyed by the sedan-carriers to the hospitals, and the several places where the disease prevailed, thoroughly disenfected and cleansed in every part.

On the 1st of June the first case of cholera occurred in the Seventh Ward. The name of the person attacked was

Abraham Devoe, residing at No. 21 Catharine street. On the 2d of June a case occurred at No. 218 Cherry street, and terminated fatally in six hours. The cases in the Ward, from this time up to the 20th of June, were merely sporadic. From this date, however, to the 1st of August, it appeared with great violence at Nos. 430, 446, 448 and 445 Water street, and shortly after at No. 151 Cherry street. From these localities the disease followed to East Broadway, near Rutgers street; and after a period of two weeks, again broke out in Scammel, Cherry, Water, Walnut, Madison and Division streets. From the 10th of June to the 20th of August, one hundred and seventy deaths by cholera occured in this Ward. Many of the population in the localities referred to were emigrants, and persons utterly destitute of the ordinary comforts of life. Such of those as sickened with the disease were taken to the cholera hospitals, where every attention was given them, and after a brief period the disease subsided, and appeared next in the Twelfth and Sixteenth Wards. The cholera first appeared in the Sixteenth Ward, June 23. On this day, a case occurred in Thirty-third street, near the Tenth avenue, and on the 9th of the same month the disease became epidemic from Twenty-fourth to Fortieth street, and from the Eighth avenue to North river. On the elevated ground in the neighborhood of Thirty-fifth street, from the Ninth avenue to the North river, it raged with great violence. In many instances eight and ten persons from one house were seized and died of the disease. The cholera was equally malignant in Thirty-third street, near the Tenth avenue, and continued epidemic in these localities until the latter part of August. In the Twelfth Ward, the first case of cholera occurred on the 8th of June, in Forty-

second street, between the Ninth and Tenth avenues. The disease in a few days was disseminated over the whole Ward, and continued with unabated violence for a period of nearly two months. At Harlem and Manhattanville the first cases occurred about the middle of July. As many as thirty deaths by cholera occurred, and about the middle of September the disease here was entirely extinct.

The cause of its long continued and malignant character in this part of the city, can only be accounted for from the fact that there were found many filthy localities spread over the surface which was occupied by the epidemic. Several bone boiling establishments were discovered, with immense piles of bones scattered around, while heaps of the same were found with meat attached, and all in an advanced state of decomposition, and exhaling a stench which was horrible in the extreme. These, together with manure heaps, pools of stagnant water and a horse killing establishment, were believed to contribute much towards prolonging the epidemic character of the disease; in consequence of which, the Sanatory Committee were obliged to close these establishments until the disease ceased to exist among us.

Owing to the alarming extent of the malady in this part of the city, the Sanatory Committee were compelled to establish additional hopitals for the reception of cholera patients. Accordingly, on the 15th of July they opened the public school house No. 17, in Thirteenth street, as a cholera hospital, and placed the same in charge of Dr. O. P. Wells, and on the 27th of July, they established another in public school house No. 16, in Thirty-fifth

street, and appointed Dr. WILLIAM P. BUEL as physician to the same. They also passed a resolution appointing two physicians for each of the Eleventh, Twelfth, Thirteenth, Sixteenth, Seventeenth and Eighteenth Wards, whose duty should be to visit from house to house, and ascertain their sanatory condition, and the health of their inmates, and report from time to time the result of their investigations to the Sanatory Committee. To effect this important measure, the following named gentlemen were appointed:

Eleventh Ward, J. J. MEIGS, M.D., C. B. DAYTON, M. D.
Twelfth Ward, LEWIS A. SAYRE, M. D., JAMES BATHGATE, M. D.
Thirteenth Ward, THOMAS F. ROCHESTER, M. D., GEO. W. IVES, M. D.
Sixteenth Ward, JOHN W. HUBBELL, M. D., JOHN S. CARTER, M. D.
Seventeenth Ward, JAMES MANLEY, M. D., H. F. QUACKENBOS, M. D.
Eighteenth Ward, D. J. CARROLL, M. D., HENRY E. COX, M. D.

On the 26th of August the cholera had assumed an epidemic type, and extended from the localities in the upper part of Cherry and Water streets, over a large proportion of the north-eastern part of the city, taking in the Seventh, Thirteenth, Tenth and Eleventh Wards, in consequence of which it was deemed necessary to establish a hospital in public school house No. 5, on the corner of Stanton and Sheriff streets. This place was opened on the 2d of July, and Dr. ISAAC GREENE was appointed to superintend the same, and closed on the 17th of September.

The disease, while it raged with unabated violence in the upper Wards, was rapidly declining in most of the lower Wards of the city, and although this fact was generally noticed in the newspapers of the day, yet it was thought advisable by the Sanatory Committee to authorize the Resident Physician, at the earliest opportunity, to make an official announcement to the business public that they need apprehend no danger in visiting the business portion of our city. In accordance with these instructions, he published the following card:

TO THE BUSINESS PUBLIC.—As the season of the year is fast approaching when it will become necessary for merchants and travellers to leave their several homes in the different sections of the United States, with a view to the various objects which trade and other pursuits require, and believing many who contemplate visiting our city may be prevented from so doing by fear, and the many exaggerated reports which may have attained currency in the country concerning cholera in New York, I deem it due to the cause of truth and the business interests of our city, to counteract the evil tendency of such rumors by a statement of facts, obtained through the politeness of Dr. WHITE, the City Inspector, which, by the following statistics, shows the number of deaths as having occurred by cholera during the last week, in the business portion of our city, which comprises the First, Second, Third, Fourth and Fifth Wards, are as follows:

In the First Ward there have occurred, from the 11th to the 18th of August, eight deaths by cholera; in the Second, two, and in the Third, two. In the Fourth, Sixth and Fourteenth, thirty-five, thirty of which were in the William street hospital; in the Fifth, twelve. Total, fifty-nine.

The disease, most unquestionably, reached its culminating point about July 21, since which time (as shown by the weekly reports

of the City Inspector, and the subsequent reports of the Sanatory Committee,) it has been rapidly declining.

I am, therefore, of the opinion that merchants and others who desire to visit our city, may do so with impunity, as I believe the business portion of the same to be as free from the epidemic as any other part of our Union.

SETH GEER, M. D., *Resident Physician.*

The cholera declined with great rapidity about the latter part of August, and continued to diminish until the 6th of September, when it was thought advisable to discontinue making daily reports to the public.

As a precautionary measure, however, the Thirteenth street hospital remained open to the eleventh of September; the Stanton street, to the seventeenth, and the Centre street until the ninth of October.

The duration of the disease, with the exception of isolated cases introduced into the city from emigrant vessels, was from May 10th to October 1st, making a total of one hundred and forty-three days.

The number of persons admitted into the five hospitals were 1901. Males, 985; females, 916. Deaths, 1021; cured, 880. Per centage of cures, 46.29; deaths, 53.71.

For the characters and habits of the patients, the stages of the disease in which they were received, and the general treatment of the sick, I refer the Sanatory Committee to the registers, and the communications of the able gentlemen in charge of the several institutions.

The following table exhibits the weekly mortality in the city of New York by cholera, and that from bowel complaints, during the past twenty-two weeks (May 20th to October 14th), and also thesame during the corresponding periods of last year.

1849. WEEK ENDING	Total mortality.	Cholera asphyxia.	Cholera infantum.	Cholera morbus.	Diarrhœa.	Dysentery.	Inflammation of the stomach and bowels.	Other diseases of stomach and bowels.	Mortality from bowel complaint.
May 26	365		2		3	3	11	2	19
June 2	257		2		4	2	6		14
" 9	228				2	1	9		12
" 16	245				3	4	8	1	16
" 23	260			2	1	4	7	3	15
" 30	283				3	2	5	1	11
July 7	253				2	3	4	1	10
" 14	269				1	2	7	1	11
" 21	324		2		4	3	14	2	25
" 28	403		2		1	4	16	1	24
Aug. 4	386		2		3	2	16	2	25
" 11	337		1		2	4	18	1	26
" 18	311				1	3	20	2	26
" 25	269				2	1	13	2	18
Sept. 1	227				1	2	11	1	15
" 8	257				2	3	13	1	19
" 15	271				1	2	14	1	18
" 22	286				1	1	8	1	11
" 29	226					1	9		10
Oct. 6	269						6		6
" 13	282						8		8
Totals	6262		27	2	43	47	220	23	362

1848. WEEK ENDING	Total mortality.	Cholera asphyxia.	Cholera infantum.	Cholera morbus.	Diarrhœa.	Dysentery.	Inflammation of the stomach and bowels.	Other diseases of stomach and bowels.	Mortality from bowel complaint.
May 27	322		2		3	3	11	2	21
June 3	270		2		4	2	6		14
" 10	269				2	1	9		12
" 17	425				3	4	8	1	16
" 24	472			2	1	4	7	3	17
July 1	512				3	2	5	1	11
" 8	484				2	3	4	1	10
" 15	714		2		4	3	14	2	25
" 22	930		2		1	4	16	1	24
" 29	1020		1		2	4	18	1	26
Aug. 5	1128		2		3	2	16	2	25
" 12	1111		1		2	4	18	1	26
" 19	1030				1	3	20	2	26
" 26	969				2	1	13	2	18
Sept. 2	749				1	2	11	1	15
" 9	530				2	3	13	1	19
" 16	375				1	2	14	1	18
" 23	366					1	8		9
" 30	323						9		9
Oct. 7	269						8		8
" 14									
Totals	15,219		14	2	43	47	220	23	349

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June 2	257		2		4	2	6		14
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" 16	245				3	4	8	1	16
" 23	260			2	1	4	7	3	17
" 30	283				3	2	5	1	11
July 7	253				2	3	4	1	10
" 14	269				1	2	7	1	11
" 21	324		2		4	3	14	2	25
" 28	403		2		1	4	16	1	24
Aug. 4	386		2		3	2	16	2	25
" 11	337		1		2	4	18	1	26
" 18	311				1	3	20	2	26
" 25	269				2	1	13	2	18
Sept. 1	227				1	2	11	1	15
" 8	257				2	3	13	1	19
" 15	271				1	2	14	1	18
" 22	286				1	1	8	1	11
" 29	226					1	9		10
Oct. 6	269						6		6
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" 29	226					1	9		10
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" 13	282						8		8
Totals	6262		27	2	43	47	220	23	362

In concluding this general historical outline of the cholera, as it has recently appeared among us, I take occasion to remark upon the faithful and untiring devotion which the Sanatory Committee and my medical *confreres* have evinced in the discharge of the very responsible duties imposed upon them, and finally tender to them my sincere acknowledgments for the uniform confidence and courtesy extended to me during the period of our official connection.

SETH GEER, M. D.,
Resident Physician.

New York, September, 1849.

NO. II.

REPORT OF PROFESSOR W. H. ELLET,

On the analysis of the atmosphere, in the City of New York, during the Cholera in 1849.

The Honorable the Sanatory Committee
of the Board of Health :

GENTLEMEN :—I have the honor to report to you the result of the experiments in which I have recently been engaged on the atmosphere of this city. The object which I had in view was to determine whether the air contained in due proportion those substances which constitute a normal atmosphere, and more especially to ascertain whether any foreign matter could be discovered which might be looked upon as the cause of the prevailing epidemic.

The observations of Dr. PROUT, made in London in the year 1832, seemed to indicate the presence of a foreign body in the atmosphere at that time and place—presenting itself simultaneously with the appearance, and continuing throughout the duration of the cholera in that year. Assuming the correctness of Dr. PROUT's experiments, and that the existence of the same disease amongst us at the present time would be probably attended with a similar change in the condition of the atmosphere, it seemed desirable to institute rigorous experiments upon the subject. I

therefore determined to submit large masses of air to such processes as would be calculated to insulate from it, and present in a separate form, or in new states of combination, any foreign body which it might contain. It was evident from the wide extension of the epidemic, that its cause—supposing it to be atmospheric—was to be sought in the general atmosphere of the city, rather than in those limited localities where its high degree of virulence indicated more of *exciting* than of *predisposing* agencies.

This consideration, together with others of personal convenience, induced me to select as the place of experiment, the laboratory of the College of Physicians and Surgeons, in Crosby, near Spring street. Here Prof. TORREY, presented me many facilities which I could not elsewhere have enjoyed. The situation is a central one in the city; there is nothing in the neighborhood tending to contaminate the atmosphere, and but few cases of cholera have occurred in its immediate vicinity.

Two sets of experiments were commenced on the 11th day of July. The design of the first was to separate by a cold of 32°F. whatever substances might be condensible from the air at that temperature; and of the other to subject it in considerable masses to various chemical agencies suitably selected.

Although the results of these experiments have been entirely *negative*—indicating so far as they go, the presence of no foreign matter in the atmosphere which could be regarded as the source of disease, I deem it proper to describe the modes by which they have been reached, if for no other purpose than the information of future investigators who may engage in a similar research.

The arrangement adopted for obtaining condensable products from the air, though novel, was simple and efficacious. A tube of large bore coming from the external air, at a height of about five feet from the surface of the ground, was connected with one of the necks of a large Wolfe's bottle—a similar tube passing from the other neck, being tightly fitted into an aperture in the flue of an air furnace, having a good draught. It was found that with this arrangement, when a moderate fire was kindred in the furnace, a powerful draught of the external air was *solicited* through the apparatus, and continued active for at least twenty-four hours after the fire had burned out. The Wolfe's bottle was kept constantly surrounded by ice; a fire was built every morning in the furnace; and the operation was continued day and night for nearly a fortnight.

Large quantities of liquid matter were of course condensed; and these were drawn off from time to time, and submitted to chemical examination. The liquid was found to have the sensible qualities of a richly aerated water. After allowing the minute quantity of dust which had been mechanically introduced with the current of air, to subside, it was perfectly transparent, and distitute of taste and odor. It exhibited no decided acid or alkaline reaction to test-papers, although more delicate reagents showed the presence of carbonic acid. Traces also of chlorine—probably in the state of chloride of sodium, which is generally present in the atmosphere near the sea—and of ammonia, were also detected; but their quantities were too small for accurate determination with the means at my disposal.

Portions of the clear liquid were also tested with suitable reagents, for the purpose of determining the presence of organic matter. This was found, but in quantities apparently not greater than is usual in rain water that has fallen through the atmosphere of a city in warm weather.

Both the clear liquid, and its sedimentary deposit were subjected to rigorous microscopic examination, both by myself and by Mr. Frey, whose expertness in such researches is well known; but no appearance of organized beings, either animal or vegetable was observed.

For the purpose of determining whether any foreign substance could be separated from the air by purely chemical agents, I employed as an aspirator for drawing it through them, a gas holder containing about sixty gallons. The aspirator having been filled previous to each experiment with water, and the discharge of the latter being so adjusted as to occupy from twenty-four to thirty-six hours, a current of air was slowly drawn through the liquids employed, which were contained in a series of Wolfe's bottles, arranged with delivering tubes drawn to capillary orifices.

The liquids through which the air was passed in the successive experiments, were solutions of iodide of potassium in starch water; of basic, neutral and acid acetates of lead; of nitrate of lead; of nitrate of silver; of baryta; of potassa; of hydrochloric acid; and of the chlorides of gold and of platinum. Without entering into unnecessary detail, I may state that these experiments afforded results of the same general character with those obtained by the examination of the liquid procured by refrigeration. Hydrosulphuric acid could not be detected. Carbonic acid, chlorine, ammonia, and organic matter were found in varying, but never in sufficient quantities to justify a suspicion that they could affect the salubrity of the atmosphere which contained them.

Repeated eudiometrical analysis of the air were also performed by means of an apparatus capable of affording much more accurate results than the instruments generally employed for the purpose. The average of twelve experiments gave in the hundred parts by measure, 20.421 parts of oxygen, and 79.579 of nitrogen. These numbers approximate very closely to those obtained some years since by Dr. Hare, at Philadelphia, and indicate no departure from the normal constitution of the atmosphere.

At an early period of the prevalence of the epidemic, the public attention was much directed to a peculiar principle called "ozone," which was asserted to be present in the air, and to be a probable cause of the pestilence. Several persons exhibited the results of experiments which they considered as proving its presence; and certain chemical relations supposed to characterize it were made the basis of modes of treating the disease, which were announced as having proved eminently successful. I deemed it therefore my duty to institute numerous and cautious experiments to ascertain whether there was any substance present in the air capable of producing the chemical effects attributed to "ozone." In the course of this investigation, whose details are foreign to the object of this report, but will be made public through some scientific journal, I was forced to the conclusion not only that no such

peculiar principle or condition existed in the atmosphere at the time; but that the experiments of those European chemists who have announced the production by artificial means, of such a new form of matter, or such a modified or "allotropic" condition of any of those forms previously known to us, are unsatisfactory.

I have the honor to be,
Gentlemen, very respectfully
Your obedient servant,

WILLIAM H. ELLET.

New York, Sept. 18, 1849.

No III.

CENTRE and THIRTY-FIFTH STREET HOSPITALS.

No. 1.—CENTRE STREET HOSPITAL.

NEW YORK, 26th September, 1849.

To the Sanatory Committee of the Board of Health:

GENTLEMEN:—The undersigned, late Physician to the CENTRE STREET AND THIRTY-FIFTH STREET CHOLERA HOSPITALS, has the honor to submit the following

REPORT:

That on the 17th day of May, 1849, it was ascertained beyond question, that the disease which had for several days existed in the rear of No. 20 Orange street, was none other than the ASIATIC CHOLERA. Seven patients were on that day removed thence, by order of the Board of Health, to the building, 127 Anthony street. This building was a kind of store-house, or packing-house for meat, entirely unfit for hospital purposes, and used as a mere temporary receptacle. Four of the seven patients died on the night of the 17th. The three survivors were removed on the 18th, to the building afterwards known as the CENTRE STREET CHOLERA HOSPITAL.

This building, commonly designated by the name of Monroe Hall, was occupied as a public house or tavern. It stands on the north-west corner of Centre and Pearl

streets, in the Sixth Ward of the City of New York. It has two fronts, one of sixty feet, on Centre street, another of forty feet, on Pearl street. The Centre street front looks towards the east—the Pearl street front towards the south. The ground on which it stands is low, being a part of what was formerly known as the Collect. From Broadway, which runs parallel to Centre street, and about two hundred yards further west, there is a gradual descent towards Centre street, of thirty or forty feet. The "Five Points" lie in a north-easterly direction, distant two hundred yards. No. 20 Orange street, the starting point of the epidemic, is within fifty feet of the "Five Points."

Monroe Hall is a building of three stories, besides an attic and basement story. The basement was occupied as an eating house and refectory. The first floor was a porter house or tavern. The second floor was fitted up as a kind of theatre, and used for exhibitions of "Model Artists," and the like. The third floor was a drill room for military companies. The attic was divided into small apartments, which were occupied as tenements, by a number of families. These families, as may be supposed, were not long, after the first patients were brought, in effecting their escape from the premises, with their moveable effects.

The third floor was the only portion of the building which, for the first thirty days, was occupied for the reception and treatment of patients. The apartments in the attic were used for culinary purposes, and for eating and lodging rooms for the attendants.

In proceeding to give, in some detail, the numerical results of treatment in Centre street hospital, I deem it

proper to consider them in two periods. During the first, the patients were all confined to the third floor. During the second, they enjoyed the benefit consequent upon the more spacious accommodations obtained by the opening of the second floor.

The whole number of names entered upon the Register of Centre street hospital, while under my care, is four hundred and ninety-two. Of these, two were dead when brought in. Twenty-one others were dying. Deducting these, there are left four hundred and sixty-nine persons who were subjects of treatment.

First period, commencing 17*th of May, ending* 16*th of June*, 30 *days.*

Admissions	159
Deaths	97 or 61.9 per cent.
Cures	62 " 38.1 "

Second period, commencing 16*th June, ending* 5*th August*, 52 *days.*

Admissions	310
Deaths	146 or 47.1 per cent.
Cures	164 " 52.9 "

Showing a difference of about fifteen per cent. in favor of the second period—leading us to the fair inference, that as the medical treatment was precisely the same, about fourteen or fifteen in the hundred fell a sacrifice to the crowded and insufficient accommodations of the first period.

The following is a statement of the comparative mortality, in *each stage* of the disease, during the two periods:

First period—first stage—Deaths 19 pr. ct.—Cures 81 pr. ct.
Second " — " " — " 16 " — " 84 "
First " —collapse stage—Deaths 82.4 per cent.
" " " " —Cures 17.6 " "
Second " " " —Deaths 65.6 " "
" " " " —Cures 34.4 " "

Showing a greater difference in the collapse, or most severe stages, than in the milder ones of the first stage.

First period—partial collapse—Deaths 48.4 per cent.
" " " " Cures 51.6 " "
Second " " " Deaths 43.0 " "
" " " " Cures 57.0 " "

Showing still a decided difference in favor of the second period.

A comparison of the results of treatment in the different stages, shows very forcibly the importance of prompt attention to the first symptoms. Thus, in the *first* stage of the disease, the *cures* range from eighty-one to eighty-four per cent., the *deaths* from sixteen to nineteen, while in the *collapse* stage the *cures* range from seventeen to thirty-four per cent., the *deaths* from sixty-five to eighty-two per cent.

The following is a statement of the number admitted in each stage of the disease, out of the whole number of patients:

First stage.....122
Partial collapse.....146
Complete collapse.....195
Moribund.....21
Consecutive fever.....6

SEX OF PATIENTS.

Males.....234 | Females 258

Average duration of previous diarrhœa, in four hundred and sixty-nine cases—three days and six hours.

Average time in hospital, in four hundred and eighty-three cases—two days and eight hours.

Table of ages of patients, and comparative mortality.

AGE.	DIED	CURED	TOTAL
Birth to one year	2	.....	2
One year to five years	6	7	13
Five years to ten years	3	3	6
Ten years to twenty years	15	17	32
Twenty years to thirty years	67	69	136
Thirty years to forty years	80	63	143
Forty years to fifty years	66	35	101
Fifty years to sixty years	22	18	40
Sixty years to seventy years	6	4	10

OCCUPATIONS IN 224 CASES:

Blacksmiths	8	Oystermen	2
Boatmen	5	Oakum pickers	2
Butchers	2	Players	2
Bookbinder	1	Painters	6
Barber	1	Pressmaker	1
Carpenters	6	Paper hanger	1
Clerks	3	Pedlars	2
Cooks	5	Quarryman	1
Cartmen	2	Shoemaker	1
Cabinet makers	2	Sailors	28
Cap maker	1	Servants	26
Coachman	1	Ship carpenters	6

Druggist	1	Sail makers	1
Engineers	3	Seamstresses	4
Farmers	2	Stone cutters	3
Foundryman	1	Tailors	5
Glazier	1	Tin smiths	6
Hatters	2	Teachers	3
Hostler	1	Tailoresses	2
Laborers	51	Watch makers	2
Land agent	1	Washerwomen	6
Lawyer	1	Wool picker	1
Masons	4	White washer	1
Machinists	2	Weaver	1
Nurses	4		
			224

NATIVITIES IN 457 CASES:

England	30	France	2
Germany	27	British Provinces	4
Ireland	252	Sweden	1
Scotland	5	Poland	1
United States	135		
			457

HABITS IN 269 CASES:

Temperate	80	Intemperate	151
Habitual Drinkers	38		
			269

No. 2.—THIRTY-FIFTH STREET HOSPITAL.

During a portion of the epidemic, the disease raged with fearful mortality in the Sixteenth Ward, lying in the north-west section of the city, between Fifth avenue and the North river. On the 27th July, the undersigned received orders from the Sanatory Committee to open an additional hospital in the public school in Thirty-fifth street, between Eighth and Ninth avenues, and to transfer thither, as soon as possible, the medical staff, nurses, attendants, and hospital equipage of Centre street hospital. This was effected without delay, and on the 29th of July five patients were admitted.

The public school in Thirty-fifth street has an elevated and airy position, overlooking the adjacent portions of the city. The neighborhood is as yet rather sparsely populated. There are a great many pits and pools of stagnant water, from which, and from the bone-boiling and similar establishments, very offensive effluvia emanate. Numerous rag pickers and bone collectors, principally German, inhabit this section of the city. The personal and domestic habits of these people, are well known to be filthy in the extreme. There is also a numerous Irish population. These two classes furnished nearly all the patients.

The force of the epidemic had considerably abated before the hospital was opened.

The whole number treated was 119—of whom

Died	65	Males	66
Recovered	54	Females	53

Admissions in first stage,.....42
" partial collapse,.....29
" complete " 41
" consecutive fever,..... 7—119

Of those admitted in first stage...Died, 7, or 16.6 per ct.
Cured, 35, or 83.4 "

Of those in partial collapse.....Died, 16, or 55.6 "
Cured, 13, or 44.4 "

Of those in complete collapse....Died, 36, or 87.8 "
Cured, 5, or 12.2 "

Of those in consecutive fever....Died, 5, or 71.5 "
Cured, 2, or 28.5 "

Table of ages of patients, with the mortality of each age.

AGE.	DIED.	CURED	TOTAL
Birth to one year	.....	.....	.....
One year to five years	3	4	7
Five years to ten years	5	6	11
Ten years to twenty years	4	8	12
Twenty years to thirty years	15	15	60
Thirty years to forty years	22	15	37
Forty years to fifty years	9	2	11
Fifty years to sixty years	3	3	6
Sixty years to seventy years	3	.....	3
Seventy years to eighty years	1	.....	1

OCCUPATION.

Artist 1
Butcher 1
Barber 1
Boatman 1
Baker.....1
Carpenter 1
File-cutter..... 1
Gardiner 1
Grocer 1
Laborer 1
Mason 1
Nurses 2

Cooks.....3
Cooper.....1
Clerk 1
Cap-maker 1
Cartmen 5
Cabinet-maker 1
Ciger-maker 1
Confectioner.....1
Dyer.....1
Painters 2
Porters 2
Pedler 1
Plasterer 1
Stone-cutter 1
Servants 79
Tailor..... 1
Washerwomen 4
Weavers..... 2

NATIVITY.

United States 21
England 8
Germany.....26
Ireland 34
Scotland 1
British Provinces 2
Norway 1
France..... 1

CONCLUDING REMARKS AND OBSERVATIONS.

1. Character and description of the disease.

No points of difference were observed between the present and former epidemics of cholera in this city—nor between it and the recorded description of the disease in other places. The well known diagnostic symptoms of Asiatic cholera were fully and strongly developed. The vomiting and purging of the rice color fluid, the hoarse whispering voice, cramps, cyanosis, asphyxia, and suppression of all the secretions, which marked the great majority of the cases, pronounced in unmistakable language the character of the disease. During the earlier stages of the epidemic, there were not wanting those who questioned its identity with the Asiatic cholera; or at least imagined that it existed in a modified and milder form. Long before its close, it is believed that the most skeptical ceased to entertain any doubt on the subject.

2. Contagious or non-contagious character of the disease.

That the cholera of the present season originated, *de novo*, at 20 Orange street, scarcely admits a question. No connection, mediate or immediate, by persons or things, has been traced or alledged to exist between the disease prevailing at 20 Orange street and any similar disease, in any other locality. In that place all the predisposing causes were existing in the utmost intensity, and on that

spot the epidemic selected its first victims. But did the subsequent history of the disease exhibit any evidence of contagion? The writer of this report answers unhesitatingly in the negative. The convictions of his own mind, deduced from a close and impartial observation of facts bearing on this point, are complete. The admitted fact that a number of nurses and attendants in the hospitals under his charge, contracted the disease, and several fell victims to its violence, does not impair in the least degree the force of this conviction. These persons, in common with the whole population, were under the full operation of the epidemic influence. Superadded to this, they were subjected to many of the strongest predisposing causes. The great majority were intemperate persons. Add to this the laborious and exhausting nature of their duties—being on watch the whole of every second night—and it can, as it seems to me, be a matter of no surprise, nor require the supposition of contagion, that eight or ten, out of forty or fifty persons, employed during the epidemic, should have contracted the disease. Of eight individuals, who were at different times attached to the medical staff of the two hospitals, not one suffered an attack. The most of us enjoyed uninterrupted health from first to last.

3. Therapeutic treatment.

Entertaining the strong conviction, that the profuse and exhausting diarrhœa is the essential feature of the disease, upon which all the concurrent and subsequent phenomena, directly or indirectly hinge, our indications of cure were deduced from this conviction—our first efforts directed to arrest the evacuations. In our efforts to accomplish this point, we did not lose sight of the co-existent fact, that all

the important secretions of the system were arrested—all the great secretory organs smitten, as it were, with paralysis. With remedies to check the diarrhœa, were combined those calculated to restore the secretions. These are the two indications of cure.

To accomplish the first, opium, in its different forms, is probably the most efficient single remedy. It was administered both by the mouth and rectum, and with great efficiency and success. It is, however, obnoxious to the objection, that while it checks diarrhœa more effectually than any other remedy, it also impedes the restoration of the secretions. Despite this objection, I regard its use as indispensable. Combined with opium, and to subserve a similar end, camphor was extensively administered, and with excellent results. The solution of camphor in chloroform, affords a happy and efficient mode of administration—superior, it is believed, in some respects, to any other form.

In addition to these two remedies, and in combination with them, the vegetable and mineral astringents, of every form and variety, were extensively used.

Tannin, pure, or in the various drugs of which it constitutes the active principle—acetate of lead, nitrate of silver, sulphates of zinc, alumina and copper, were all used, and with happy effects. The combination of opium and acetate of lead, swallowed, or thrown into the rectum, forms a remedy of singular, and, I think, unsurpassed efficiency.

To accomplish the other leading indications, viz: to restore the suppressed secretions, calomel was, in the great

majority of cases, administered, in combination with opium and astringents.

In doses of from one to five grs., (the latter dose not often repeated,) it is believed to have been efficacious. The blue pill was sometimes substituted, or alternated, with similar results, but with perhaps less efficacy.

In the early part of the epidemic, we placed a sufficient number of patients to afford, as was thought, a fair test, under treatment, with large doses, say thirty grs. of calomel, uncombined with opium. In our hands this treatment proved an entire failure. The progress of the symptoms did not appear in the slightest degree to be arrested, or even retarded. The treatment was therefore abandoned.

In addition to the use of mercury, designed principally to act on the biliary secretion, remedies were also directed to restore the action of the kidneys and the flow of the urine. The two principally relied on were the sesquichlorid (muriated tincture) of iron, and the sweet spirits of nitre. They certainly appeared to act with much success in restoring the urinary secretion, and giving relief to the brain in the secondary or consecutive form of the disease. The tinct. mur. fer. was administered in doses of fifteen to twenty drops, combined or not with half teaspoonful doses of spirits nitre, and repeated every two or four hours.

If the diarrhœa which ushers in the attack, has continued unchecked for a period of time, more or less extensive, varying in different individuals, from less than an hour to many hours, or even days, we arrive at that stage of the disease, and that peculiar condition, to

which the term collapse has been applied. Collapse may be *partial* or it may be *complete*.

It has been seen in the former part of this report, that a considerable majority of all the cases treated were in partial or complete collapse. It has also been observed that a very large proportion had a fatal result.

What is the condition of a patient in collapse, and what the indications of cure? The blood of such a patient has, by the profuse serous, and watery evacuations, been almost deprived of its fluidity, and rendered incapable of circulation in the minute vessels. It has become loaded with urea, carbonaceous matters, and other materials, which in a state of health are constantly climinated, and whose removal is indispensable. The vital energy of the system has become exhausted by the profuse discharges, and the violence of the spasmodic muscular action. The animal heat, ordinarily produced by the oxygenation of the blood, has ceased to be furnished; the patient cold and pulseless, is almost the same as a corpse.

The indications plainly are to restore to the system vital energy, warmth, and fluidity, to return to the blood that of which it has been robbed. The means to accomplish these are unhappily little in our power. Dr. Stevens, of St. Thomas, aimed to do so by administering his saline solution. Unfortunately it cannot pass into the circulation, because the stomach instantly rejects it. The injection of the saline fluid immediately into the veins has met with no better success. We cannot do the work of nature's laboratory by any such bungling substitutes. We attempt to restore the vital warmth by the application of heat in

various ways to the surface, and to the extremities. Vain attempt! As well might we hope to warm a frozen lake by a fire kindled upon the ice which covers its surface.

We have another object to accomplish, in this as in all the stages of the disease: to alleviate by every proper means the patient's sufferings, which in collapse are often excruciating, is an indispensable duty. The cramps and spasms continue with fearful violence and severity. The thirst is often tormenting and agonizing. The anasthasia produced by the chloroform inhalation sometimes comes in here very happily, and seldom fails to furnish the patient some temporary relief to his sufferings; that, however, is all.

The attempt to arouse the vital energy by diffusible stimuli, alcohol and ammonia, was generally made, but with very little avail. These agents may goad on the circulation for a while, but they contribute nothing towards the restoration of health. They even do harm, by loading the blood, already too highly carbonated, with still more of carbonaceous material. In the case of inebriates, who frequently came in after a debauch of several days, with their blood and brains loaded with alcohol, these remedies appeared to do actual injury, and to hasten the fatal termination.

In the remarks made upon the futility, in a majority of cases of collapse, of the attempts to restore warmth to the system, by the application of external and artificial heat, it is not intended to throw any discredit upon the propriety and frequent utility of this remedy.

In the earlier stages of the disease warm applications are of great utility, and most comforting to the patient. A variety of methods were tried, but the dry heat produced by the application of warm water in india rubber bottles and bags was found the most convenient and manageable, and most agreeable to the patients.

The cholera patient who survives the first stage of the disease, and lives through the dangers of collapse, has yet to encounter other perils, perhaps quite as formidable. The SECONDARY or CONSECUTIVE FEVER often lies between him and restoration to health. The symptoms of this stage assume a variety of forms. Sometimes they are of a typhous character. Not unfrequently they bear a marked resemblance to the congestive fevers of the Southern States. The pathological condition appears to depend, in a great measure, upon the depraved condition of the blood, produced by the causes already described. Strongly marked cerebral congestion was one of the most frequent accompaniments.

The *treatment* consisted in the application of cups and blisters to the temples, and back of the neck. Counter irritants to the extremities. Internally, mercurials and diuretics were administered with good effects, to restore the bilious and urinary secretions.

This stage of the disease, as indeed was true of every other stage, proved most fatal to those who had been addicted to the free use of alcoholic stimulants. This class, if they survived the earlier stages, very uniformly ran into consecutive fever, and most generally succumbed under it.

4. POST MORTEM APPEARANCES.

Numerous examinations were made of every stage of the disease. A detailed description of the post mortem appearances will not be expected on the present occasion.

It is sufficient to observe, briefly, that those dead in collapse presented an engorged condition of the abdominal viscera, and the mucous surfaces. The vessels were distended, as though all the blood of the body had rushed in this direction.

Those who died in the consecutive fever, exhibited strongly marked appearances of congestion of the brain and spinal marrow, with more or less effusion into the different cavities.

In many rabid cases, the examination revealed very slight departures from a perfectly healthy condition, and would have rendered it very difficult, from a mere inspection of the post mortem appearances, to form even a conjecture as to the cause of death.

The undersigned cannot conclude this report, without rendering a merited tribute to those gentlemen who were associated with him in the medical staff of the hospitals, and who ably and zealously seconded all his efforts. To Messrs. H. N. WHITTELSEY and THOMAS M. FRANKLIN, First Assistant Physicians, and to Messrs. EDWIN B. STIMSON, H. M. LYONS and CHARLES KENWORTHY, Second Assistants, his thanks are sincerely tendered.

Respectfully submitted,

WILLIAM P. BUEL, M. D.,
Late Physician Centre street and Thirty-fifth street Cholera Hospitals.

NO. IV.

WILLIAM AND CENTRE STREET HOSPITALS.

No. 1.—WILLIAM STREET.

NEW YORK, October 1st, 1849.

To the Sanatory Committee :

GENTLEMEN :—The hospital in William street, Public School house No. 1, was opened to the reception of patients on the 9th of June, and was closed on the 27th of August. During that time, 712 were admitted; in June 148, in July 333, in August 231. Of this number, 390 were males, and 322 were females. The following tables exhibit the number of persons received, deceased, and discharged cured; the disease and condition; the sex; the occupation; the age; the residence; the nativity; the color, and per centage of each.

STAGE AND CONDITION.	ADMITTED.		DIED.		DISCHARG.		TOTAL.			PER CENTAGE.	
	MALE	FEM.	MALE	FEM.	MALE	FEM.	ADM.	DIED.	DISC.	DIED.	DISC.
First Stage.	73	58	2	4	71	52	129	6	123	4.65	95.35
Par. Col...	132	111	50	40	82	71	243	90	153	37.04	62.96
Collapse....	141	124	133	114	9	9	265	247	18	93.20	6.80
Dead & dy'g	31	16	—	—	—	—	47	47	—	100.00	0.00
Other dis.	13	15	8	10	5	5	28	18	10	64.38	35.62
	390	322	193	168	167	137	712	408	304	57.30	42.70

Whole number of cases of cholera 637, of which 343 died, 195, independent of the dead and dying, and other

diseases, within twelve hours after admission. Per centage 53.84. Exclusive of the 265 collapsed or pulseless patients, per centage 25.80. With premonitory diarrhœa there were 514; without it 50; unknown 73.

It may be well to add that 35 cases are included under the head of cholera which reacted and died with delirium tremens and consecutive fever. In all, the habits of the persons were intemperate, and their constitutions very much impaired by long continued dissipation.

OCCUPATION OF MALES.		pr. ct.	OCCUPATION OF MALES.		pr. ct.
Laborers	136	34.87	Musician	1	0.25
Mariners	67	17.18	Clockmaker	1	0.25
Clerks	14	3.59	Sashmaker	1	0.25
Shoemakers	8	2.05	Painter	1	0.25
Carpenters	11	2.82	Broker	1	0.25
Brickmasons	10	2.56	Tanner	1	0.25
Farmers	8	2.05	Locksmith	1	0.25
Porters	5	1.28	Fisherman	1	0.25
Mechanics	5	1.28	Weaver	1	0.25
Blacksmiths	5	1.28	Tracklayer	1	0.25
Tailors	5	1.28	Umbrella maker	1	0.25
Cartmen	4	1.02	Ropemaker	1	0.25
Printers	3	0.77	Engraver	1	0.25
Pedlars	3	0.77	Bookgilder	1	0.25
Cooks	4	1.02	Bookbinder	1	0.25
Booksellers	2	0.51	Coachmaker	1	0.25
Merchants	2	0.51	Surgeon and Apothecary	1	0.25
Servants	2	0.51	Physician	1	0.25
Barbers	2	0.51	Marketer	1	0.25
Engineers	2	0.51	Saddler	1	0.25
Brass-finishers	2	0.51	Butcher	1	0.25
Bar-keepers	2	0.51	Farrier	1	0.25
Sailmakers	2	0.51	Nurse	1	0.25
Trav. Agents	2	0.51	Tinsmith	1	0.25
Chairmakers	2	0.51	Artf. Toothmaker	1	0.25
Bakers	2	0.51	Goldbeater	1	0.25
Segarmakers	2	0.51	Sugar-refiner	1	0.25
Stewards	2	0.51	Marble-polisher	1	0.25
Stonemasons	2	0.51	School-master	1	0.25
Ropemakers	2	0.51	Piper	1	0.25
Grocer	1	0.25	Coppersmith	1	0.25
Matmaker	1	0.25	Unknown	38	9.74
Hostler	1	0.25	TOTAL	390	

The females (322) were either married or servants, or had no particular avocation.

AGES OF PERSONS WITH CHOLERA.	ADMIT'D.		DIED.		DISCH'D.		TOTAL.			PER CENT.	
	Males.	Fem.	Males.	Fem.	Males.	Fem.	Admt.	Died.	Disch'd	Died.	Disch'd
From 1 to 10 years.....	9	6	3	3	6	3	15	6	9	40.00	60.00
" 10 to 20 "	30	36	11	12	19	24	66	23	43	34.85	65.15
" 20 to 40 "	228	187	120	102	108	85	415	222	193	53.49	46.51
" 40 to 60 "	65	49	45	32	20	17	114	77	37	67.54	32.46
Over 60 "	11	8	7	6	4	2	19	13	6	68.42	31.58
Unknown.....	8	....	2	....	6	....	8	2	6	25.00	75.00
	351	286	188	155	163	131	637	343	294	53.84	46.16

NUMBER FROM EACH WARD.		pr. ct.	NUMBER FROM EACH WARD.		pr. ct.
1st Ward.....	88	12.36	12th Ward.....	7	0.98
2d "	17	2.39	13th "	18	2.53
3d "	10	1.40	14th "	31	4.35
4th "	183	25.70	15th "	2	0.28
5th "	25	3.51	16th "	11	1.54
6th "	60	8.43	17th "	14	1.97
7th "	44	6.18	18th "	16	2.24
8th "	15	2.10	Vessels in port.....	46	6.44
9th "	3	0.42	Strangers.....	44	6.18
10th "	23	3.23	Unknown.....	35	4.91
11th "	30	4.21			
			TOTAL.....	712	

NATIVITY.		pr. ct.	NATIVITY.		pr. ct.
United States.....	122	17.13	Denmark.....	2	0.28
Ireland.....	428	60.11	Sweden.....	5	0.70
England.....	41	5.76	Holland.....	2	0.28
Scotland.....	9	1.26	New Foundland.....	1	0.14
Wales.....	5	0.70	Belgium.....	2	0.28
Germany.....	63	8.85	West Indies.....	3	0.42
France.....	3	0.42	Canada.....	3	0.42
Portugal.....	2	0.28	Nova Scotia.....	3	0.42
Spain.....	2	0.28	Unknown.....	14	1.96
Prussia.....	1	0.14			
Norway.....	1	0.14	TOTAL.....	712	

COLOR.	Males.	Females.	Total.	Per cent.
White.....	379	311	690	96.91
Black.....	11	11	22	3.09
	390	322	712	

The above summary exhibits the statistics of the institution under my charge, as recorded from day to day in the register.

A detail of the treatment is more suited to the pages of a medical journal, than a document like the present. If any person desires it, the minutes of the transactions of the hospital, placed in the possession of the Board of Health, will afford the information. It may not, however, be out of place to say, that, in my judgment, astringents and purgatives, copiously and promptly administered, are infinitely the best remedies. To tannin and calomel, combined with opium and camphor, I give the decided preference. In the curable stages, I do not doubt that the disease, in most instances, will readily yield to these agents. External applications are useful adjuvants, but they possess little power in themselves. The restoration of the secretory organs, especially the liver, to action, and the arrest of the serous evacuations, appear to be the only indications that offer the least chance to preserve the blood in a sufficiently normal state, for the purposes of circulation, and the continuance of life.

Before leaving the subject, I call the attention of the Committee to the large number admitted in the usually *fatal stage of collapse;* to the number of *dead and dying* when received; to the number with other maladies, previously treated, *in very little better condition;* to the number of cases which terminated fatally, at various periods, within the short space of *twelve hours;* to the absence of all knowledge in relation to the history of patients, and the neglect of physicians to communicate in their certificates the remedial agents administered; to the distance that

many were brought, with a disease in which repose and time are so important; and to their own knowledge of the generally degraded and vitiated condition of individuals who become inmates, by necessity, of a transient public establishment.

To Dr. John M. Lawrence, who vacated the valuable appointment of senior physician of the Bellevue Hospital, to assume the perilous and arduous position of assistant physician to a cholera hospital, I am under very great obligations. His talents and knowledge peculiarly fit him for the position, and his zeal and attention to the sick, at all hours of the twenty-four, entitle him to the confidence and approbation of the authorities of the city.

To Mr. Kellock, superintendent of "out door poor," I am extremely indebted for attention in directing the removal and burial of the dead. His disposition to oblige at a moment's notice, kept the hospital entirely free from the effluvia arising from the rapid decomposition of the bodies.

If any neglect has occurred in the police of the institution, or if any individual was deprived of a single article necessary to comfort, or to restoration to health, with discretionary powers placed at my disposal by the Sanatory Committee, the fault was my own.

With very great respect, yours, &c.,

ALEX. F. VACHE.

No. 2.—CENTRE STREET HOSPITAL.

NEW YORK, OCTOBER 9th, 1849.

To the Sanatory Committee:

GENTLEMEN:—Simultaneous with the close of the hospital in William street, by the Sanatory Committee, with the laudable desire to restore the premises at the earliest period, after the usual summer vacation of the pupils, to the Commissioners of Schools; the well adapted and thoroughly ventilated third and fourth story rooms in the building, previously under the charge of Dr. BUEL, at the corner of Centre and Pearl streets, were re-opened for the reception of patients, in order to meet promptly the exigencies that arose from the continued prevalence of the epidemic in the lower portions of the city.

From the 27th of August to the 3d of October, 65 patients were admitted—in August, 19; in September, 45; in October, 1. 36 were males and 29 were females. The tables denote the maladies, etc., of each.

STAGE AND CONDITION.	ADMIT'D.		DIED.		DISCH'GD		TOTAL.			PER CENTAGE.	
	Males.	Fems.	Males.	Fems.	Males.	Fems.	Admt.	Died.	Disc'd	Died.	Disc'd
First stage,.....	8	9	....	....	8	9	17	....	17	.....	100.00
Partial collapse,.....	8	9	3	3	5	6	17	6	11	35.29	64.71
Collapse,.....	10	8	8	7	2	....	18	15	3	83.33	16.67
Dead and dying,.....	4	1	4	1	....	1	5	5	....	100.00	.....
Other Diseases,.....	6	2	4	1	2	1	8	5	3	62.50	37.50
Total,.....	36	29	19	12	17	17	65	31	34	47.69	52.31

Cases of cholera 52, of which 21 died; per centage 40.31. Exclusive of the 18 collapsed or pulseless patients when received; per centage 17.65. Of those included with cholera, who subsequently died with consecutive fever, were males 2, females 3; Total 5. Those who had premonitory diarrhœa were 40; without it, 6; unknown, 6.

AGE.	ADMIT'D.		DIED.		DISCH'D.		TOTAL.			PER CENT.	
	Males.	Fem.	Males.	Fem.	Males.	Fem.	Admit	Died.	Disc'd	Died.	Disc'd
From 1 to 10 years	1	3	1	1	....	2	4	2	2	50.00	50.00
" 10 to 20 "	4	8	1	....	3	8	12	1	11	8.33	91.67
" 20 to 40 "	17	10	10	5	7	5	27	15	12	55.56	44.44
" 40 to 60 "	10	7	4	5	6	2	17	9	8	52.94	47.06
Over 60 "	4	1	3	1	1	....	5	4	1	80.00	20.00
	36	29	19	12	17	17	65	31	34	47.69	52.31

OCCUPATION OF MALES.		pr. ct.	OCCUPATION OF MALES.		pr. ct.
Laborers	6	16.67	Plate maker	1	2.78
Seamen	7	19.44	Tanner	1	2.78
Farmers	2	5.56	Custom House Officer	1	2.78
Coopers	2	5.56	Marble Polisher	1	2.78
Mechanics	2	5.56	Stone Cutter	1	2.78
Printer	1	2.78	Fisherman	1	2.78
Plasterer	1	2.78	Watchman	1	2.78
Bar-keeper	1	2.78	Dyer	1	2.78
Umbrella maker	1	2.78	Engineer	1	2.78
Painter	1	2.78	Unknown	3	8.33
			Total	36	

NUMBER FROM EACH WARD.		pr. ct.	NUMBER FROM EACH WARD.		pr. ct.
1st Ward	10	15.38	14th Ward	2	3.08
2d "	2	3.08	15th "	1	1.54
3d "	4	6.16	16th "	1	1.54
4th "	11	16.92	18th "	1	1.54
5th "	1	1.54	Vessels in port	5	7.69
6th "	15	23.08	Strangers	4	6.16
7th "	1	1.54	Unknown	5	7.69
10th "	1	1.54			
13th "	1	1.54	Total	65	

NATIVITY.		pr. ct.	NATIVITY.		pr. ct.
United States	13	20.00	Germany	4	6.16
Ireland	41	63.00	Portugal	1	1.54
England	5	7.69	Total	65	
Scotland	1	1.54			

COLOR.	Males.	Females.	Total.	Per cent.
White	36	29	65	100
Black	....	....	....	
	36	29	65	

On mature reflection, I have no further remarks to make in reference to the epidemic, than will be found in the report relating to the William street hospital. As a matter of record, it is proper to state, that Dr. Lawrence continued associated with me, and fully confirmed the opinion expressed on that occasion, of his professional ability and personal character.

In surrendering to you the important position entrusted to my charge, and in severing from you in my official capacity, I cordially thank you for the kindness evinced on every occasion. During the entire period of the fearful crisis, your confidence impelled me to the performance of duty, and your responsibility incited the exertion to protect it from unmerited censure. If I have fulfilled your expectations and realized your desires, the greatest point in my ambition has been achieved, and I retire from the appointment perfectly satisfied with myself.

With very great regard and respect,

I remain your obd't serv't,

ALEX. F. VACHE.

NO. V.

THIRTEENTH STREET HOSPITAL.

NEW YORK, Sept. 22d, 1849.

JAMES KELLY, ESQ.,
Chairman of the Sanatory Committee
of the Board of Health.

SIR:—In offering a few observations on the disease that called for the establishment of the Thirteenth street Cholera Hospital, recently under our charge, we would first enumerate the general phenomena of the malady as they presented themselves before us during the epidemic; and that they may be stated with greater precision, we will mention them as they occurred—in the premonitory, incipient, partial and complete collapse stages.

The premonitory stage has received the popular term of cholorine.

Languor and lassitude, occasional nausea and vertigo, with co-existing disorder in the bowels, were the prominent symptoms of this stage.

It was at this time that most of the empirical remedies, which attained any popular renown, were most serviceable.

The incipient stage of cholera was attended by frequent evacuations from the bowels that had lost their fœcal

character, denominated "rice water discharges;" the feeling of debility that attended them was great, and the exhaustion extreme. Taking the disease at this point, subsequent symptoms were often prevented, and life preserved by the appropriate treatment of diarrhœa. After the diarrhœa had existed for a longer or shorter space of time, vomiting of almost colorless fluid, peculiar to cholera, commenced, and with it the period we would designate as the partial collapse.

The vomiting was not always full and effectual, consisting rather of apparently unsatisfactory retchings than a complete discharge of the contents of the stomach; though sometimes the discharges were copious—forcibly and freely expelled—simultaneously with the vomiting, sometimes before cramps took place; and the pain that attended them constituted a great part of the sufferings of the patient, who would beg that friction might be applied to the parts affected. In this stage the pulsation became feeble and frequent; the temperature of the skin diminished; the countenance shrunk, livid, or pallid; and the respiration hurried, occasionally checked by spasms of the diaphragm or intercostal muscles.

After several hours, from four to six, eight or ten, the vomiting and spasms began to subside, by occurring at longer intervals, and with less severity. The partial was then succeeded by the stage of complete collapse, denoted by an almost or quite imperceptible pulse; a cold, moist or wet surface; blueness of the skin, most conspicuous in the face and hands, which wore a shrunk and soddened appearance; a moist and cold tongue; and a voice of mingled huskiness and feebleness.

The sources of suffering were a sense of weight and oppression at the precordia, which induced the patient to implore for fresh air, and to seek relief in changing his position.

During this stage the spasms were generally too feeble to cause much pain. The insensibility of the stomach was great—the most powerful stimulants failing to affect it.

The kidneys, in common with the other glands, suspended their secretory functions.

It was in the above almost hopeless state that a majority of those brought to the Thirteenth street Cholera Hospital were admitted, as may be seen by referring to the annexed tables. Reaction commenced whenever, by the powers of the constitution or the remedies employed, there was a subsidence of the cramps, vomiting and diarrhœa, before complete collapse took place. The patient then became calm and composed; the skin of moderate warmth, though somewhat cooler than natural; the pulse emerged to a condition of moderate force, without much acceleration; and the renal secretions returned. When only these symptoms were manifested, the stage of reaction was the last of the disease and the commencement of convalescence. But as I have remarked, the majority of cases that terminated fatally were admitted in a state of complete collapse, from which, if they reacted, it was not unfrequently to pass on to a fever, whose malignancy was proportioned to the severity of the collapse that preceded it.

This fever has been termed consecutive. It appeared after the patient had remained in collapse a longer or

shorter time. Reaction having apparently commenced, the patient would often remain in nearly the same condition for one or two days, without any very definite indication of the organs affected. Subsequently the vessels of the conjunctiva became distended with blood, especially that portion of it covering the inferior hemisphere of the eye. The pulse was more developed, though not frequent—generally not exceeding ninety—accompanied by a tendency to sleep. As the fever progressed, the eye became more and more injected; the intellect more and more torpid; though the patient could still be roused to answer questions, and even to make one or two sensible remarks about his condition; but the instant the conversation ceased, the eyes were turned up in the orbit, exposing through the half-closed lids the red sclerotica, and he fell again in a state of stupor. The countenance was often flushed, sometimes considerably so, yet the temperature of the surface was below the natural standard. The duration of the disease was from six to ten days, often terminating fatally.

Doctor Cullen, in describing the phenomena of fevers, selected the intermittent form, as embracing all the essential symptoms necessary to be known, to enable any person to form a correct idea of the nature of fevers in general.

We, in like manner, have given a description of cholera, embracing all that is necessary to be known, to enable any person to form a right comprehension of the disease. Although every one familiar with the cholera, or its history, well knows that in many cases the disease is ushered in with symptoms so violent, and runs a course so

rapid, as to make it difficult or impossible to recognize any distinct stages.

With reference to the treatment of cholera pursued at the Thirteenth street Cholera Hospital, we would premise our remarks by expressing the conviction, that with the public, (and perhaps in our own profession,) a specific remedy for the disease is too eagerly sought, to the neglect of modifications required by different localities, different epidemics of the malady, and by different periods of the same epidemic.

In cholera as in other diseases, to be successful, the treatment must be adapted to the varying circumstances of the case before us—its severity, the period of its progress; its peculiar phenomena; the age, habits, constitution and occupation of the patient should be duly considered.

Hence the necessity which arises, in describing our medical management, to do so in general, (and to those wishing to follow the same mode of treatment) perhaps, unsatisfactory terms. The premonitory and incipient stages were treated by nearly the same remedies, differing mostly in the energy of their administration.

We deem it unnecessary to mention all the remedial agents used by us. Those most relied on in the stages above mentioned, were foot baths of a strong infusion of capsicum, with mustard plasters to the stomach and abdomen. External heat applied by us with India rubber bags, filled with hot water, with varied combinations of opium, submur-hyd, or acetate of lead, injuctions composed of solutions of tannin or acetate of lead, combined with tincture of opium, were generally sufficient to check the disease at these periods.

When it had passed to partial collapse, dry friction of the extremities was directed after the capsicum bath. Subsequently an ointment, composed of ung. hyd. two parts, camphor and capsicum, each one part, was thoroughly rubbed in the skin of the extremities, and along the whole length of the spine.

During the recent epidemic, as in those of 1832 and 1834, this ointment seemed to us to be a highly beneficial application from the friction produced in its employment, as well as by its derivative effect, and the absorption that may have taken place of its ingredients. In conjunction with the use of this ointment, some of the remedial agents used in the previous stages, were continued in this stage. Musk and camphor were used at almost every stage of the disease. During the stage of complete collapse, external treatment was mostly relied on, and internal medication directed merely to mitigate the discharges from the stomach and bowels. When these persisted, we did not generally direct internal stimulants at this period, although they seemed so urgently called for. From the cases which we have seen in which brandy, wine and ammonia, &c., &c., were given, we do not believe the chances for rallying from collapse were increased; but we think that those who rally under the use of stimulants are liable to a still more malignant form of consecutive fever than those who react from remedies applied externally. When moderate reaction occurred, it required no special medication.

If consecutive fever supervened, the remedies employed were those applicable to congestive fevers. Blisters to the lower extremities—cupping or leeching the abdomen, chest or head, with such internal remedies as each particular case seemed to require.

To our medical assistants, Messrs. Salter, Hyslop and Wilson F. Bell, M. D., our thanks are due for the faithful manner in which they discharged their duties.

To our First Assistant, E. B. Stimson, M. D., we desire to return our thanks for the able and efficient manner in which he discharged his duties at all times, particularly for the ability and zeal exhibited when he had the entire charge of the hospital during our illness.

It is also due to the Sanatory Committee of the Board of Health, to state that the hospital was furnished with every thing requisite for the comfort and well-being of the patients, and that all our requisitions for things necessary to their welfare, were promptly supplied.

The following tables afford the aggregate number of admissions, deaths and discharges that have occurred at the Thirteenth street cholera hospital, from its organization on the 15th July, to its close on the 11th September, 1849, together with statistics regarding the sex, nativity, age and occupation of those admitted.

Number of admissions,	275
Females,	139
Males,	136
	275

NATIVITY.		AGE.	
Ireland,	161	Between 1 and 10	34
England,	27	" 10 " 20	29
Germany,	22	" 20 " 30	87
Scotland,	4	" 30 " 40	70
France,	4	" 40 " 50	34
West Indies,	3	" 50 " 60	14
Denmark,	1	" 60 " 70	5
United States,	53	" 70 " 80	2
	275		275

HABITS.

Habitual Drinkers,	111
Occasional drinkers,	59
Temperate,	70
Habits unknown,	35
	275

OCCUPATIONS.

Carpenters	5	Shoe makers	6
Seamstresses	18	Farmers	7
Engineers	4	Grocers	2
Brewers	1	School teachers	1
Tailors	4	House servants	47
Wheelwrights	3	Nurses	5
Washerwomen	4	Dock builders	2
Segar makers	2	Blacksmiths	5
Laborers	62	Tailors	6
Stone cutters	11	Paupers	8
Straw pressers	3	Carmen	2
Soap boilers	3	Occupation unknown.	64
		Total	275

STAGE OF DISEASE ON ADMISSION.

Complete collapse	126
Partial Collapse	93
Premonitory symptoms and incipient	20
Reaction	27
In articulo mortis	7
Relapse	2
	275

COLOR.

White	264
Colored	11
	275

RESULTS.

Discharged cured	136
Deaths from cholera	122
Deaths from diseases supervening	17
	275

All of which is respectfully submitted.

OVID P. WELLS.

NO. VI.

STANTON STREET HOSPITAL.

STANTON STREET HOSPITAL.
SEPT. 14, 1849.

To the Board of Health
of the City of New York:

GENTLEMEN:—In compliance with a resolution of your Board, I transmit a synopsis of the practice of the hospital under my charge, during the recent epidemic.

NUMBER OF PATIENTS ADMITTED.

Suffering from diarrhœa	40
In partial collapse or first stage of cholera	40
In stage of collapse	120
In consecutive stage	6
Suffering from dysentery	14
Suffering from pain in the abdomen and cramps in the extremities, under the influence of spirituous liquors	14
Suffering from cholera infantum	4
TOTAL,	238

DIED.

In the stage of collapse	75
" Consecutive stage	23
Of Delirium tremens	5
" Typhoid fever	3
" Bronchitis	2
" Dysentery	3
" Cholera infantum	1—112

DISCHARGED CURED.

Of Diarrhœa	33
" Partial collapse or first stage of cholera	25
" Collapse	18
" Consecutive stage	23
" Cholera infantum	3
" Dysentary	7
" the effects of intemperance	14
" Typhoid fever	3—126
	238

More minute and full statistical information is furnished by the following abstract from the hospital register.

Of the forty patients admitted with diarrhœa, there were

DISCHARGED CURED.		DIED.	
Of Diarrhœa,	33	Of Typhoid Fever,	2
" First stage of cholera,	1	" Delirium Tremens,	1
" Stage of collapse,	1		—
" Typhoid fever,	2		3
	37		

Rate of mortality, 7½ per cent.

Of the forty patients admitted in the first stage of cholera, there were

DISCHARGED CURED.		DIED.	
Of First stage,	24	In stage of collapse,	4
" Stage of collapse,	1	" Consecutive stage,	4
" Consecutive stage,	2	Of Typhoid fever,	1
" Typhoid fever,	1	" Delirium tremens,	3
	28		12

Rate of mortality 30 per cent.

Of one hundred and twenty patients admitted in the stage of collapse of cholera, there were

DISCHARGED CURED.		DIED.	
Of stage of collapse,	16	In stage of collapse,	68
" Consecutive stage,	17	" Consecutive stage,	16
	—	Of Typhoid fever,	2
	33	" Delirium tremens,	1
			87

Rate of mortality, 72½ per cent.

Of six patients admitted in the consecutive stage of cholera, there were

DISCHARGED CURED.		DIED.	
Of consecutive stage,	4	In consecutive stage,	2

Rate of mortality, 33⅓ per cent.

Of fourteen patients admitted with dysentery, there were

DISCHARGED CURED.		DIED.	
Of Dysentery,	7	Of Dysentery,	3
		In collapse stage of cholera,	3
		" consecutive stage "	1
			—

Rate of mortality from Dysentery, $21\frac{5}{14}$ per cent.
" " " Cholera, $28\frac{9}{14}$ " "

50 per cent.

Of fourteen patients suffering from the effects of Intemperance, all were discharged, cured.

Of four children admitted with Cholera infantum, there were

DISCHARGED CURED.		DIED.	
Of Cholera infantum,....	3	Of Cholera infantum,.	1

Rate of mortality 25 per cent.

OF THE PATIENTS WHO DIED IN COLLAPSE.

6	died	within	1	hour	of their admission.
2	"	"	2	"	" "
4	"	"	3	"	" "
4	"	"	4	"	" "
5	"	"	5	"	" "
6	"	"	6	"	" "
7	"	"	7	"	" "
2	"	"	8	"	" "
3	"	"	9	"	" "
2	"	"	10	"	" "
3	"	"	11	"	" "
4	"	"	12	"	" "
1	"	"	13	"	" "
1	"	"	15	"	" "
2	"	"	16	"	" "

1	died	within	17	hours	of their admission.
1	"	"	18	"	" "
3	"	"	20	"	" "
3	"	"	22	"	" "
1	"	"	24	"	" "
7	"	"	26	"	" "
1	"	"	30	"	" "
1	"	"	36	"	" "

From ninety-six patients the hour of attack was, it is believed correctly ascertained:

3	were attacked at	1	A. M.	5	were attacked at	12	M.
9	" "	2	"	1	" "	1	P. M.
11	" "	3	"	3	" "	2	"
2	" "	4	"	6	" "	3	"
3	" "	5	"	1	" "	4	"
3	" "	6	"	3	" "	5	"
10	" "	7	"	1	" "	9	"
5	" "	8	"	10	" "	10	"
2	" "	9	"	6	" "	11	"
6	" "	10	"	2	" "	12	"
4	" "	11	"				

Of the two hundred and thirty-eight cases admitted, there were,

MALES,.....123 FEMALES,.....115

NATIVITY.

United States,.....	59
Ireland,.....	101
Other countries,.....	78
	238

In continuation of the report, allusion will only be made to such facts as were observed during the progress of the disease, and believed worthy of record, without attempting a general description of cholera and its treatment.

It would appear superfluous to allude to the symptoms of cholera at this time, so well are they known to every medical man at all familiar with the disease. Yet as the discharges are generally understood to be white or very light colored, it may be proper to observe, that in very many cases they were of dark amber or mahogany color. Such cases we thought more likely to terminate fatally than those having the true rice-water discharges. Another peculiarity of the dejections was observed in some cases admitted during the first stage; the consistence was like yeast, and the color resembling ashes and water. Cramps in the extremities were of very general occurrence.

In the treatment from the commencement of diarrhœa to the stage of collapse, our remedies were selected with the object of arresting the discharges as speedily as possible. With this view, opium, with some aromatic and stimulant, was first given, and mustard applied to the epigastrium, with dry heat to the extremities; after which, if unsuccessful, calomel, combined with opium or acetas plumb, was given, and frequently repeated if necessary. Injections of acetas plumb, ½ dr. in water 8 oz., were also used with some success.

Cold to the epigastrium we often found relieved vomiting; internal remedies appeared ineffectual in allaying this symptom. Stimulants were not generally found admissable during the stage of collapse, except in very small quantities.

In the consecutive fever, stimulants were not borne well, and the only reliance were counter irritants to the neck and extremities, with cold to head, and supporting the patient with arrow root, beef-tea, &c.; yet, in some cases, diuretics were used with apparent advantage. Here the mur. tinct. ferri was alone found advantageous, and not in the stage of collapse, as has been recently recommended.

The ol. terebinthinæ and chloroform, with brandy, highly recommended in the Philadelphia Medical Examiner, as being very effectual in the stage of collapse, was tried without any benefit. The cramps in the stomach we found frequently relieved by chloroform, internally, and very often by applying it externally to the part effected. Bandages to the extremities relieved the cramps in some cases.

Bleeding from the arm was resorted to in two cases, with marked and decided benefit.

Post mortem appearances were very similar in all the cases examined—lungs congested, bronchial tubes containing mucus; heart healthy, both ventricles filled with dark blood; liver engorged with dark blood; gall bladder full, and emptied with difficulty; stomach and small intestines highly injected, and thickened from effusion between the coats, presenting, when pressed between the fingers, a doughy feel; large intestines healthy; peritoneal coat of the small intestines red, dry, and adhesive, when taken hold of; kidneys congested; bladder very much contracted and empty.

Dr. Bezoin R. Masters, late Assistant Physician of Bellevue Hospital, whose aid I was fortunate in securing,

is entitled to full commendation ; unassisted by any person but myself, his duties, although constant and arduous, were at all times promptly and cheerfully performed, with decided skill and ability.

To the Sanatory Committee I desire especially to acknowledge my great obligations for the promptness and liberality with which all my requisitions were supplied.

With many thanks for the kindness and consideration which I have, during the brief period of my recent engagement, received from your Board,

I am yours, most respectfully,

ISAAC GREENE, M. D.

The Editor of the British & Foreign Medico-Chirurgical Review from The Author

ON THE

MODE OF ORIGIN AND PROPAGATION

OF THE

EPIDEMIC CHOLERA,

IN HUDDERSFIELD,

AND THE NEIGHBOURHOOD,

IN THE AUTUMN OF 1849.

BY JOHN TAYLOR, M.D., LOND.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS IN LONDON, AND PHYSICIAN TO THE HUDDERSFIELD INFIRMARY; FORMERLY PROFESSOR OF CLINICAL MEDICINE IN UNIVERSITY COLLEGE, LONDON, AND PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

[*Extracted from the* "MEDICAL TIMES" *of March 8th, and 29th, and April 12th, 1851.*]

LONDON:
PRINTED BY WILLIAM TYLER, BOLT-COURT.

1851.

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Fellow of the Royal College of Physicians in London, and Physician to the Huddersfield Infirmary. Formerly Professor of Clinical Medicine in University College, London, and Physician to University College Hospital.

Case 1.(a)—The first case of cholera in this neighbourhood occurred at the vagrant office, in the town of Huddersfield, on the 18th January, 1849. Francis Donoghue, aged 50, an Irishman, left Hull on Monday, 15th January, and walked in two days to Selby, where he arrived on Tuesday evening. He slept at Selby, and walked next day to Leeds. He had nothing to eat by the way but four raw turnips, which he got from a field by the way-side. On Thursday he walked from Leeds to Huddersfield, a distance of sixteen miles, and had nothing but bread to eat. He arrived at Huddersfield at 5 p.m., and was then well. His clothes were wet with rain, which had fallen during the day. At 10 o'clock the same evening he began to be purged, and had purging, vomiting, and severe cramps all the night. He had medical aid at half-past eight o'clock on Friday morning. I saw him at half-past three p.m., with Mr. Duke, assistant to Mr. Clarke, surgeon to the Union. Between this time and his death, which took place in fifty-seven hours from the time of attack, he was seen by several other medical gentlemen in the town. The symptoms were unequivocally those of Asiatic cholera. Copious vomiting; numerous abundant and liquid alvine

(a) The numbers prefixed to the cases correspond with the general table which will be found in a subsequent page, and indicate the order in which the cases occurred.

evacuations, not however free from bile; severe and very general cramps; suppression of urine; the peculiar collapsed expression of cholera; husky voice; surface cold, livid, and covered with abundant perspiration; pulse barely perceptible at best, and during a great part of the illness not at all. No *post-mortem* inspection was made.

After he was visited professionally, he was placed in a room by himself. During the first nine or ten hours of the attack, other persons slept in the same room. The room was afterwards well cleaned, and the bedding, I believe, destroyed. The disease did not spread.

The man stated that the keeper of the house in which he lodged at Selby died of cholera, which was then, I believe, prevailing in that town. The period of the death of the person referred to could not be clearly ascertained, but it was within a few days before Donoghue's visit. Two men had also died in the next house. I believe the cholera was not in Hull at that time; for only one death from cholera occurred in Hull in the quarter ending March 31st, 1849.

It seems not unlikely that this patient contracted the disease at the house he slept in at Selby, unless (which is not probable) he had previously been exposed to the cholera poison at Hull. The operation of the poison would be favoured by the man's long journeys on foot, want of proper food, and exposure to cold and wet.

Case 2.—The next case, as far as I have been able to learn, occurred July 9th, 1849, in a female at Colne-bridge, a few houses with some mills, about three miles to the north-east of Huddersfield. The patient recovered. She was attended by Mr. Booth, surgeon, who considered the case to be one of cholera. She had been eating cucumbers and fat meat a little before the attack. There was no reason to suspect that the patient had been exposed to any source of contagion.

Case 4 (b) occurred in Huddersfield.—William Leonard, aged 38, a gardener, was attacked, August 22nd, at five a.m., with violent purging, vomiting, and cramps; collapse soon followed. This passed away, and was succeeded by consecutive fever, with delirium. He died comatose, August 28th. He was a very intemperate man, and had lived on

beer alone for fourteen days before the attack, with the exception of a dinner of cold mutton on one day. For a month preceding the fourteen days he had practised total abstinence. He was attacked when in a beer-house, and, not being fit to be removed, he remained and died there. The patient's residence was in a back yard, or large court, in High-street. There was a privy in the yard in an offensive condition. He lived in a good house, and the locality altogether was far from being one of the worst. The water is good, being that used in the town generally. The drains were not bad. A fortnight before his attack, he was at Leeds on business. It is not known whether he was near any cholera patient when there. He was attended by Mr. Rhodes, surgeon, Huddersfield, to whom I am indebted for most of the facts I have stated.

This patient was waited upon by an old man—a drinker, and in bad health—but the disease was not communicated to him. The mistress of the house (who never saw the patient) and another female had some diarrhœa afterwards. The surgeon in attendance likewise suffered from diarrhœa for a day. He had no other cholera patient at the time. No case of cholera occurred near this place until September 22nd (see Case 37), and there was no apparent connexion between the two cases.

Case 5.—Mrs. Hardy, aged 60, was attacked August 26th. Had vomiting, very copious rice-water stools and cramps. She was attended by Mr. Tatham, and recovered. She resides in Lower Head-row, Huddersfield. The situation is low—part of the yard is dirty. There is one privy for five houses, and it was in an offensive state. The house is an old one, the drains are good, the water is good. This patient is in comfortable circumstances, and is not aware that she had been exposed to any source of contagion.

Case 8 (b).—James Atkinson, aged 49, cordwainer, was attacked with diarrhœa August 29th. After seven days

(b) It is stated in the report of the Registrar-General for the quarter ending September 30th, 1849, on the authority of the Registrar of Huddersfield, that the first case of cholera was registered on the 2rd of August, and the second on the 9th. This is an error. I have ascertained from the Registrar of Huddersfield, that the two cases referred to in his report are those marked 4 and 8 in my report.

this passed into cholera, of which he died in four days more. He was a very intemperate man. He took beer chiefly. Did not do a week's work for three months before his death, in consequence of drinking. He took very little food. He lived in Fountain-street, in the lower part of Huddersfield. There are one or two open privies near the house, which were in a very offensive state. Other houses, however, were nearer to the privies than the one in which he lodged. The water is good. It is not known that he had been exposed to the contagion of cholera.

The 9th case was undoubtedly one of Asiatic cholera, and occurred at Cliff-end, a small village two miles *west* of Huddersfield. Grace Bake, aged 29, married, and has several children. In the night between the 3rd and 4th September 1849, was attacked with diarrhœa, having to get up several times. At ten o'clock on the morning of the 4th September she washed her children. At half-past eleven o'clock, the neighbours, hearing a knock against the partition-wall, entered the house, and found her upon the floor. She was very soon visited by Mr. Holroyd, assistant to Mr. Machill, who afterwards saw other cases of cholera, and is satisfied that this was one. This patient was livid, had severe cramps, in addition to vomiting and purging. She died at six p.m., probably not more than seven hours after the symptoms of cholera appeared. No one had any communication with her after the disease was known to be cholera, but her husband, her sister, and the clergyman of the place, the Rev. J. W. Gibson, to whom I am indebted for many of the facts of this and other cases subsequently occurring in the same locality. Part of the linen used by the patient was burnt; the rest was sprinkled with lime before it was washed; the house was fumigated; the husband and children left it, and it has not since been occupied. About thirty persons in the adjoining houses were afterwards affected with diarrhœa, which the medical men ascribed to fright.

The linen used by Grace Bake was washed by her two sisters and another female. Two of these continued well, but a third suffered from diarrhœa, which began four or five days after washing the linen, and continued for some time.

After the most careful inquiry, I have not been able to discover any ground for supposing that this patient had been exposed to contagion. She had visited Leeds, but this was in the month of July. There were very few deaths from cholera in Leeds in July; the patient's husband had not heard that the disease existed in Leeds at that time; and the time which elapsed between Grace Bake's visit to Leeds, and her attack, (five or six weeks,) is too long to permit the supposition that the disease had been contracted there.

This case was the first that occurred in the district in which the patient lived. The house is situated on the brow of a hill, at a considerable elevation, and on the north side of a rather narrow valley. It is in a "fold," consisting of two short rows of cottages about twelve feet apart. At one end of the fold, there is a cow-house with an open drain from it. This is generally full of dirty liquid, and smells offensively. This drain is about eight yards from the door of the house in which the patient lived, and is nearer to another house in which there was no cholera. On the other side of the fold, not quite opposite to the house, and about fourteen yards from it, is an old privy, at present used as a receptacle for ashes, dirt, and other refuse. There is a well of good clean water in the yard, and another in one of the rooms of the house. The family used both. The water in the yard appeared to me very good—that in the house was described to be so,—but I did not see it. There had been no change in the appearance of either, and no reason to suspect any contamination. The water has not been examined with the microscope.

Several other cases occurred in this fold, but not until *nine days* had elapsed from the death of Grace Bake. They will be noticed hereafter. (See Cases 20, 23, 76.)

Case 10.—On the same day on which Grace Bake was attacked, and only a few hours later, the next case occurred in the village of Lindley, situated about a mile to the north of the place last described, and above two miles to the north-west of Huddersfield,—Mrs. Hirst, aged 53, the wife of a carcase-butcher in easy circumstances. Sept. 4, had diarrhœa all day, but without pain. In the evening, cramps supervened, and she then sought medical aid. She was attended by Mr. Holroyd, assistant to Mr. Machill, the surgeon of the place.

She died at eight o'clock, a.m., on the 5th, in less, probably, than twelve hours after the diarrhœa had merged into cholera.

Mrs. Hirst was much afraid of cholera. She was greatly alarmed when she heard of the case which occurred only a few hours before she was taken ill herself. (No. 9.) It does not appear probable that there was any contagion in this case. I shall mention all the facts bearing on this point which, after careful inquiry, I have been able to elicit. Her husband had not been to buy cattle, or for any other purpose, to any place where cholera existed. She had paid a visit for a few days to Halifax, and returned about a week before her fatal illness. Her husband says she was not near to any cholera patient there. Halifax has been remarkably free from cholera during the epidemic. Whether *any* case existed there at the time of Mrs. Hirst's visit I am not able to say, but I believe there was no case. When at Halifax she had some looseness of the bowels to which she had been subject for several years.

A female relative residing at Hull paid Mrs. Hirst a visit in July. She came to Lindley about the middle of July, and staid a fortnight. The first case of cholera in Hull occurred on the 17th of July. Her husband died of cholera some time after her return to Hull, when the disease had become epidemic. More than a month elapsed between this person's departure and the illness of Mrs. Hirst,—a period probably too long to permit us to ascribe the illness of the latter to contagion derived from this source. No communication by box, parcel, or in any other way, afterwards took place between the parties.

Lindley stands upon elevated and gently-sloping ground, and has a population of perhaps from 1000 to 2000 persons. There are several circumstances about Mr. Hirst's house deserving of notice. Cattle are slaughtered in a building near to the house; the butcher's shop is within a few yards of the door, and the slaughter-house is under the same roof. There is an ash-place attached to a privy, also within a few yards of the door; into this are thrown blood, entrails, and other refuse. The smell was very offensive at the time of the cholera, and even at the time of my visit, six weeks afterwards. To the south of the house, and within twenty yards, was a dung-heap and a building for calves, with a good deal of liquid manure about it. This place was very offensive at the time of my visit, although it had been cleaned at the outbreak of the cholera. Two cottages are situated close to this filthy place, and their doors open upon it, yet no cholera occurred in them. Within forty or fifty yards of the house is a burying-ground attached to a chapel, but it is not at all crowded. At a distance of 200 yards there is also a place in which offal is boiled for pigs, and the smell from which is very offensive. The drains in the house are said to be good, and I did not perceive any smell from them. The water used for drinking and cooking is obtained from a spring at a little distance, which is also used by many other persons, and is said to be very good.

Only two other cases of cholera occurred at Lindley: the one on the 9th, the other on the 11th September. (See cases 16 and 19.) Both patients were in attendance upon Mrs. Hirst. About the same time, the cases of diarrhœa, many of them accompanied with rice-water evacuations, were very numerous.

Case 12.—William Rhodes, aged 44, a quarryman, was attacked Sept. 6th with cramps, purging, and vomiting. He never fell into a state of collapse, and he recovered. Mr. Allatt, who attended the man, considers the case to have been one of Asiatic cholera. The man resided at Clough, a little west of Paddock, and about half a mile east of the place where the first case in this district occurred (No. 9). He had worked at the railway near Huddersfield; he had had no known communication with any other cholera patient.

Other cases afterwards occurred at Clough, (cases 25, 33, 41, 45.) The place consists of a few small cottages, most of them only one storey high, without any thorough ventilation. The ground is elevated. There are no drains; the refuse is thrown on the surface. The water is procured from a neighbouring well, which is supplied by a spring at a distance; it appears to be very pure.

A nephew of the last patient, a blind boy, named Haigh, hearing that his uncle was ill, went on the first night of his

illness to visit him, and, whilst in the house, was attacked with vomiting and purging. He returned to his home, about a quarter of a mile distant, at a place called "Johnny Moore-hill," where the epidemic afterwards prevailed with the greatest virulence. He procured some medicine from a druggist, and recovered. This case could not be said to amount to cholera, although the attack may have been due to the choleraic poison. If so, it is not likely that the disease was taken from the uncle, the period of incubation being too short; and no other cause can be assigned than the local or general epidemic influences beginning to operate about that time.

About September 22nd, when cholera prevailed in the adjoining houses, this boy, his mother, and several other children, left their home in alarm, and went to reside in the middle of the village of Paddock. They went to a daughter, who was married and had three or four children. They had only one room, and both families now lived in it, yet none of the daughter's family were attacked with cholera. The blind boy's mother had diarrhœa, which began before she left her own home, and continued after she joined her daughter.

Case 14.—Mrs. P. Ganter, aged about 35, was attacked September 8th, at 5½ a.m., with vomiting, purging, and severe cramps. The evacuations were very copious and like rice-water. Mr. Tatham, who attended her, has no doubt the case was one of cholera. He visited her eight times the first day. She recovered. This patient is not aware that she had been exposed to the contagion of cholera. She lives in Church-street, Huddersfield. The house is rather confined. Close to the back of the house is a mews, into which a window of the house opens. Near to this window was a manure heap, on which was thrown offensive refuse. There are also privies with open cesspools. The smell from these places was so offensive, that they were obliged to keep the back window always closed. The drains are not complained of. The water is that used generally in the town. It is brought from a distance of two or three miles, and is very good. There was no known contamination of it about the time of the patient's illness.

Case 16.—Mrs. George Crossland, aged about 30. On Sunday, September 9th, cooked the dinner, and partook of it, but before the afternoon service was so unwell as to be obliged to remain at home. She died about 9 p.m., after not more than about eight hours' illness.

She was the daughter of Mrs. Hirst, of Lindley, (Case 10;) lived in the same house with her mother, and nursed her. She was very much afraid of cholera during her mother's illness, and took brandy freely to keep it off. The day before her attack she took, of her own accord, some salts and senna as an aperient. This acted freely, and Mr. Holroyd, in consequence, sent her some laudanum. In the evening of the same day, however, she again took medicine containing Epsom salts. This patient may have taken the disease from the same causes which gave it to her mother, or she may have received it by contagion. In the latter case, the period of incubation could not have exceeded five days.

Case 17 occurred on September 9th at Lane, about half a mile from Huddersfield. The patient, Joseph Thornton, was attended by Mr. Booth and recovered. There was no known exposure to contagion. The situation is low and unhealthy.

Case 18.—Mrs. Walker, aged 60, resides at Paddock-brow, about half a mile from the nearest patient previously affected with cholera, was attacked September 11th, and was attended by Mr. Allatt. She was pulseless. I saw her September 23rd. She was still in bed and had diarrhœa. She recovered. This woman keeps a mangle, and rarely leaves her house. Had not been out of the row in which she lives for five weeks before her illness, nor more than two or three doors from her house for several days. Her house is situated from a quarter to half a mile from the place where cholera was afterwards most prevalent.

Case 19.—Mary Craven, aged about 35, lived at Lindley. A married woman, pregnant, and within a few days of her expected delivery. She nursed the only two additional patients who suffered in the same village, (Cases 10 and 16.) She also washed their bed and body linen. Was attacked with diarrhœa Sept. 11, at 11 or 12 o'clock a.m. Was seen

professionally at 6 p.m.; at 7 p.m. had cramps, and died on the 12th, at 2 a.m. Her own residence was in one of a fold of cottages, and about twenty yards from the house of the patients she nursed. There was a heap of ashes and other refuse within a few yards of the door of her house. This patient was exposed to the same local influences as the other two, as well as to contagion. She was attacked seven days after the one, and two days after the other, of the patients to whom she attended.

Case 20.—A female, aged 72, living at Cliff-end, and in a house exactly opposite to Bake's (Case 9), and close to the bad-smelling drain. She was attacked September 13th, had the usual symptoms, and died on the 15th. If this patient caught the disease by contagion, the period of incubation would be nine days. She had no communication, however, with the only patient who was attacked before her.

Case 22.—A patient of Mr. Booth's; had the disease in Union-street, Huddersfield. Was attacked September 16th, and recovered. No other case occurred within about half a mile of this. No source of contagion was known.

Case 23.—The husband of the last patient but one (Case 20) was attacked next. On Sunday, September 16th, the day after his wife's death, was not very well. He worked on the Monday, but looked pale and ill. On Tuesday night he began to be purged, and died on the Thursday, at three or four o'clock p.m., after about forty hours' illness. It is stated, that not long after his wife's death there arose a suspicion that she was not dead. He then got upon the bed, leaned over the body, and put his face to the mouth of the corpse, to feel whether the breathing had ceased. The interval between the commencement of the illness of the wife and that of the husband was three days.

Case 76.—The next case at Cliff-end occurred in the person of the son of the last patient. Their houses are two doors only from each other. He had diarrhœa for about a week; became worse September 29th. Went to Huddersfield Oct. 2nd. He got some porter there; was much worse soon after his return home in the evening, and died on the 3rd, after an illness of about eleven hours.

The son of the last patient was attacked October 1st, (the day before his father's diarrhœa merged into cholera.) The case was not severe, and he recovered.

The sister and also the mother of the last patient had diarrhœa. The former began October 5th. Both recovered.

Two daughters of the old woman and man (Cases 20 and 23) washed the linen after their deaths. One of them has remained well. The other has long had an habitual looseness, and it did not become worse.

Case 25.—This was the second case, (the first was No. 12,) and the first fatal case, which occurred at "Clough," near Paddock. The patient was a female, aged 78. She died Sept. 17th, after 21 hours' illness. This patient was attacked ten days after the first case occurred near to her. Her son had come from Manchester a short time before. He was not ill. It is not known whether he had been near to any person affected with cholera.

Case 26.—Joseph Hinchliffe, aged 61, residing in the village of Paddock, was attacked Sept 17th. He had the cholera in a mild form, and recovered.

Case 27.—Mrs. Hirst, aged about 60. Was not well Sept. 16th. Was better on the 17th, and out of doors. On the 18th, at two o'clock a.m., began to suffer from the usual symptoms of cholera, and died about 4 p.m. the same day. She was attended by Mr. Allatt. I was sent for, but arrived only just after the death. This patient lived at Brierley Mill, near Paddock, about half a mile from any patient who had as yet suffered. The house stands in the bottom of the valley, close to both the river and canal, and not far from an old fish-pond, to be noticed hereafter. There was an offensive privy near to the house. The patient was in comfortable circumstances, and there is reason to believe that there had been no communication with any cholera patient.

Case 29.—Hannah Taylor, aged 55, was attacked Sept. 19th, at 10 or 11 o'clock, p.m., and died after 12 hours' illness. This patient lived at Paddock, in a part known by the name of "Johnny Moore-hill." This was the place where the disease raged with far greater severity than anywhere else in this district. The present may be regarded as the first case of the epidemic in this spot, although a pre-

vious case (probably one of diarrhœa only) has been already given.

Case 30.—Wm. Moss, aged 23, a railway labourer. Lodged in the house of the last patient, and they began to be ill within an hour of each other. He went to Huddersfield (about a mile distant) on the evening of the 19th; returned home about 11 o'clock; complained of cramps and diarrhœa. He died in 10 hours. This man left Paddock Sept 15th, and went to Eccles, near Manchester. He returned on the 17th. He said his mother, who lived in Lancashire, had had the cholera, but recovered. He did not visit her, and was not within two miles of her.

Case 31.—John Alpine, aged 34, a railway labourer. Resides at Johnny Moore-hill. Was attacked Sept. 19th. He passed through the stage of collapse, and died in the consecutive fever, after more than a week's illness.

Case 32.—Sarah Taylor, aged 23; married. Began at 4 a.m., Sept. 21st. Died after 19 hours' illness. She was the daughter-in-law of Hannah Taylor, (Case 29.) She lived near to Hannah Taylor, and nursed her. This patient was attacked in about 30 hours after the commencement of the illness of her mother-in-law.

Case 33.—Mary Tomlinson, aged 20, single. Resides at "Clough," near Paddock, about a quarter of a mile from the last patient. Was attacked at 6 a.m., Sept. 21. The disease was well marked. The patient recovered without consecutive fever. This patient lived in the same house with her grandmother, who died of cholera, Sept. 17th, (Case 25.) If she caught the disease by contagion, the period of incubation was within four or five days.

Case 34.—Allen Micklethwaite, aged 17, strong and healthy. Resides at Johnny Moore-hill. Came home from his work (about a mile distant) at 6 p.m., Sept. 21st, having had several loose stools during the day. At 8 o'clock I saw him in a state of collapse. He died in seven hours after his return home. Micklethwaites ascribe the introduction of cholera into their house to the blind boy (mentioned Case 9) who lived next door, and often visited them. If this were correct, the period of incubation would be not less than 14 days. It has been ascertained that this patient, although

visited by us, was treated exclusively by a homœopathic practitioner.

Case 38.—Aaron Taylor, aged 55, was attacked Sept. 22nd, early in the morning. He recovered, after a protracted illness. He was the husband of Hannah Taylor (Case 29,) and the father-in-law of Sarah Taylor (Case 32.) There was an interval of about 58 hours between the attack of the wife and the husband.

Case 39.—Ezra Whitely, aged 2½ years, residing at Clough, near Paddock. Began Sept. 22nd, and died in four hours. This patient was dying when first visited.

Case 41.—Thomas Dyson, aged 16 months, residing at Clough. Had had diarrhœa for a long time. Became worse Sept. 22nd. Collapse and death, 23rd. This child lived next door to Tomlinson (Case 33.) There was an interval of somewhat more than 24 hours between the beginning of the two cases.

Case 40.—Charles Potter, aged 23, railway labourer, residing at Johnny Moore-hill. Was attacked Sept. 22nd, at two p.m. Died in the consecutive fever after some days.

Case 37.—Thomas Wood, an engineer, was attacked in the morning, Sept. 22nd. Lives in the Dock-yard, at the bottom of Dock-street, Huddersfield—a low situation, and near the canal. Had been up at work all the night before the attack, and had taken some beer, but no food. He recovered. This man lived in the neighbourhood of, but was never in, the beer-shop in which Leonard (Case 4) died three weeks before he was attacked.

Case 35.—Mrs. Micklethwaite, aged 46. Began with diarrhœa on the morning of Sept. 22nd. Passed into cholera in the succeeding night. She recovered, after some weeks' illness.

Case 36.—John Micklethwaite, aged 44, husband of the last patient. Began with diarrhœa, at ten p.m. Sept. 22nd. Collapse succeeded in the night, and death after 17 hours' illness.

These two patients were the parents of Allen Micklethwaite (Case 34.) They both attended upon their son, and rubbed his limbs assiduously for the cramps. If the disease were caught from the son, the period of incubation in the

wife did not, probably, exceed 12 hours, nor in the husband 28 hours.

Case 45.—Jane Dyson, aged 4½ years, residing at Clough, sister to Thomas Dyson (Case 41.) Began Sept. 23rd, at twelve o'clock at noon. Died in 18 hours. There was an interval of about 24 hours between the beginning of the illnesses of the two children.

Others were attacked at Johnny Moore-hill, but I cannot give the names and dates of attack of each. (See general table of cases, where the dates are now given.)

Case 54.—Sept. 25th. I began to suffer from the cholera poison myself. I first visited a cholera patient Sept. 18th, but only saw her after death (Case 27.) The next one that I saw was Sept. 21st, at eight p.m. I visited, on this occasion, a considerable number in succession, at Johnny Moore-hill, the place where the disease exhibited the greatest malignancy. Sept. 22nd.—I did not see them, in consequence of a sick headache, of which I am frequently the subject. On the 23rd, I was called up at six a.m., to visit the same series of patients. I took the precaution of getting my breakfast before going, especially as I had taken scarcely any food the day before. In the afternoon I saw them again. On the 24th, at five p.m., I accompanied some gentlemen into the fields near Paddock, to select a site for a Cholera Hospital. The grass was damp. I went next to inspect an old fish-pond, which will be described hereafter. I next visited all the cholera patients, and got home late in the evening, after a hurried day's work. Sept. 25th.—My habitual morning evacuation was rather liquid. About nine o'clock I felt rather chilly. Soon after, I visited the cholera patients again. The chilliness and feeling of general indisposition increased in the afternoon, but I did my usual work until five p.m. I then took some warm tea, and lay down upon a sofa near the fire. During the evening I felt hot and chilly at the same time. My head was heavy, and I had a good deal of vertigo and singing in the ears. Pulse frequent, about 120. About nine p.m. had a second evacuation, scanty, but liquid. I was now visited by three of my medical friends (Mr. Greenwood, Mr. Clarke, and Mr. Booth.) By their advice I took a dose of an astringent

mixture, with ℥xx. tinct. opii. I went to bed, and slept until twelve o'clock. On awaking, I had two, or perhaps three motions, within a short time, all scanty, and moderately liquid. The most striking symptom, however, at this time was the prostration of strength. I have suffered many illnesses, attended with much debility; but the sense of sinking which I now felt exceeded anything that I ever experienced in my life. When placed upon a night-chair, I could not maintain the erect position without being supported. All this had supervened within a few hours, and after only a very small amount of liquid had been discharged by the bowels. My medical friends were again sent for, and thought the prostration so serious, that they did not consider it proper to leave me, and very kindly remained all night. I repeated the astringent (mist. cretæ, conf. aromat., tinct. catechu, with tinct. opii.) The bowels were very little moved afterwards. At three a.m. I had a starch enema, with 40 minims of tinct. opii. This quieted the bowels more than anything. They were not again moved for some hours. Soon after three o'clock I vomited. This was the only time I was sick. The matter ejected seemed to consist almost exclusively of the medicine which had been taken. I had after this a large sinapism on the abdomen.

Sept. 26.—Prostration considerable; tongue thickly furred; a loathing of all food; skin hot; pulse 100 or more; several scanty motions, accompanied with tenesmus. From this time my improvement was progressive, and I considered myself convalescent by the 30th September.

The symptoms I have detailed were rather those of dysentery than of the severer forms of cholera. Yet I feel persuaded they were the effects of the choleraic poison. My medical attendants thought the same, both on account of the extreme prostration of strength, and the characteristic expression of countenance. I am satisfied, likewise, that the poison was imbibed at Paddock, either from the patients or the place. I was quite well before visiting the patients, and was not elsewhere exposed to any known causes of the disease. The period of incubation would probably be between 84 and 50 hours.

Case 63.—Richard Berry, aged 64. Resides at Folly Hall, half a mile from Huddersfield, and about a mile from Paddock. He was engaged as a nurse, and went to Johnny Moore-hill, to wait upon Mrs. Micklethwaite and her two sons, all ill of cholera in one house. He began his attendance on the evening of Sept. 25, or the morning of Sept. 26, and was then quite well. On the morning of Sept. 29, he was attacked with cholera, was removed to his own residence the same day, and died on the 30th, within 24 hours from the beginning of the illness. There can be very little doubt that this man took the disease at Johnny Moore-hill. The period of incubation was within three days.

This patient lived in a cellar alone with his wife, who waited upon him. On the evening of the day following his death, she had cramps in the legs but no diarrhœa. His son, also, who waited upon him, but did not live in the same house, soon after his father's death, had pains in the bowels, but no diarrhœa. The disease did not spread in the place into which it was thus introduced.

A few other facts may be briefly mentioned in connexion with the cholera at Johnny Moore-hill.

Edward Brook and his wife, a young couple, left their residence at this place on the first outbreak of the cholera, retiring to a village about a mile distant. They returned home on Friday, Oct. 5th, at ten o'clock, a.m. On Sunday morning, at one o'clock, the wife was seized with cramps in the feet and purging, but no vomiting. Medical aid was obtained, and on the 8th, when the facts were communicated to me, she was better. The husband at the same time had pain in the bowels, but no diarrhœa. A brother of the husband, who lived with them, and who went away and returned with them, continued well when I last heard, a fortnight afterwards. The house was shut up in their absence, and after their return they had no communication with their neighbours before they were taken ill.(a)

Case 67.—John Wm. Taylor, aged 3, left Johnny Moore-hill Sept. 28th, and went with his father and another child, to Meltham, a village five miles distant. Sept. 30th, he was attacked with vomiting and purging, and died within 24 hours. The disease was considered to be cholera. The father and several other persons in his house had diarrhœa before he left home. Here, no doubt, the poison was imbibed at home, and produced its effects about 48 hours after leaving the place. The mother, grandmother, and several other children, went to Liverpool at the same time that the father went to Meltham. No communication passed between the divided parties before the child's death.

(a) Dec. 21st, 1849.—I have learned to-day that Mrs. Brooke is subject to cramps. She has had them before and also since the attack described above. It is possible, therefore, that she may not have suffered from the cholerale poison.

There was no cholera in Meltham at the time the child went there, and the disease did not afterwards spread.

A family, living at Johnny Moore-hill, next door to Micklethwaites', immediately after the first death in their neighbour's house, (*i. e.*, on September 22,) left their home in terror, and moved to the house of a relative in the centre of the village. The two families, consisting of a considerable number of persons, now lived together in a small house, and occupied, in some instances, the same beds. Diarrhœa had appeared among those migrating before they left home. The disease did not further appear in them, nor were their relatives or new neighbours affected.

DESCRIPTION OF "JOHNNY MOORE-HILL," AT PADDOCK.

The village of Paddock is situated about a mile to the west of Huddersfield. It stands upon the side of a hill, and at the opening of a series of valleys extending for about seven miles to the Standedge hills. The population amounts to several thousands, and consists chiefly of operatives. The drainage is bad. The place called Johnny Moore-hill consists of two rows of houses at the western extremity of the village. They stand upon very steep ground, which is excavated to receive them, and at an elevation of 150 or 200 feet from the bottom of the valley. The houses are two storeys high, the back part of the lower storey being buried in the ground. Each house consists, for the most part, of one lower room, and a chamber over it. The windows and

doors are all in the front, so that there is no thorough ventilation. The drains are very imperfect, the lower rooms damp in various degrees, and the privies badly constructed. The cesspools are open to the air, and very offensive. It is the poorest and most filthy part of the village, and Mr. Allatt, the surgeon, states that it is a place notorious for the prevalence of fever and other forms of disease. About 150 yards west of Johnny Moore-hill, and in the bottom of the valley, there is an old fish-pond. The water in this had been nearly all drawn off a few months before the cholera broke out. The pond has not been cleared out for thirty years, and contains a large accumulation of moist vegetable matter uncovered by water. The surface of this pond measures 1400 square yards.

It has been stated (I cannot say how accurately) that for two days before cholera appeared at Johnny Moore-hill the wind blew from the west, and, consequently, from the pond, towards the dwellings. Mr. Allatt informs me that during nearly the whole of the time in which the cholera was prevailing, the wind was easterly. Had this direction of the wind anything to do with the immunity from the disease experienced by the village at large? and had it any connexion with the appearance of the disease in three places situated respectively at the distance of one, one, and four miles to the west of Johnny Moore-hill during the prevalence of the epidemic at the latter place?

Water.—Nearly all the people at Johnny Moore-hill use the water of a spring which is near to them and partly under the embankment of the Huddersfield and Manchester Railway. They have used the same water for years, but they complain that it has been much harder and less pleasant to the taste; and that after rain it is often muddy, since the construction of the railway embankment a year or two ago. For two months preceding last July or August, two of the families had their drinking water from the portion left at one extremity of the fish-pond. This water is said to be not unpalateable.

Population.—Most of the houses were overcrowded. Many of the occupants were intemperate persons, a number of the lodgers being navigators.

The following table(a) will show the number of houses, the number of inhabitants in each house (distinguishing adults and children), the numbers attacked with cholera and diarrhœa, the number of those dying, and the number of those who fled at the outbreak of the disease.

Lower Row.

Number of House.	Number of Inmates. Adults.	Children.	Total.	Had Cholera.	Diarrhœa.	Died.	Fled.
1	6	0	6	1	2	1	3
2	4	0	4	0	3	0	0
(a) 3	2	4	6	4	0	1	0
4	2	4	6	0	3	0	0
5	2	5	7	0	4	0	0
6	5	4	9	1	0	1	8
7	2	3	5	1	0	1	4
8	3	5	8	1	7	1	0
9	5	4	9	5	1	2	1
10	2	3	5	0	0	0	5
11	3	0	3	0	(b) 1	0	3
	36	32	68	13	21	7	24

Upper Row.

(c) 1	6	0	6	4	0	2	0
2	1	5	6	0	4	0	0
3	1	2	3	0	1	0	3
4	2	3	5	2	0	1	0
5	4	2	6	0	0	0	0
6	2	4	6	1	2	0	0
7	2	2	4	0	2	0	0
8	2	4	6	0	2	0	0
	20	22	42	7	11	3	3
Total...	56	54	110	20	32	10	27

(a) There was only one room in this house.
(b) This patient was attacked after her return. (Mrs. Brook.)
(c) There was only one sleeping room in this house for six adults.

It appears that out of nineteen houses there were only two in which neither cholera nor diarrhœa occurred. The occupants of one of these two all fled, and so escaped. The

(a) For many of the facts contained in this table, as well as in the whole account of the disease at Paddock, I am indebted to the kindness of Mr. Allatt. He has taken great pains to obtain correct information, and has most freely permitted me to make use of it.

occupants of the other were distinguished from their neighbours by their superior cleanliness. No other reason appears to explain their immunity. The house was rather crowded, containing six persons, of whom four were adults, and the cholera occupied the adjoining house on each side.

Of those who fled, all escaped the cholera except one child and a man.

The disease prevailed most in the most crowded houses, except in the house just referred to as distinguished for cleanliness.

There are two cottages placed across the western extremities of the two rows of houses, but situated fifteen or twenty yards from them. In one of these live three persons, adults; in the other eight persons, six being children. None of these eleven persons suffered except one in the second house, who had diarrhœa severely. All these people are members of one family. They are superior to their neighbours in Johnny Moore-hill in station, in cleanliness, and in their habits generally, and do not associate with them.

The inhabitants of the two rows of houses, at the outbreak of the cholera, amounted to 110, including both sexes and all ages. Of these, 52 were attacked with either cholera or diarrhœa, being nearly one-half. A further analysis of the cases is exhibited in the following table :—

Adults Exposed.	Attacked with Cholera.	Proportion.	Attacked with Diarrhœa	Proportion.
56	13	1 in 4½	19	1 in 3 about
Or Excluding those who Fled.				
40	13	1 in 3	17	1 in 2½ ,,
Children Exposed.				
54	7	1 in 8	13	1 in 4 ,,
Or Excluding those who Fled.				
43	6	1 in 7	13	1 in 3½ ,,

Of those exposed to the disease, therefore, the proportion of attacks of cholera was twice as great in adults as in children. The attacks of diarrhœa occurred in nearly the same proportions in the two classes of subjects.

Of the thirteen adults attacked with cholera, seven, or fully one half, died; and of the seven children attacked with cholera, three died, or rather less than one half.

Case 64.—Mary Goodyear, aged 39, residing at Cropper's-row, Huddersfield, had diarrhœa for some days. Was attacked with cholera Saturday, Sept. 29, and died in eight or nine hours. She was attended by Mr. Clough. This was the first case that occurred in this part of Huddersfield. The place in which she lived is unhealthy, and the drains are bad. There was a privy close to the house, which had not been cleaned out for twenty years, and the contents of which had soaked into the ashpit, made under the hearth within the house. It has been stated that this patient washed the clothes of cholera patients from Paddock, but the statement is not correct. She had walked one day through Paddock, and near to Johnny Moore-hill, but without stopping there, and had not in any other known way been exposed to contagion.

Case 65.—John Goodyear, aged 45, a cloth finisher, the husband of the last patient, was attacked with cholera Sept. 30th. He recovered.

Case 68.—John Goodyear, the son of the two last patients, aged five years, was attacked with cholera October 1st. He recovered.

Case 71.—Abraham Smith, aged 39. Attacked with cholera Sept. 30th, died Oct. 3rd. Resided at Cropper's-row, fifteen or twenty yards from Mary Goodyear. This man stood on one occasion within the door of Goodyear's house during her illness for about ten minutes, but did not enter further. Was treated by Mr. Booth.

In the beginning of October the cholera broke out at Slaithwaite, a village about 4½ miles west of Huddersfield. For the particulars of the cases which occurred there I am indebted to Mr. Roberts, surgeon.

Case 72.—Mary Quarmby, aged 74. Performs the duties of a barber. Lives in the centre of the village. She was attacked Oct. 1st, and died in twelve hours. She was attended by Mr. Dean, but was not seen until two or three hours before she died. No communication with any cholera patient could be traced.

The house in which she lived consists of two rooms; it is in the middle of a row of houses, and in a narrow street. She and her son (the next patient) alone lived in the house. They were dirty people. There were two heaps of ashes and refuse near the house but no bad drains. The water they used was the same as that used by their neighbours. It was supplied by a spring not far distant, and is considered good.

Case 81.—Josh. Gledhill, aged 50, son of the last patient, and lived in the same house; was attacked October 4th, and died after fourteen hours' illness. The case was well marked and was attended to by Mr. Roberts.

No other cases occurred in this part of the village.

Case 82.—A child, aged 18 months, lived near the river side in Slaithwaite, (at Dry-Mill,) was attacked, October 4th, with well-marked cholera. It rallied after twelve hours, but died a day or two after in convulsions. Mr. Roberts believes it is not possible that this child could have had any communication with the preceding patients. The locality is low, as well as near the water.

The next cases occurred at "Water-Side," in Slaithwaite, about 200 yards to the east of the village.

Case 83.—Charles Varley, aged 36, a small clothier, was attacked October 7th, and died in a few hours, with all the ordinary symptoms of cholera. He was attended by Mr. Dean.

Case 86.—Mrs. Varley, the mother of the last patient, aged 57, was attacked October 10th, and died after fifteen hours' illness.

Cases 87 *and* 88.—Mr. Varley, the husband of the last patient, and also Mrs. Charles Varley, his daughter-in-law, were both attacked at the same time as Mrs. Varley, or within two or three hours of each other. These two recovered. The last three patients were treated by Mr. Roberts. All the four patients lived in the same house. The house is situated in the bottom of the valley, having a canal immediately behind and the river immediately in front of it. The privies are not very near. The drains are bad. The water is of good quality. Charles Varley was an Odd Fellow, and had been at his lodge and got too much to drink the night

before his attack. No communication with any other cholera patient could be traced.

No other cases of cholera occurred in this village, but diarrhœa prevailed extensively.

A solitary case of cholera occurred at Miln's Bridge, a small village about two miles west of Huddersfield, and in the valley leading to Slaithwaite.

Case 80.—John Saville, aged 57, a small farmer; on the morning of October 4th began to suffer from diarrhœa; in the following night the diarrhœa passed into cholera, and he died at 10 p.m. on the 5th of October. There were three persons living in the house; they were cleanly people, and their circumstances easy. The house is small, and stands close to the river side. The cow-house is near to it but is kept clean. The drains are not offensive. No communication with any other cholera patient could be traced.

During the time that I have been occupied in preparing the preceding account, and since it was written, I have become acquainted with the existence of a number of additional cases of cholera in Huddersfield and the immediate neighbourhood. This additional information does not invalidate the correctness of the particulars already given. Instead of adding a detailed account of the fresh cases, I propose to record, in a tabular form, a brief account of all the cases in the order in which they occurred, adding to the Table a few memoranda respecting such cases as call for further notice. I believe the Table will contain a complete account of all the cases which occurred in the practice of the surgeons resident in Huddersfield. It may not, perhaps, include all the cases which occurred in the adjacent villages, but I shall insert all with which my inquiries have made me acquainted.

The diagnosis in these additional cases is that of the surgeons whose names are attached to them. [*See Table.*]

Notes on some of the cases in the preceding Table:—

No. 3.—This patient was at Liverpool, in a district infected with cholera. He was taken ill very soon after getting home. The disease did not spread in his own neighbourhood.

No. 13.—This man had been at a crowded public meeting

in the Philosophical Hall the night before he was attacked. There was no known exposure to contagion. There was suppression of urine in the attack during a period of eighteen hours.

No. 21.—This was the child of No. 9. After the mother's death, the father went to live at Mold-green, near Huddersfield. The child probably carried the seeds of the disease with it, and was attacked ten or eleven days after. This may therefore be regarded as the period of incubation. The disease did not spread at Mold-green.

No. 48.—This man came from Bradford, where cholera was very rife at the time. He was attacked very soon after reaching Huddersfield. The disease did not spread from the house in which he was ill, although it is in one of the worst quarters of the town.

No. 58.—This man was living at Johnny Moore-hill. He left that place to avoid the disease, and was attacked soon after at his new residence.

Nos. 89 and 91.—These patients both lived in the same house. There was no discoverable source of contagion in the first case. The locality, as the name indicates, is swampy.

No. 92.—This man lived at Slaithwaite. There had been no cholera there for three weeks before he left the place. He was attacked in the first night of his imprisonment.

No. 93.—No contagion could be discovered in this case, but there is a great quantity of filthy refuse near the house.

Having given a complete list of all the cases with which, after much inquiry, I have become acquainted, I am now prepared to examine them, in order to see whether they contain matter calculated to contribute anything towards the elucidation of the very important questions respecting the mode of origin and propagation of the cholera.

I.—CONTAGION.

I. *Origin of the Disease.*—The cases which I have given occurred in *thirty-seven* distinct places. In *thirty* of these places the disease did not arise from any known source of contagion. The following are the names of the places:—Colne Bride, Quay-street, Lowerhead-row, Union-row,

Fenton-square, Stock's-buildings, Cliff-end, Lindley, Fartown, Paddock, Clough, near Paddock, Johnny Moore-hill, Brierley Mill, Thomas-street, Cross Church-street, Lane, Ash Brow, Dead Waters, Swallow-street, Rashcliffe, Hill-house, Old Post-office-yard, Cropper's-row, Dyke-end-lane, Old-street, Slaithwaite, Greenside, Milnsbridge, Lime-kilns, Crosland Moor.

If this statement requires any modification it is in respect to Quay-street, (Case 4,) Lindley, (Case 10,) and Cropper's-row, (Case 64.)

In the remaining *seven* places, the disease showed itself in persons coming from an infected district. In only *one* of the seven places did more than one case of cholera occur, (viz., Castle-gate,) and in this instance there was no known communication between the first patient and the one or two subsequent ones. The disease does not seem to have spread, therefore, in a single instance. It is probable that in each of these seven instances the patient acquired the disease in an infected locality, and moved to the place in which the disease broke out during the period of incubation. The following are the names of the seven places:—Vagrant Office, Huddersfield; Bradley Mills, Mold Green, Castle-gate, South-parade, Meltham, Huddersfield Prison.

The disease broke out nearly at the same time in distinct points, at considerable distances from each other, and under circumstances which make it appear in the highest degree improbable that it could have been conveyed from one place to another.

From these facts it seems fair to conclude that the cholera did not arise by contagion in any, perhaps, of the thirty places first mentioned; and that it is impossible to suppose that it could have sprung from contagion in all the thirty places.

II. *Propagation of the Cholera.*—1.—We have just seen that, in seven places, the first persons attacked had come from an infected district, yet the disease did not spread in any one of these instances. At page 17 the particulars have been given of the removal of a family under circumstances highly favouring the spread of a contagious disease, yet without any such effect having followed.

2.—In fifteen out of the thirty-seven places in which the cholera was observed, more cases than one occurred in each place.

In two of these fifteen places (Lindley and Dead Waters) all the cases occurred in the same house. In these places, therefore, it is difficult to judge whether the disease was propagated by contagion, or whether all the cases arose from the same source.

In the thirteen remaining places the cases occurred in more houses than one. Under what circumstances did the disease occur in a second or a third house, after it had appeared in the first one, in these thirteen places?

In two of them it may be regarded as certain that there was no communication between the houses first and those afterwards affected. (Paddock and Slaithwaite.)

In nine of them there was very probably no communication. (Quay-street, Union-street-lane, Swallow-street, Thomas-street, Castle-gate, Rashcliffe, Clough, Cropper's-row (?).)

In two of them there was communication between some houses, and none, probably, between others. (Cliff-end, Johnny Moore-hill.) (a)

(a) Having enumerated the places in the text, any one may verify the facts by a reference to the details previously given. To save trouble to the reader, however, I here subjoin briefly the particulars of a few of the most important places.

Cliff-end.—Five cases in all. 1st case, Sept. 4th. 2nd case, in another house, Sept. 13th; no known communication. 3rd case, Sept. 15th, child of the first patient; attacked after removal to a distance. 4th case, husband of second patient. 5th case, son of the last patient, Oct. 2nd. There was communication between Nos. 2, 4, and 5. If the last got the disease from either of the others, the period of incubation could not have been less than sixteen days.

Thomas-street.—Two cases. 1st, Sept. 7th. 2nd, Sept. 23th. No known communication, and also an interval of three weeks between the two.

Cropper's-row.—Four cases. 1st, Sept. 29th. 2nd, husband of No. 1, Sept. 30. 3rd, son of Nos. 1 and 2, Oct. 1st. 4th, Oct. 1st; this patient once stood for a very short time in the door of the residence of one of the others, but did not enter. There *may* have been contagion; if so, it must have been very virulent.

Slaithwaite.—Seven cases. 1st house.—1st case, Oct. 1st; 2nd case, Oct. 4th. 2nd house.—3rd case, Oct. 4th; no communication with 1 and 2. 3rd house.—4th case, Oct. 7th; 5th, 6th, and 7th cases, Oct. 10th. The inmates of the 3rd house had no communication with the two other houses.

Clough.—1st house.—1st case, Sept. 6th. 2nd house.—2nd case, Sept. 17th, four or five houses from 1st; probably no communication, patient aged 78; 3rd case, grand-daughter of second patient. 3rd house.—4th case Sept. 22nd; cannot say whether any communication, patient aged two years 4th house.—5th case, Sept. 22nd, aged one year; 6th case, aged four years, Sept. 23; communication possible.

Johnny Moore-hill.—1st house.—1st and 2nd cases, Sept. 19th; 3rd, Sept. 22nd, husband of No. 1; 4th, daughter of Nos. 1 and 3, Sept. 25th. Nos. 3 and 4 *might* be from contagion.

2nd house.—1st case, in night between Sept. 19th and 20th; too soon to have sprung by contagion from first house, even if any communication, which is improbable. Two other houses are interposed between first and second houses.

3rd house.—1st case, in night between Sept. 20th and 21st; daughter-in-law of first patient in first house. Lived in a different row of houses, but nursed mother-in-law. If by contagion, only twenty-four hours incubation. 2nd case, Sept. 27th; began with cholera, but with diarrhœa sooner. Husband of last patient.

The two first cases in house 1, and the case in house 2, if not also the first case in house 3, probably all arose from one cause, and not from each other. The last is the only one of them in which contagion seems *possible*.

4th house.—1st case, evening of Sept. 21st; the patient worked a mile off home, and had diarrhœa when at work during the day; lived in a different row from house 1. Had no communication. Began same day as first patient in house 3. No communication.

2nd and 3rd cases, in night between 22nd and 23rd Sept.; the father and mother of No. 1. 4th case, Sept. 25th, another son. The three last *may* have been from contagion.

5th house.—Near to 2nd house. 1st case, a child, Sept 21, (diarrhœa only?) Probably no communication.

6th house.—1st case, Sept. 22nd; denied any communication with preceding. This house is in Lower-row, seven doors from house 1, Lower-row, next house to No. 2.

7th house.—1st case, Sept. 22nd; *probably* no communication, but not *certain*.

8th house.—Next to last. 1st case, Sept. 23rd; can't say whether had any communication with house 6; had not with others. If contagion, period of incubation could not exceed twenty-four hours.

9th house.—Lower-row, next to house 5. 1st case, Sept. 24th, diarrhœa, (and on 15th October became cholera?) might have and probably had communication with house 5.

10th house.—Next to No. 1. 1st case, Sept. 24th; one or two had diarrhœa. A child removed, and died elsewhere of cholera. Might have had communication with house 1.

(a) Since the above remarks were written, I have seen the "Report of the General Board of Health on the Epidemic Cholera of 1848 and 1849." At page 34 cases are referred to which strongly support the opinion that the disease, caught by visiting an infected place, was taken from the *place* rather than from the *patients*.

In two cases (Nos. 63 and 54) the disease was undoubtedly contracted by visiting an infected place. But whether it was acquired by contagion, or by exposure to certain local influences, independently of contagion, it is not easy to determine. Another case, (Mrs. Brooke's, described at page 16,) in which a person, having left her home at Johnny Moore-hill, for fear of cholera, returned to it after a time, and was taken ill before holding any communication with her neighbours, would point to the inference, that the disease was acquired from the place rather than from the people. But there is room for doubt whether the illness in this case was an effect of the choleraic poison. (a)

Thus, a careful analysis of the cases leads me to the conclusion that in some of them the disease *certainly* was not propagated by contagion, and that it *probably* was not in most of them. In others, again, it *may* have been, and in a few it is not improbable (although it is by no means certain) that it was propagated by contagion.

If cholera were propagated exclusively by contagion, the poison must be the most virulent, and the most certain in its operation of any of the contagious poisons known to us. Small-pox, measles, and scarlet fever, of all contagious febrile diseases, are perhaps those which are the most certain to be communicated to exposed persons who have not already suffered from them. But these diseases are never observed to spread with a rapidity which admits of an instant's comparison with that of cholera. In the case of cholera, one house is affected to-day, two or three others, near or distant, to-morrow, and as many more each succeeding day, until a whole neighbourhood has been devastated within a period of time that would barely suffice for the propagation of any other contagious fever from one patient to half a dozen more. If the contagion of cholera were so virulent as this opinion supposes, comparatively few persons visiting an infected district could hope to escape the disease; and persons removing from an infected place would *generally* become the centres of a fresh circle of cases, whereas, in this district, the disease never spread from such persons. Washerwomen, and those employed in attending to the dead, would almost always suffer; yet, in the few cases in which I have had the opportunity of inquiring, these persons do not appear to have suffered at all, except when they were at the same time exposed to the other supposed causes of the disease.

Again, diarrhœa accompanied the cholera in such a manner as to lead almost inevitably to the conclusion, that both diseases depended upon the operation of the same poison. In one class of persons, and under one class of circumstances, diarrhœa resulted; in another class, or under other circumstances, cholera. Diarrhœa began to be epidemic a little before, and ceased to be so a little after the cholera. It prevailed to an extent to which, I believe, it is never observed to prevail, except when in conjunction with cholera. Both diseases visited the same places to a great extent. In some houses, villages, and towns, one of these diseases; in others both, occurred. But very few places altogether escaped both. (See the account off Cliff-end and Lindley, pp. 4 and 5.) Within a few days after the appearance of the epidemic in a neighbourhood, it often happened that a great majority of the houses had been visited either by diarrhœa or by cholera. It is not possible to suppose that diarrhœa, when observed under such circumstances, could be propagated to any material extent by contagion; and, if this statement be true of the diarrhœa, it can scarcely be less true of the cholera. (a)

2.—ENDEMIC INFLUENCES.

Having arrived at the conclusion, that the epidemic cholera neither arose nor was propagated, exclusively, nor even to a very material extent, by contagion, I proceed now to inquire what influence was exercised in the production of the disease by causes of a local character.

In *all* the cases in which I have had an opportunity of making the inquiry, (and that is in nearly all of them,) some of the local causes, whose influence is now generally recognised, were found to exist. Many of the places in which the cholera broke out, and all those in which it prevailed to any extent, were previously well known for their unhealthiness, and have been noted for their liability to fever and other epidemic diseases. The following circumstances, either singly or in various combinations, are those which have been chiefly remarked in the places visited by the epidemic:—

1. The accumulation of filth of various kinds, foul privies, bad drains, or the absence of drains, and want of personal cleanliness.

The good effects of cleanliness are strikingly exhibited by the fact, that only one house at Johnny Moore-hill was free

(a) Abundant evidence will be found in the Report of the General Board of Health, already referred to, in support of the opinion, that the epidemic cholera and the diarrhœa, prevalent at the same time, were due to the action of the same poison. See p. 89 et seq.

from both diarrhœa and cholera, and that the occupants of that house were distinguished from their neighbours by their superior cleanliness. (See pp. 19 and 20.)

2. Overcrowded dwellings and bad ventilation. The bad effects of these circumstances were especially seen at Johnny Moore-hill, where they were observed in almost every house, and where the disease prevailed most in the most crowded houses. (See p. 20.) The good effects of removing from such places are noticed at p. 20.

3. A number of the cases occurred in low situations, and near to a river or canal. (See Cases 5, 25, 82, 83, 86, 87, 88, 89, 91.)

The disease broke out in a very severe form, but did not prevail extensively, in several elevated and very airy situations, (Cliff-end, Lindley, Clough, near Paddock, and even Johnny Moore-hill.) In all these instances, the ordinary local causes were present in a high degree, and seem to have operated injuriously, in spite of the beneficial influence of an otherwise pure air.

4. There were abundant materials for the production of malaria in several places, *e. g.*, the old fish-pond near to Paddock and Cliff-end; also at Dead Waters, a marshy locality, as the name indicates. It has been stated (p. 18) that, before the cholera appeared at Johnny Moore-hill, the wind blew from the fish-pond towards the dwellings. After this time the wind was generally easterly, and it is possible this circumstance may have had something to do with the immunity of the general village of Paddock, and with the spread of the disease to several places west of Paddock, as Milnsbridge and Slaithwaite.

5. The condition of the water was inquired into in most places. There was no reason to suppose that it was contaminated in any instance, unless in the case of two families, who had used the water in the old fish-pond in July and August. (See p. 18.)

The influence of the local causes just enumerated appears to have been increased by the personal state or habits of various individuals. Such are—

1. *Intemperate Habits.*—These are noticed in several cases, and certainly existed in many more.

2. One patient was attacked soon after eating cucumbers and fat meat. (Case 2, p. 2.)

3. *Previous Liability to Diarrhœa.*—In Cases 10 and 41 this is mentioned. In one instance, however, an individual subject to diarrhœa, and exposed to the cholera, escaped all further illness. (P. 11, remarks on Case 76.)

In one case the disease came on after purgative medicine had been taken, the patient being exposed at the same time to all the other causes of cholera. (Case 16.)

4. Fatigue, poor food, and exposure to cold and wet, seem to have exercised considerable influence in Case 1.

5. Great fear of the cholera preceded its appearance in several cases. (10 and 11.)

6. *Adult age* appears to have been a predisposing cause. (See p. 20.)

Thus, in this neighbourhood, the disease visited those places only in which one or more of the local influences just referred to were to be found. The same statement, with comparatively few exceptions, is equally applicable to the country at large. We are forced, therefore, to the conclusion, that these local influences are intimately connected with, and that in most, although not in all cases, they may even be said to be necessary to, the development of the disease.

In this fact is probably to be found the explanation of another, viz., that the villages near the town were more severely visited by cholera than the town of Huddersfield itself. The hygienic condition of many of the villages is worse than that of the town. In the epidemic of 1832 not more than one or two cases of cholera occurred in Huddersfield; and I have not heard that any more were seen in the neighbourhood. A large part of the town stands upon a gentle declivity. Its position offers natural facilities for drainage; and the elevation and the arrangement of the streets render it more likely to be well ventilated by the frequently high winds than are many other towns. In 1832 active preventive measures were very generally adopted, under the apprehension of a visit from the epidemic; and these, no doubt, contributed largely to the immunity which the district at that time enjoyed. Since then the rapid increase in the population has led to a

multiplication of local nuisances; and the usual consequences of these would probably have been experienced in a more severe visitation from the last epidemic, had it not been for the efforts of the Commissioners not long before appointed under a new improvement Act. These gentlemen, with an enlightened appreciation of their duties, and in the face of opposition raised on account of the expense, caused many of the worst nuisances to be removed long before the cholera came among us. The jurisdiction of the Commissioners does not extend to those smaller places which were most severely visited, and which, instead of the moderate outlay occasioned by sanitary improvements, have now to bear the far heavier and more permanent expense of maintaining the widows and orphans of those who have been prematurely cut off.

One of the most remarkable singularities of the epidemic cholera is this: that after attacking one, two, or three persons in a bad locality, it will cease there. Again, it will altogether pass by other neighbouring spots, as bad or worse than the one visited. One house will be invaded, whilst others much nearer to the nuisance, supposed to be concerned in the production of the disease, will escape. (See remarks on the cases at Lindley, at Fountain-street, and at Cliff-end.) Upon any hypothesis respecting the origin of cholera, I can only suggest one explanation of these facts, viz., that of a number of persons exposed in an infected place, some are but little or not at all susceptible of the disease; some are exposed to the poison during a shorter period of time, or in a greater state of dilution; others escape it altogether, from changes in the wind, &c.; and the rest are attacked, some with cholera, some with diarrhœa or dysentery, and others with cramps, pains in the bowels, indigestion, and other forms of indisposition.

3.—ATMOSPHERIC PECULIARITIES.

But will air, rendered impure by emanations from filth and foul drains, and by want of ventilation, of itself explain the origin and propagation of cholera? I believe it will not; for all these sources of impurity have existed all over the

country, and in a greater degree than they now do, for years before the epidemic cholera appeared. If this cause were an adequate one, I do not see why the disease should not revisit us every year.

We are forced, then, I think, to adopt the hypothesis that, in addition to local nuisances, and probably also to contagion, there must be some peculiar condition of the atmosphere. That remarkable and unusual atmospheric phenomena of various kinds were observed in many places, is a matter of fact, and has been recorded in several publications upon the disease. The precise nature of these changes remains yet to be ascertained.

Upon this hypothesis we can explain most of the circumstances attending the origin and propagation of the cholera; its simultaneous eruption in separate and distant spots in the same neighbourhood; the escape of other individuals or places apparently equally exposed; the very rapid diffusion of the disease (like influenza) in the same town and over large districts, or even over a whole country; the occasional eruption of it in ships at sea, and also its progress from one country to other and distant countries. When the poisoned atmosphere passes over a town or village, it operates principally upon those who inhabit the worst places, either because its noxious effects are increased by the local atmospheric impurities, (which is probably the principal reason,) or because the persons who inhabit such places, from being less favourably situated in respect to food, clothing, habits, and mental condition, are more liable than others to suffer from any cause of disease applied to them.

The facts I have stated render it impossible for me to believe that cholera was *commonly* propagated by contagion in this district; but I by no means deny, as I have already intimated, that it may have been so propagated in some instances. Many cases, moreover, have been published, upon the authority of trustworthy observers in other places, which seem to prove the contagiousness of the disease. There seems to be nothing to forbid the supposition, that a disease which arose from local impurities or from atmospheric influences may afterwards

spread by contagion. Typhus, small-pox, measles, &c., must, in the first instance, have arisen without contagion, yet it is certain they are now propagated by contagion. Why may not the causes which *first* produced these diseases, frequently *reproduce* them now? Is it not probable, in fact, that they do? If so may not the same thing occur in the case of cholera? I will only add, in conclusion, that I have collected the *facts* I have recorded with much care, and have endeavoured to reason upon them without any bias. Before I analyzed these facts, I had never formed, nor ever expressed, any opinion respecting the mode of origin and propagation of the cholera. The opinion I have now formed and expressed has been suggested to me solely by my own personal inquiries.

Period of Incubation.—When the cholera has been acquired in a particular place, whether through contagion or through other local influences, it is desirable, when possible, to determine the period of incubation of the poison. In most of my cases it is not possible do this, because the patients lived amongst, and so had been long exposed to, the nuisances which probably had a share in producing the disease. Three cases, however, afford more precise information upon this point. In Case 63 the disease broke out within three days of the time when the patient first visited the spot at which he must have imbibed the poison. In Case 54 the disease broke out in about three and a half days, or eighty-four hours, after the first exposure. In Case 21 the patient, a child, left an infected place, and was not attacked until ten or eleven days after, having removed to a spot where there was no other case of cholera either before or after.

Treatment, Mortality, &c.—There is nothing deserving of remark in the treatment of the cholera at Huddersfield. Various plans were tried by various practitioners, but the cases were too few in number to admit of any plan being tested upon a large scale. The mortality did not differ materially from that observed in many other places. The deaths were 39, the recoveries 54. These numbers are given only because a widely different account has been published.

The treatment of the premonitory diarrhœa was very

generally successful; and, by adopting the plan of house-to-house visitation, many such cases were discovered and stopped, and no doubt many lives were saved.

My experience of the last epidemic would lead me, in the event of another such visitation, to urge the great importance of house-to-house visitation, with a view to the discovery and treatment of all cases in the early and still manageable stages; the removal of the healthy from infected and over-crowded houses to places of refuge in proper situations; the clearing away of all nuisances; lime-washing the houses, and other similar measures.

The most beneficial measures, however, are those sanitary improvements which can only be carried out at leisure, and when no epidemic is pressing upon us. By these means, no doubt the cholera, and also many other diseases, might be, to a great extent, prevented, and much suffering and many lives might be spared. The want of proper persons, possessing the requisite authority to undertake this duty, in some cases, and a mistaken view of the expense in others, have hitherto prevented it from being discharged in a manner adequate to meet existing evils. The great fact, established upon most ample evidence, that it costs very much less to remove nuisances, and thereby to prevent disease, than to maintain the families of disabled men, and to make provision for their widows and orphans, is not yet sufficiently known to, or is not properly appreciated by, the local authorities of most towns and villages. The result is that, in this district as elsewhere, if cholera returned to-morrow, it would find us unprepared to stay its progress. The places that were cleansed have generally relapsed into their former filthy condition, and would afford all the facilities for the development and spread of the disease which they unhappily supplied during the last epidemic. It is to be hoped, that the Huddersfield Improvement Commissioners, who have shown that they understand what is required of them, will not be prevented from doing it effectually within the limits of their jurisdiction, by any short-sighted outcry against the necessary expenditure.

Nearly the whole of the preceding report was written in

December 1849, and January 1850. Since it was completed, the Report of the General Board of Health, already referred to, has been published. The results arrived at are the same in every important particular. The inquiries were totally unconnected with each other.

Huddersfield, December, 1850.

LONDON:
PRINTED BY WILLIAM TYLER:
BOLT-COURT.

CHOLERA IN HUDDERSFIELD, BY JOHN TAYLOR, M.D.

Number and Name of Patient.	Age.	Date of Attack.	Occupation.	Residence.	Died or Recovered.	Medical Attendant.
57. Wright Cawood	76	— 26	...	Windsor-court, Castle-gate.	Do.	Mr. Clarke.
58. John Robinson	29	— 26	Excavator.	054 Post-office-yard.	Do.	Mr. Tatham.
59. Mary Ann Ferguson	39	— 26	... ...	Leeds-road, near Lane.	Died.	Mr. Booth.
60. Jane Taylor	5	— 27	... ...	Johnny Moor-hill.	Recovered.	Mr. Allatt.
61. David Taylor	28	— 27	Cloth Dresser.	Do.	Do.	Mr. Tatham.
62. Josh. Shepherd	43	— 25	Do.	Rash-liffe.	Died.	Do.
63. Richard Berry	63	— 29	Mason.	Cropper's-row.	Do.	Mr. Clough.
64. Mary Goodyear	59	— 29	... ...	Do.	Recovered.	Mr. Clarke.
65. John Goodyear	45	— 30	Finisher (husb. of No. 64).	Union-row.	Do.	Mr. Sissons.
66. Mary Walshaw	3	— 30	House-ports.	Meltham.	Died.	Mr. Rawcliffe.
67. John William Taylor	3	Oct. 1	(Son of Nos. 64 and 65)	Cropper's-row.	Recovered.	Mr. Clarke.
68. John Woodhead	... ...	— 1	Commercial Traveller.	Dog-house-yate.	Do.	Mr. Sissons.
69. Robert Whitehead	30	— 1	Hand-loom.	Old-street.	Do.	Do.
70. Harriet Marriott	74	— 1	Cloth Dresser.	Cropper's-row.	Died.	Mr. Booth.
71. Mary Ann Berry	17	— 1	Barber.	Slaithwaite.	Do.	Mr. Tatham.
72. Thomas Berry	17	— 2	... ...	Cartworth.	Recovered.	Mr. Tatham.
73. Hannah Pick	17	— 2	... ...	Do.	Do.	Do.
74. Margaret Heaton	37	— 2	... ...	Do.	Do.	Do.
75. Maria Moxon	38	— 2	(Son of No. 23).	Cliff end.	Died.	Dr. Robinson.
76.	...	— 3	... ...	Folly-hall.	Recovered.	Mr. Sissons.
77. Ruth Berry	... ...	— 3	Nursemaid.	Green-side.	Do.	Do.
78. Emma Wilcock	...	— 3	... ...	Sleep-close.	Do.	Mr. Tatham.
79. Hannah Charlesworth	61	— 3	... ...	Milnthorpe.	Died.	Dr. Robinson.
80. John Saville	57	— 3	Farmer.	Slaithwaite.	Do.	Mr. Roberts.
81. Josh Gledhill	30	— 4	(Son of No. 72).	Do.	Do.	Do.
82.	18 months.	— 4	... ...	Do.	Do.	Do.
83. Charles Varley	36	— 7	Clothier.	Do.	Do.	Mr. Dean.
84. Susan Shaw	41	— 8	... ...	Lingards.	Recovered.	Mr. Tatham.
85. Hannah Clarkson	42	— 10	... ...	Ash house.	Do.	Do.
86. Mrs. Varley	57	— 10	(Mother of No. 83).	Slaithwaite.	Died.	Mr. Roberts.
87. Mr. C. Varley	69	— 10	(Husband of No. 86).	Do.	Recovered.	Do.
88. Mrs. C. Varley	... ...	— 10	(Wife of No. 83).	Do.	Do.	Do.
89. Mrs. Shaw	... ...	— 11	... ...	Dead-waters.	Do.	Mr. Sissons.
90. Mrs. Jackson	36	— 11	Cloth Dresser	Rashcliffe.	Do.	Mr. Booth.
91. Rachel Thorp	...	— 22	... ...	Dead waters.	Do.	Mr. Tatham.
92. David Sykes	50	Nov. 1	... ...	Hollin-hall-green.	Do.	Mr. Clarke.
93. George William Taylor	2	— 21	... ...	Crosland-moor.	Died.	Mr. Allatt.

Number and Name of Patient.	Age.	Date of Attack.	Occupation.	Residence.	Died or Recovered.	Medical Attendant.
		1849.				
1. Francis Donoghue	50	Jan. 18	Tramp.	Attacked in Vagrant Office.	Died.	Mr. Clarke.
2. Mrs. Thomas	38	July 9	House-work.	Colne-bridge.	Recovered.	Mr. Booth
3. Josh. Whitaker	27	Aug. 9	Mill-worker.	Bradley Mills.	Do.	Mr. Clarke.
4. William Leonard	38	Aug. 22	Gardener.	High-st., attacked in Quay-st.	Died.	Mr. Rhodes.
5. Mrs. Hardy	65 (?)	— 26	... ...	Lower-head-row.	Recovered.	Mr. Tatham.
6. Mrs. Gibson	... ...	— 29	... ...	Union-row.	Do.	Mr. Sissons.
7. Mrs. Kershaw	... ...	— 30	Factory-worker.	Fenton square.	Do.	Do.
8 James Atkinson	50	— 30	... ...	Stock's-buildings.	Died.	Mr. Tatham.
9. Grace Bake	29	Sept. 4	... ...	Cliff-end.	Do.	Mr. Machill.
10. Mrs. Hirst	53	— 4	... ...	Lindley.	Do.	Do.
11. Jonathan Booth	... ...	— 5	Factory-worker.	Fa'town.	Recovered.	Mr. Sissons.
12. William Rhodes	45	— 6	Mason.	Clough (Paddock).	Do	Mr. Allatt.
13. Abraham North	35	— 7	Weaver.	Thomas-street.	Do.	Mr. Greenwood.
14. Mrs. Ganter	35 (?)	— 8	Shopkeeper.	Cross Church-street.	Do.	Mr. Tatham.
15. John Leonard	... ...	— 9	Factory-worker.	Quay-street.	Do.	Mr. Sissons.
16. Mrs. George Crosland	30	— 9	... ...	Lindley.	Died.	Mr. Machill.
17. Josh. Thornton	... ...	— 9	Slubber.	Lane.	Recovered.	Mr. Booth.
18. Martha Walker	60	— 11	Mangler.	Paddock.	Do.	Mr. Allatt.
19. Mary Craven	35	— 11	Nurse.	Lindley.	Died.	Mr. Machill.
20. ——— ——— F.	72	— 13	... ...	Cliff-end.	Do.	Dr. Robinson.
21. Grace Bake's Child	1½	— 15	... ...	Attacked at Mold-green.	Do.	Mr. Booth.
22. Henry Beaumont	27	— 16	Cloth Miller.	Union-street.	Do.	Do.
23. ——— ——— M.	70 or more.	— 16	(Husband of No. 20).	Cliff end.	Died.	Dr. Robinson.
24. John Stocks	30	— 17	Spinner.	Swallow-street.	Recovered.	Mr. Maxon.
25. Sarah Tomlinson	78	— 17	... ...	Clough (Paddock).	Died.	Mr. Allatt.
26. Josh Hinchliffe	61	— 17	Labourer.	Paddock.	Recovered.	Do.
27. Mrs. Hirst	53	— 18	... ...	Brierley Mill.	Died.	Do.
28. James Leonard	52	— 18	... ...	Swallow-street.	Recovered.	Mr. Sissons.
29. Hannah Taylor	55	— 19	... ...	Johnny Moore-hill.	Died.	Mr. Allatt.
30. William Moss	23	— 19	Excavator.	Do.	Do.	Do.
31. John Alpine	40	— 20	Excavator.	Do.	Do.	Do.
32. Sarah Taylor	24	— 21	(Wife of No. 61).	Do.	Do.	Do.
33. Mary Tomlinson	20	— 21	... ...	Clough.	Recovered.	Do.
34. Allen Micklethwaite	18	— 21	Cloth Dresser.	Johnny Moore-hill.	Died.	Do.
35. Hannah Micklethwaite	46	— 22	(Wife of No. 36).	Do.	Recovered.	Do.
36. John Micklethwaite	44	— 22	Weaver.	Do.	Died.	Do.
37. Thomas Wood	45	— 22	Engineer.	Dock-yard.	Recovered.	Mr. Booth.
38. Aaron Taylor	56	— 22	Cloth Dresser.	Johnny Moore-hill.	Do.	Mr. Allatt.
39. Ezra Whiteley	2	— 22	... ...	Do.	Died.	Do.
40. Charles Potter	29	— 22	Excavator.	Do.	Do.	Do.
41. Thomas Dyson	1	— 22	... ...	Clough.	Do.	Do.
42. Eli Wilson	... ...	— 23	Factory-worker.	Lane.	Do.	Mr. Sissons.
43. William Byrom	43	— 23	Labourer.	Johnny Moore-hill.	Recovered.	Mr. Allatt.
44. Mary Ann Byrom	7	— 23	(Child of Nos. 43 and 55).	Do.	Do.	Do.
45. Jane Dyson	4	— 23	... ...	Clough.	Died.	Do.
46. James Dransfield	14	— 24	Factory-boy.	Johnny Moore-hill.	Recovered.	Do.
47. Sally Byrom	2	— 24	(Child of No. 55.)	Do.	Died.	Do.
48. Patrick Connell	26	— 24	Excavator.	Castle-gate.	Recovered.	Mr. Clarke.
49. Rachael Dransfield	18	— 25	... ...	Johnny Moore-hill.	Do.	Mr. Allatt.
50. Mrs. Kaye	... ...	— 25	House-work.	Rashcliffe.	Do.	Mr. Sissons.
51. Martha Brown	19	— 25	... ...	Thomas-street.	Do.	Mr. Tatham.
52. Ann Taylor	23	— 25	(Daughter of No. 38).	Johnny Moore-hill.	Do.	Mr. Allatt.
53. Samuel H. Micklethwaite	21	— 25	(Son of Nos. 35 and 36).	Do.	Do.	Do.
54. J. Taylor	... ...	— 25	Physician.	South Parade.	Do.	Mr. Greenwood, Mr. Clarke, and Mr. Booth.
55. Ann Byrom	28	— 25	(Wife of No. 43).	Johnny Moore-hill.	Do.	Mr. Allatt.
56. Mrs. Wilson	... ...	— 26	(Wife of No. 42).	Lane.	Died.	Mr. Sissons.
57. William Eastwood	39	— 26	... ...	Hill-house.	Recovered.	Mr. Tatham.
58. John Robinson	29	— 26	Excavator.	Windsor-court, Castle-gate.	Do.	Mr. Clarke.
59. Mary Ann Ferguson	30	— 27	... ...	Old Post-office-yard.	Do.	Mr. Tatham.
60. James Jagger	5	— 27	... ...	Leeds-road, near Lane.	Died.	Mr. Booth.
61. David Taylor	28	— 27	Cloth Dresser.	Johnny Moore-hill.	Recovered.	Mr. Allattt.
62. Josh. Shepherd	42	— 28	... ...	Lane.	Do.	Mr. Tatham.
63. Richard Berry	62	— 29	Man Nurse.	Rashcliffe.	Died.	Do.
64. Mary Goodyear	39	— 29	... ...	Cropper's-row.	Do.	Mr. Clough.
65. John Goodyear	45	— 30	Finisher (husb. of No. 64).	Do.	Recovered.	Mr. Clarke.
66. Mary Walshaw	... ...	— 30	House-work.	Union-row.	Do.	Mr. Sissons.
67. John William Taylor	3	— 30	... ...	Meltham.	Died.	Mr. Rawcliffe.
68. John Goodyear	5	Oct. 1	(Son of Nos. 64 and 65).	Cropper's-row.	Recovered.	Mr. Clarke.
69. Robert Whitehead	... ...	— 1	Commercial Traveller.	Dyke-end-lane.	Do.	Mr. Sissons.
70. Harriett Marriott	... ...	— 1	House-duties	Old-street.	Do.	Do.
71. Abraham Smith	39	— 1	Cloth Dresser.	Cropper's-row.	Died.	Mr. Booth.
72. Mary Quarmby	74	— 1	Barber.	Slaithwaite.	Do.	Mr. Dean.
73. Hannah Pick	17	— 2	... ...	Castle-gate.	Recovered.	Mr. Tatham.
74. Margaret Heaton	37	— 2	... ...	Do.	Do.	Do.
75. Maria Moxon	32	— 2	... ...	Rosemary-lane.	Do.	Do.
76. ——— ——— M.	... ...	— 2	(Son of No. 23).	Cliff end.	Died.	Dr. Robinson.
77. Ruth Berry	... ...	— 3	... ...	Folley-hall.	Recovered.	Mr. Sissons.
78. Emma Wilcock	... ...	— 3	Nursemaid.	Green-side.	Do.	Do.
79. James Charlesworth	61	— 3	... ...	Sheep-close.	Do.	Mr. Tatham.
80. John Saville	57	— 3	Farmer.	Milnsbridge.	Died.	Dr. Robinson.
81. Josh. Gledhill	50	— 4	(Son of No. 72).	Slaithwaite.	Do.	Mr. Roberts.
82. ——— ———	18 months.	— 4	... ...	Do.	Do.	Do.
83. Charles Varley	36	— 7	Clothier.	Do.	Do.	Mr. Dean.
84. Susan Shaw	41	— 8	... ...	Lime kilns.	Recovered.	Mr. Tatham.
85. Hannah Clarkson	42	— 10	... ...	Ash brow.	Do.	Do.
86. Mrs. Varley	57	— 10	(Mother of No. 83).	Slaithwaite.	Died.	Mr. Roberts.
87. Mr. Varley	60	— 10	(Husband of No. 86).	Do.	Recovered.	Do.
88. Mrs. C. Varley	... ...	— 10	(Wife of No. 83).	Do.	Do.	Do.
89. Mrs. Shaw	... ...	— 21	... ...	Dead-waters.	Do.	Mr. Sissons.
90. Jasper Dawson	36	— 22	Cloth Dresser.	Rashcliffe.	Do.	Mr. Booth.
91. Richard Thorpe	... ...	— 28	... ...	Dead-waters.	Do.	Mr. Sissons.
92. David Sykes	50	Nov. 1	... ...	Huddersfield-prison.	Do.	Mr. Clarke.
93. George William Taylor	2	— 21	... ...	Crosland-moor.	Died.	Mr. Allatt.

THE

STETHOMETER:

AN INSTRUMENT FOR ASCERTAINING THE DIFFERENCE IN THE MOBILITY OF OPPOSITE SIDES OF THE CHEST,

And thus facilitating Diagnosis.

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[*Reprinted from the London Journal of Medicine.*]

LONDON:
RICHARD'S, PRINTERS, 100, ST. MARTIN'S-LANE.

THE STETHOMETER:

AN INSTRUMENT FOR ASCERTAINING THE DIFFERENCE IN THE MOBILITY OF OPPOSITE SIDES OF THE CHEST.

It is well known, that the movements of the walls of the chest, during respiration, correspond to, and are influenced by, the state of the organs placed within or beneath them. Hence, then, in our investigations of the diseases of these organs, we invariably, and frequently in the first instance, examine these movements. In many cases, the modifications produced by disease are so great as to be at once evident to the eye, or to the eye assisted by the hand. In other, and much more numerous cases, the eye and hand cannot detect these alterations. Indeed, nothing is more common than to find observers, at the same moment, differing in opinion as to the greater or less mobility of a portion of one or other of the sides of the chest. Even the indefinite terms here used, "greater" or "less", become sources of fallacy and doubt. It is, therefore, very desirable that a mode of investigation, admitted by all to be most important, should be exact for observation, and definite for description. I felt this more particularly some years ago, when engaged in clinical demonstrations to students, of the different modes of investigating diseases of the chest; and I at that time endeavoured by various contrivances, constructed chiefly on the principle of the callipers, to surmount the difficulty. My friend, Dr. Sibson, was, however, then more successful than myself; and he introduced an instrument which, in his hands, has afforded very important results in the investigation of the respiratory movements of the chest and abdomen, both in health and disease.[1] Subsequent consideration led me to believe that an instrument, simple, portable and economical, capable of ready application and of affording very valuable

[1] Medico-Chirurgical Transactions, vol. xxxi.

information, might be constructed, on a different principle to any which I had previously seen. I explained my requirements to Mr. Delolme, chronometer maker, whose ingenuity has supplied many ingenious and beautiful contrivances of an analogous kind. The result has been the little instrument which I am now going to describe, and which has, in the hands of those who have used it, as well as in my own, been found very efficient for the intended object: viz., to measure the comparative mobility of opposite sides of the chest. It is susceptible of many other applications, to some of which I shall again refer, and which will, no doubt, suggest themselves to those into whose hands it may fall.

DESCRIPTION. The little instrument (see fig. 1[1]) consists of a flat case, not unlike a watch-case: on its upper surface is a graduated dial and an index, which stands at a maximum number, that may be considered a "zero." This case contains a very simple movement, by means of which the index can be acted on. A silk cord, which may be of a sufficient length to surround one-half, or the whole circumference of the chest, passes through an aperture in one side of the case. This cord acts on the index. When the cord is drawn out, or extended for the space of one quarter of an inch, it will be observed that the point of the index will once traverse the circumference of the graduated dial. In other words, when the index has gone once round, it shows that the cord has been extended one fourth of an inch. It will be further seen, that the dial is graduated, or divided, into fifty equal parts. Each of these parts is, therefore, equal to the fiftieth part of a quarter of an inch; that is, to the 1-200th of an inch.[2] The index is further capable of going round a second time, on an additional quarter inch of the cord being drawn out. Hence, two revolutions of the index are equal to half an inch of movement—an extent of motion sufficient for all practical purposes. The pedestal and circular foot, shown in the same figure, will be subsequently referred to.

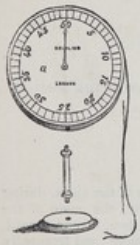


Fig. 1.

MODE OF APPLICATION. It is quite evident, that if the instrument be so placed that extension be made on the cord, the amount of the extension will be shown by the movement of the index on the dial. For example (as in fig. 2) if the instrument is laid flat on the spine, and held in its place by the first and second fingers of the left hand, whilst the cord is carried round the chest, and pressed on one of the ribs, or the sternum, by the fingers of the right hand, then, when the individual under examination expands the chest during inspiration, the amount of expansion will be communicated to the cord, and thus indicated on the dial. The cord may then be directed around the opposite side of the

[1] The instrument is reduced in this sketch to half size.

[2] In making and recording observations with this instrument, I am in the habit of expressing a fact thus: right apex, 30; left, 15. It will be understood that the motion is as 15 to 30, without reference to these figures being eighths, fiftieths, or two hundredths.

chest, and thus will be at once seen any difference which exists in the relative mobility of the two at the point under examination. It will, of course, be absolutely necessary in every examination, such as this, that corresponding parts of the chest be compared. For all useful purposes it may be said, that the movements of the opposite sides of the chest, in health, are identical—the difference which exists over the region of the heart, at the left side, is too immaterial to interfere with practical conclusions. The instrument may be applied to any part of the chest in the mode here described. In figure 3 it is shown as applied on the sternum, and beneath one of the clavicles. The latter position is one of considerable importance, from its connexion with the deposition of tubercle towards the summit of the lung. I find that on applying the instrument here the cord may, in this instance, be directed towards the arm more conveniently than in any other direction, as shown by the black line, and pressed against a point near the insertion of the deltoid muscle. The cord may also be directed downwards, or outwards, and upwards, or inwards, (as shown by the dotted lines,) and retained on any fixed point. Nor, as is evident, need this point be a part of the body. It may be on the bed, or on a chair, etc., always providing that the direction of the cord be such as to receive the impression of the movement of the part of the body under examination, and that its position be symmetrical at both sides.



Fig. 2.

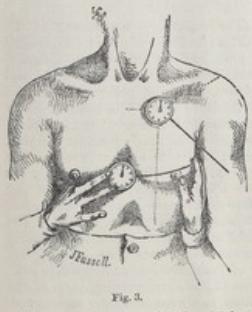


Fig. 3.

The use of the pedestal, which can be fixed in the side of the case with the foot attached, is shown in Figure 4. The instrument

thus used is intended to ascertain the modifications of the movements over a limited or circumscribed spot. On this spot, the foot is placed and held, (as shown in the figure,) by the fingers of the left hand, the cord directed towards this point is held between the fingers of the right hand. These fingers, being thus made the fixed point, must be kept steady and not allowed to touch the part under examination. The spring in the instrument is sufficiently strong to resist the gentle tension made by the fingers, and the instrument itself being pushed forward or raised by the elevation of the part during inspiration, the movement of the index, as when the case is applied on the flat surface, becomes the measure of this elevation.



Fig. 4.

Such being the mode of using the instrument, it will be necessary to say a few words on the precautions which are required in securing accuracy in the result. 1st. It is absolutely imperative that corresponding portions of the chest be examined, and that the mode of applying the instrument, and the point at which it is applied at each side, be identical. 2nd. Care must be taken that the patient breathes in the same manner whilst opposite sides are being examined. 3rd. It should be seen that the cord, when the observation is commenced, is held sufficiently tense to act on the index.

All this requires attention and some little effort; for, as in all other matters, there is little that is of any value which can be obtained without some labour. Though to some few patients these carefully conducted examinations may be irksome, yet to the vast majority they are far otherwise. In the latter, they beget confidence in the medical attendant, and frequently at once inspire a feeling which renders all future intercourse not less pleasing than it is conducive to successful treatment.

Uses of the Instrument. The expansive movements of the chest during inspiration may be said to be the measure of the capacity of the lungs for air. So they are; and by an accurate measurement of the one, we might estimate tolerably correctly the other, and thus be able to compare the relative breathing powers of different individuals. It is exceedingly difficult to make this comparison by external measurement; for the movements of analogous or corresponding parts of the chest, even in those with unimpaired respiratory powers, are remarkably different in different individuals. For not only are such movements modified by age, sex, and occupation, but they are influenced by peculiarities beyond the influences apparently of any fixed or recognized rule. I therefore do not propose to use this instrument in testing whether A has a better breathing power than B. The spirometer of Dr. Hutchinson is a far more correct means of estimating this quality. I propose my instrument as a means of ascertaining any want of symmetry, independently of malformation, and therefore indicative of disease, in the movements of the corresponding parts of the same chest. It is not my intention to discuss here the various conditions, under which this want of symmetry may occur. Obstruction, for example, of a bronchus by a foreign body in, or by a tumour pressing on it, will interfere with the movements of the side of the chest containing the lung to which such bronchus is distributed. Disease of the substance of the lung, tuberculous, malignant, or inflammatory, and emphysema affecting the air-cells, also prevent the free expansion of the lungs or parts of them affected. Diseases of the pleura, viz., acute or chronic pleurisy; bands binding down the lung; pleuritic effusions or tumours in the pleura, have a like effect. Pleurodynia, and diseases of the external walls of the chest, may, of course, interfere with their free movements. However trifling the degree of limitation of motion, which may be produced by any of these diseases, or by many others not here enumerated, I believe this instrument, if properly used, will inevitably indicate it. When such limitation is thus observed, recourse must be had to the study of the general symptoms, and to the other means of physical diagnosis, to discover on what the irregularity depends. Thus, then, attention may be, in the first instance, directed by the use of the instrument to the seat of disease; or, when disease has been suspected or discovered by other means, mensuration will be useful in confirming the diagnosis, and expressing the extent of the local lesion. In both these senses, I have found the instrument an exceedingly valuable aid in the diagnosis of the earlier stages of phthisis. Indeed, it is quite remarkable how early, and to what an extent, the respiratory movements are modified by tubercular deposit. I hope, however, on some future occasion to return at some length to this and some allied subjects connected with the disease. It will, of course, be evident that if both sides of the chest are equally diseased, we lose the means of comparing them; but such an event is one of very rare occurrence. The instrument can also be used in investigating the movements of diaphragmatic or abdominal respiration, and in taking notice of any irregularity which may be produced in it by disease of any of the organs placed beneath the diaphragm.

My friend, Dr. Alex. Henry, has suggested to me, that not only can the instrument be used for measuring dynamical movements, but it can also be applied in following changes of volume, whether of increase or diminution, in solid tumours or other swellings. For this purpose, the instrument should, on every occasion of an examination being made, be placed on the same spot, and a definite length of cord should be used in encompassing the part examined; any increase of volume will, of course, be shown by the movement of the index. When it is required to follow a diminution of volume, the index, being made to revolve once or twice, should be thus retained, when the examination is commenced; the stages of its return to the zero point will be then the measure of decrease. Having thus given a brief description of this little instrument, of its mode of application, and of its uses, I present it to my professional brethren, with the desire that it may prove as useful to them as it has been to myself, and to those who have already used it.[1]

23, Harley Street, September 1850.

[1] The instrument must have a name, and I have therefore called it a Stethometer. (Στῆθος, *the chest*, Μετρέω, *I measure*.) It may be procured from Mr. Coxeter, surgical instrument maker, 23, Grafton-street East, Fitzroy-square, London, price 26s.

AN INQUIRY INTO THE REASONS WHY THE HORSE RARELY VOMITS.

BY

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[*From the "London Journal of Medicine."*]

THIS subject has, within the last two centuries, engaged the attention of numerous physiologists, the object of whose inquiries, however, has differed somewhat from ours; for the majority of them have been imbued with the prevalent opinion that the horse NEVER vomits. Thus Lamorier[1] addressed to the French Academy of Sciences *Une mémoire où l'on donne les raisons pourquoi les chevaux ne vomissent point;* and M. Flourens[2] recently published a paper on non-vomiting in the horse, heading it with the following dogma, "*Le cheval ne vomit point; c'est là ce que chacun sait.*"

Were it not that M. Flourens is one of the most recent, as well as most renowned, writers on this subject, we should not deem it necessary to contradict this statement, because it has long been known that horses occasionally, though rarely, do vomit. True it is that, since as a general proposition we cannot assert that the horse does vomit, it might be supposed that the converse statement would hold good; and so it does; but only in a sense that is too vague to warrant its being adopted as the title of a vexed scientific question.

In the pursuit of our inquiry, under present circumstances, it being indispensable that we should clear the path from error before we can hope successfully to attain the truth, we shall, firstly, quote cases of horses having vomited; secondly, analyse the opinions which, at various periods, have been propounded on this subject; and, thirdly, adduce reasons to prove why vomiting in the horse is of rare occurrence.

I. CASES OF VOMITING IN THE HORSE. In relating the history of a horse affected with spasmodic colic, M. Charlot[3] makes the following

[1] Histoire de l'Académie Royale des Sciences. Année 1733.
[2] Gazette Médicale de Paris. Année 1849, No. 5, 3 Février, p. 79-81.
[3] Recueil de Médecine Vétérinaire, tome iv, p. 128.

statement: "Speedily, and before me, the animal gathered together its limbs, made an effort, by contracting the abdominal muscles, opened its mouth, and ejected, by the nostrils, a thick, bloody, fœtid liquid, of acid taste, and mingled with fragments of forage. The act of vomiting was repeated in less than half an hour. It gave rise to the evacuation of about two pints of liquid, which had been previously swallowed, and in which two strongyli were contained." M. Tombs[1] states that a bay mare, while on a journey, vomited a gallon and a half of indigested food. Though we do not agree with the explanation which he gives of the phenomenon, his statement of fact respecting it is so plain as to be irrefutable.

These two cases appear to be conclusive evidence to prove that M. Flourens, and many of his predecessors, have sought explanations for a supposed, and not real, fact; and, therefore, have at least incurred the danger which attends those who study nature with imaginations erroneously preoccupied.[2]

II. History of the Subject. Were we to arrange the historical part of the subject chronologically, it might prove tedious; to avoid which, we shall class opinions according to the analogies they present.

1. While acknowledging that the stomach of the horse is situated at a distance from the muscular walls of the abdomen, M. Dupuy[3] attributed, in a great measure, the difficulty of vomiting in that animal to powerful compression of the œsophagus by the muscular fasciculi of the right crus of the diaphragm, in the substance of which it passes. A similar reason was, indeed, hinted at long since by Peyer;[4] but its fallacy is too apparent to need much comment, since, in the same manner as food passes freely down the œsophagus to the cardiac orifice, it may, without obstacle, retrace the same course.

2. A second class of writers have imagined the existence of a valvular apparatus at the cardiac orifice, destined to prevent the return of food into the œsophagus. Among them was Lamorier,[5] who attributed the greater part of the horse's difficulty in vomiting to the impossibility of compression of the stomach by the abdominal walls and diaphragm, alleged that a crescentic valve was so arranged as partially to close the cardiac orifice, and prevent the return of coarse food through it. Dr. Gurlt[6] has figured a spiral valve at the cardia; and Mr. Spooner,[7]

[1] Veterinarian, vol. xii, p. 797.

[2] In a report read by M. Mignon to the Société Centrale de Médecine Vétérinaire, (*Bulletin de la Société*, année 1847, tome deuxième, p. 191, et seq.), on a memoir by M. Valtat, on Vomiting in the Horse, many cases of its occurrence in that animal are related. We refer to this report as embodying much interesting information. For other cases of the same kind, see a paper by Professor Gellé, translated in the *Veterinarian*, vol. xii, p. 486, from *La Zooiatre du Midi*. See also Professor Rey, *Veterinarian*, vol. ii, p. 413. It is almost superfluous to observe, that we only cite cases which bear evidence of correctness. We take no notice of many of the cases in which horses are said to have vomited, because these cases are indefinitely related.

[3] Journal de Physiologie, Experiment. et Path., par F. Magendie, tom. i., p. 338.

[4] Meryeologia sive de Ruminantibus et Ruminatione, p. 178.

[5] Loc. cit.

[6] Die Anatomie des Pferdes in 70 lithographirten Tafeln, von D. E. F. Gurlt, tabula 14, fig. 3, text p. 66.

[7] Compendium of the Vet. Art. By James White, edited by Mr. W. C. Spooner, 1842, p. 66.

after observing that the cardiac orifice "has a sort of valve formed by the duplication of its membrane," adds, that "the œsophagus, just previous to its entering the stomach, makes an acute angle, by which means, in great measure, vomiting in the horse is almost entirely forbidden." It has not, however, been satisfactorily shewn that either of the forms of valve above alluded to, exists in the horse's stomach.

3. M. Colin,[1] after repeating Lamorier's observation, that the relative position of the stomach and large intestines protects the former from the direct pressure of the abdominal walls, correctly remarks that, at its terminal orifice, the walls of the œsophagus are very thick, and that the cavity in its interior is closed, the mucous membrane being folded like a radiated flower. To this arrangement, M. Colin attributes the impossibility, or extreme difficulty, of vomiting in horses; and he endeavours to justify his belief by applying to the stomach the theory of the hydraulic press. But such a line of argument is not justly applicable in the case of the stomach and œsophagus, which, as living and active organs, are not regulated solely by hydraulic laws.

4. A fourth class of observers have attributed the difficulty of vomiting in the horse, to the oblique insertion of the œsophagus into the stomach, and to a sphincter, which they allege to be formed at the cardia by the union of the muscular bands belonging to the stomach and œsophagus. This doctrine appears to have been first promulgated by Bertin,[2] who stated in proof of the existence of the cardiac sphincter, that the weight of a man did not suffice to expel water or air from the cardiac orifice of various stomachs, the duodenum being tied.[3] Lafosse[4] only took into account the sphincter at the cardia, whereas, of late, Berard[5] and Rymer Jones[6] have reiterated the conclusion of Bertin; and the former writer, moreover, remarks[7] that the rhythmic movement which M. Magendie discovered in the lower third of the œsophagus, does not occur in the horse, whose œsophagus, he adds, is composed, in its last eight or ten inches, of a very elastic but inirritable tissue. Since, however, the microscope, no less than the naked eye, abundantly attests the true muscular nature of that tissue, M. Berard's denial of its irritability does not seem to be sufficiently warranted.

Bertin's theory has lately found a zealous advocate in M. Flourens,[8] whose opinion on this question merits more careful study than do those of his predecessors, because he has based it on experiments which he affirms to be *unicocal and demonstrative of the obstacle to vomiting in the horse.*

Having been interested by a perusal of M. Flourens' publication at an early period of my studentship, I at once repeated some of his

[1] Recueil de Médecine Vétérinaire pratique, III serie, to ne vi, p. 478.

[2] Histoire de l'Académie Royale des Sciences. Année 1746, p. 33-4.

[3] Had Bertin reflected that sphincters perform their function by virtue of the vital power of contraction with which their constituent muscular fibres are endowed, and that therefore they are inactive after death, he would have sought other explanations for the fact he observed.

[4] Dictionnaire Raisonné d'Hippiatrique, par M. Lafosse, vol. ii, p. 139.

[5] Cours de Physiologie fait par P. Berard, vol. ii, p. 139.

[6] Cyclopædia of Anatomy and Physiology: article *Solipeda*, p. 735.

[7] Op. cit., vol. ii, p. 28.

[8] Loc. cit.

experiments, and was surprised at the very different results which I obtained from them. A just appreciation of my position in relation to M. Flourens made me diffident, and caused me to repeat my observations and the study of his paper, at different times over a period of nearly three years. The result of this plan has been more clearly to establish the fallacy of M. Flourens' conclusions, and the mode by which he arrived at them. In justice to the originator, I prefix a translation of the account he gives of them to the results I have obtained, in order to afford full opportunity to every one to arrive at his own conclusions.

Experiment I. "The stomach being filled with water and the pylorus tied, the stomach was placed on a table, and on the stomach a board; two men then mounted on the board; they pressed it by jerks, and not a single drop of water escaped through the œsophagus.[1]

"It was evident from this first experiment, that all the obstacle to the escape of water by the œsophagus was in the superior orifice of the stomach. This was rendered more evident by a second experiment.

Experiment II. "The stomach being filled with water and the pylorus tied, I introduced a metallic tube, about an inch long, through the œsophagus into the superior orifice. No sooner was the tube placed in the opening than water flowed through the œsophagus.

"The obstacle of the cardiac orifice having been surmounted, none other presented itself in the œsophagus.

"Hence the œsophagus, notwithstanding its strong circular muscle, takes no part in the phenomenon, as is even more clearly demonstrated by the following experiment.

Experiment III. "The stomach being filled with water and the pylorus tied, I cut, piece by piece, from the pharynx to the stomach, all, absolutely all, the œsophagus; the stomach was pressed, and not a drop of water escaped from it.

"The œsophagus having been removed, I was able to introduce my finger into the cardiac orifice, and I recognised two things; firstly, that the more the stomach was compressed, the more closely was my finger grasped; and secondly, that the oblique direction of the superior orifice greatly aided the phenomenon, for as soon as I rendered this orifice straight, water flowed.[2]

"Bourgelat believed that the principal cause of the *not vomiting* might be in the accumulated folds of the mucous membrane of the cardiac orifice. The following experiment proves that Bourgelat erred.

Experiment IV. "I made a large incision on the side of the stomach; I subsequently removed all the mucous membrane of the

[1] M. Flourens states that he repeated this experiment more than twenty times, and the result was always the same, except with one stomach, which he states was diseased, and describes as "percé de trous profonds tous remplis de vers." There can be no doubt it was a stomach containing bots, a condition perfectly consistent with health, inasmuch as in a considerable portion of healthy horses, bots inhabit the stomach.

[2] It is remarkable that this observation did not shake M. F.'s faith in all his results; had he connected that observation with the known fact that, *as muscles*, the stomach and œsophagus could alter their relative position, he must have seen that their vital endowments were an insurmountable barrier to the success of his purely mechanical experiments and arguments.

cardiac orifice, and even that of the œsophagus. I sewed up the opening made with the knife; I filled the stomach with water; I tied the pylorus; I recommenced compression of the stomach, and not a drop of water escaped.

"The third experiment limits the extent of the obstacle to vomit on the part of the œsophagus; here is one which limits it on the part of the stomach.

Experiment V. "I made a large incision on the side of the stomach, and I cut the two *lateral fasciculi* of the internal muscles, without however touching the internal fibres, which really are the sphincter.

"The stomach was then sewn up and filled with water, the pylorus was tied, and pressure recommenced. Not a drop of water escaped by the œsophagus.

"All these experiments are univocal; they all demonstrate: firstly, that the obstacle to the horse's vomiting is in the superior orifice of the stomach; secondly, that it is in that orifice alone; and thirdly, that it is there due, firstly, to that orifice having a sphincter, and secondly, to the direction of that orifice being *oblique*.

"Bertin had therefore suspected rightly, that the sphincter of the superior orifice of the stomach, and the oblique direction of that orifice, are the two causes why the horse does not vomit."

Such is M. Flourens' statement. My own experiments on twenty-two horses' stomachs[1] have given the following results.

A. Twelve stomachs (with from four to six inches of œsophagus attached to each) having been filled with water through the pylorus, and this aperture closed by ligatures, allowed that liquid to escape through the cardia when little or no pressure was exerted on them. In several, the water at once gravitated through the cardia; in others, a very slight manual pressure sufficed to force it through that passage. In all cases I have provided for the freedom of the cardiac orifice while pressure was applied to the surfaces of the stomach; this I have done by placing the viscus near the edge of the table, and allowing its cardiac end to project a little. M. Flourens states that he caused the stomach to be placed *on a table*, without giving any reason to suppose that he took the necessary precaution with reference to the cardia. By citing one of the above twelve experiments, it will be seen how likely the experimenter in such a case is to succeed in discovering the object of his wish, if he be not cautious in avoiding fallacies.

A horse's stomach, with about five inches of œsophagus, filled with water through the pylorus, and this secured, when placed near the edge of the table with the cardia free, allowed water to flow through it by mere gravitation; slight compression exerted on the organ sufficed to expel the water to a considerable distance. When I placed my open hand on the surface of the stomach and pressed forwards, the flow gradually diminished and eventually ceased, even though considerable pressure was applied. By directing the pressure forwards I directed the cardia downwards, and mechanically closed it: how likely, nay almost inevitable, must be a similar result for a similar

[1] M. Flourens states that he repeated his first experiment more than twenty times.

reason, when two men stand on a board placed over the stomach and press with jerks.

B. A stomach, which did not allow water to flow through the cardia when a weight of fifty-two pounds was placed on it, and another, which required the pressure of twenty pounds before the water escaped, allowed the fluid to flow when I applied very slight pressure to the cul-de-sac with my hands.

The result of this observation suggests another source of fallacy in M. Flourens' experiments. The force generated by the contraction of the stomach's muscular coat is distributed all over its superfices, whereas M. Flourens only applied vertical pressure to one aspect. It is easy to understand how the result obtained by his doing so, might have been readily altered by simultaneously compressing the extremities of the organ.

C. After filling a stomach with water through the pylorus, this orifice was closed by ligature, and the viscus placed on a table. By grasping the stomach with my two hands, and exerting very considerable pressure in different directions, no water escaped. I then placed the stomach near the edge of the table, and on it a board with fifty-four pounds of iron; a little less weight than this sufficed to make the water dribble through the cardia; with this weight it flowed in a small but continuous stream. When I inclined the board backwards, so as to exert the greatest pressure on the great curvature of the stomach, the flow of water was freer than when I held the board horizontally; as I inclined it forwards the stream gradually diminished, and eventually stopped. Through an opening made at the great curvature I removed the mucous lining of the œsophagus, and that in the immediate neighbourhood of the cardia. On pouring water through the artificial opening, it escaped through the cardiac orifice in a large stream by mere gravity.

D. Four stomachs, in the same circumstances as the above, did not give exit to the contained fluid, though grasped and firmly pressed by the hands of two persons; but the water escaped freely from all, when the lining of the œsophagus and around the cardia was removed through an incised aperture made at the great curvature.

E. A stomach, treated and placed as usual, did not allow water to escape through the cardia, when a boy mounted on a board was placed over it; but the water flowed when a man, weighing one hundred and forty-four pounds, took the boy's place.

F. One stomach, in the same condition as the preceding, did not allow the escape of any water through the cardia when a man, weighing one hundred and fifty-seven pounds, mounted on a board which had been placed horizontally over it. When a boy, weighing sixty and a half pounds was added, water flowed in a continuous stream, and the viscus burst.

G. One stomach bore the weight of two men, without any water flowing through the cardiac orifice; on making an opening into the stomach, I found the mucous membrane at the cardia very much folded.

The experiments A, B, C, D, warrant us in denying the existence of a sphincter at the horse's cardia.[1] It would have been more satisfactory had the experiments E, F, G, been completed. Nevertheless the fact that the results most closely agreed with M. Flourens when, through inexperience, I conducted them with the least caution, may not be without weight in the establishment of the truth. When once it has been proved that, owing to its laxity, the thick mucous membrane forms folds at the dead horse's cardia, which more or less completely occlude it, it is easy to understand how—according to whether the stomach be contracted or dilated at the time of death—the degree of obstruction should vary. No application can fairly be made of the possibility of this obstruction in the dead horse's stomach to the physiology of that viscus during life; because the very fact of the mucus lining of a strong muscular tube being much folded, proves that its bore is susceptible of being much dilated, provided the stimulus to dilatation be communicated to the muscular structure.

[1] I performed careful experiments on three asses' stomachs, the results of which precisely agreed with these.

Though we believe that the citation of further evidence on this point is not essential to the cause of truth, it may not be quite useless to oppose to M. Flourens the opinion published in 1847 by one of his distinguished countrymen. In M. Mignon's report, already quoted, we find the following passage. "The reporter of your commission, when in charge of the anatomical works at the Alfort school, has many times proved, while passing a current of water through the first portions of the alimentary canal, that it was extremely easy to make the water introduced into the stomach through the duodenum escape through the œsophagus; it was sufficient for this purpose to fix a stop-cock firmly and hermetically in the duodenum. The water on entering the stomach, first filled it, and visibly distended it; then dilated the cardia into a kind of funnel; finally forced its way through that orifice, and flowed outwards."[1]

A somewhat less erroneous, because less exclusive, theory than any of the preceding has been advocated by Girard.[2] After having described the arrangement of the muscular fibres at the horse's cardia, he states, that he regards that as the principal cause of the inaptitude of monodactyles to vomit; as accessories to it, the peculiar mode of insertion of the œsophagus, the shape and position of the stomach.[3] We have already confuted the evidence which has been adduced by Bertin and others, to prove that the obstacle to vomiting resides in the muscular fibres of the cardia. As to the influence of the peculiar mode of insertion of the œsophagus, and the shape of the stomach, we need only remark, that since both these conditions are susceptible of alteration by the muscular activity of those organs, no arguments, based upon the assumption of their being invariable, can be accepted as conclusive. To the position of the stomach, (near the spine, and

[1] Loc. cit.

[2] Traité d'Anatomie Vétérinaire, par J. Girard. Quatrième edition, 1841. Tome deuxième, p. 569.

[3] Haller, (*Elem. Physiol.*, edit. Lausanne, tom. vi, p. 291), states, that the narrowness and oblique insertion of the œsophagus, and the debility of the horse's abdominal muscles and diaphragm, are the causes of its inability to vomit. Haller's error is greater than it appears; for his words, "et ob musculorum abdominis atque diaphragmatis debilitatem", refer to the *physical weakness* of those structures, as proved by his adducing in proof of his statement the authority of Lamorier, (*loc. cit.*), who, having observed that rupture of the diaphragm is frequent in the horse, refers it to the *weakness* of that muscle,—an explanation which is certainly not borne out by observation.

separated from the floor of the abdomen by the enormous large intestines), even I, at one time, attributed a great part of the horse's difficulty to vomit. It occurred to me, however, that the act of parturition in the mare is one of great rapidity; that abdominal respiration during disease or severe exercise is, in the horse, very easily effected; in the performance of these functions, and in the voidance of urine and fæces, the abdominal muscles take a very active part; and yet the uterus, bladder, and rectum are as disadvantageously placed, with reference to the floor of the abdomen, as is the stomach; the fact is, that the abdomen being completely full, pressure is transmitted very effectively from its muscular walls to the contained organs.[1]

Different as are the opinions we have hitherto commented upon, they yet present one remarkable point of analogy, inasmuch as all their authors attributed the horse's difficulty to vomit to a *mechanical obstacle*. It affords me pleasure gratefully to acknowledge, that for not falling into the same error I am indebted to Dr. Sharpey, who gave me an all-important hint by suggesting an inquiry into the action of emetics on the horse. It at once occurred to me, that as the mechanical part of the act of vomiting is excited by a reflex stimulus from the nervous centre, it behoved those who undertook to demonstrate why the horse rarely vomits, to study two classes of phenomena—*the nervous* and the *mechanical*:—for it is quite obvious that if the stimulus to the expulsive effort be wanting, it is useless to attribute the impossibility of the evacuation of the stomach by the œsophagus to mechanical obstacles, for they have no opportunity of coming into operation. Moreover, since I have excluded the existence of any mechanical impediments to vomiting in the horse, it is evident that the question which forms the subject of my inquiry, can alone be solved by determining what is the action of emetics on the nervous system of the horse.

It is since I received and worked upon Dr. Sharpey's hint, that I have carefully studied M. Mignon's report, and have found that it occurred to him and his collaborators, that the efforts of those who had studied the subject as pure mechanists could but prove abortive. "Is not the stomach of the horse that vomits," asks the reporter, "in conditions which no experiment would reproduce? And do you give no consideration to the nervous element, which you forget to regard as one of the data of the problem?" As a sketch of their theory of vomiting in the horse, MM. Mignon, etc., state "that the physical and vital conditions of vomiting, in their orders of succession and importance, are the following:—

"1. Extreme distension of the stomach.

"2. Disappearance of the folds in the œsophagean mucous membrane, accompanied by dilatation of the cardia into a kind of funnel.

"3. Paralysis of the muscular coat.

"4. Energetic concurrence of the nervous force, and of the action of the expiratory muscles of the abdominal walls."

As to the first of these propositions, on which the reporters lay the

[1] The above paragraph had been written several weeks before I read the following passage. "Il nous suffira de dire que dans l'abdomen où aucun vide n'existe, l'action des muscles du bas ventre doit se transmettre sans beaucoup de perte à tous les organes contenus dans la grande cavité splanchenique." (Mignon, loc. cit., p. 251.)

greatest stress,[1] it may suffice to say that it is an unfounded assumption.[2] We grant that frequently undue repletion of the stomach is the stimulus which, through the medium of the nervous centre, brings about the reflex movements, that end in evacuation of its contents by the œsophagus; but such repletion of the viscus is not essential to the perfect manifestation of the reflex acts in question. This is abundantly testified by the sufferers on a sea voyage, in whom the act of vomiting, frequently commencing when the stomach is empty, and continuing at other times after its evacuation, must primarily be due to a nervous cause alone.

The purport of the second proposition is best given in the terms of the report: "The disappearance of the folds in the mucous membrane at the cardia, and the dilatation of this part into a kind of funnel, are a necessary consequence of the eccentric and excessive distension of the stomach. The power that separates the sides of the viscus from each other is as great at the cardia as elsewhere; and if in the first moments of repletion of the gastric pouch the œsophagean sphincter resists, it no longer does so when the repletion is excessive. The walls of the stomach, by the mere fact of their distension, carry with them the two bands of the sphincter, the rectilinear direction of which they interrupt; and then these fleshy bands, far from enclosing the cardia, as between the branches of a compass, merely surround it as a section of a funnel. Thus it is that the œsophagean orifice is transformed into a wide, open, and round orifice.

The extreme dilatation of the stomach, by destroying at the cardia, and that in a purely physical manner, the obstacle which is opposed to the escape of matters through the œsophagus, must therefore be regarded as the principal condition of vomiting. This is accordingly confirmed by experiment. The experiment alluded to, is the one quoted at p. 555, from which M. Mignon discovered that if water were poured through the duodenum into the stomach, this first became distended, then the cardia opened and allowed the fluid to escape.

We confess that we were not a little surprised, when we first reflected that those who thus mechanically and positively argued from experiments on dead horses' stomachs, for the predetermination of vital phenomena, have in the same paper loudly inveighed against the

[1] "Nous ne prétendons pas avoir tout expliqué, tant s'en faut; nous n'avons voulu établir qu'une seule chose, à savoir: que la dilatation excessive de l'estomac est la condition par excellence du vomissement dans le cheval, en raison du changement tout physique qu'elle produit dans la forme du cardia et de la conséquence physiologique, c'est-à-dire de la paralysie qu'elle amène dans la tunique contractile de l'estomac."

[2] That the extreme distension alluded to in the report, is not of that sudden kind which Magendie, (loc. cit., p. 19), noticed in the stomach of dogs about to vomit, as being produced by the deglutition of air, but its gradual distension by food, may be inferred from the terms of the report. "On se demande comment dans un aussi court instant que l'espace de quelques heures, par exemple, l'estomac peut, sans se rupturer, se distendre jusqu'à tripler et au delà sa capacité normale. La masse intérieure qui la produit n'est point tout à coup volumineuse; c'est d'une manière insensible qu'elle se gonfle en quelque sort; l'action dilatante qu'elle exerce sur l'estomac, est donc lente et graduée. . . . . C'est ainsi que nous concevons que l'estomac soit parvenu à se dilater au point de contenir 35 livres de liquide ainsi que le rapporte M. Renault."

system of determining the functions of living parts, from the results of observations on dead ones.[1]

Admitting the possibility of extreme distension of the horse's stomach, we deny that it in any degree has a mechanical tendency to open the cardia during life. Let any one look at a horse's small stomach, placed beside the enormous quantity of food that animal is capable of devouring in a short time, and he will be forced to believe that, in perfect health, the organ is susceptible of great dilatation, without either involving opening of the cardia, or impairment of its vital power of contraction. To argue that because one part of a hollow viscus is dilated, the other must be so likewise, is in opposition to known facts. Moreover, my own experiments on dead horses' stomachs, have shown me that not always is extreme distension of the viscus with water followed by escape of the fluid through the cardia; it is or is not, according to the presence or absence of mechanical obstructions. The opening of the cardia, no less than its closure, is, in the living animal, doubtlessly owing to vital contraction of muscular fibres.

The reporters found their third proposition on the general truth that extreme dilatation of a hollow muscle is attended with impairment of its contractile property; thus they make the third a necessary sequence of the first proposition. Since we have impugned the latter, the former, in our opinion, falls to the ground. In asserting that before a horse can vomit, the muscular coat of the stomach must be paralysed, Mignon, in main part, re-echoed the theory of Renault, who several years previously had announced, that not only was the muscular action of the stomach useless, but opposed to vomiting, which only occurred when that viscus became excessively distended.[2] My own experiments, conducted for the purpose of ascertaining the effect of injecting a solution of tartar emetic into the veins of the horse, force me to look on those of M. Renault with great doubt; for I have yet to learn that an experimenter has the power of exciting at will efforts to vomit in the horse; and what I have seen leads me strongly to suspect, that when after injecting tartar emetic into the veins of a horse, and cutting open the abdomen, observers have indulged in the belief that they were witnessing efforts to vomit, they have only witnessed the violent efforts made by tortured brutes in self-defence.

The theory advocated by Renault and Mignon, is, as the latter admits, founded on the doctrine first promulgated by Bayle and Chirac, and of late rendered notorious by the singular experiments with which M. Magendie defended it. The French hippiatrists, in fact, having exclusively studied the mechanical part of the act of vomiting, were forced to decide between the opposite doctrines, defended by Haller and Magendie, as to the participation of the stomach in the performance of that act. The result has been, the declaration of their adhesion

[1] On a selon nous grand tort de conclure du resultat d'expériences d'amphithéâtres trop souvent imparfaites et sur des organes morts, à ce qui doit se passer au milieu des parties que la vie anime. Ne serait-ce pas une pretention trop étrange, que vouloir surprendre, à l'aide de moyens si grossiers, si limités, si incomplets, si matériels, le secret de ces phénomènes si insaisissables, si changeants et surtout si merveilleux de simplicité, qui se produisent au sein de toute organisation vivante! (Mignon's Report, loc. cit., p. 197.)

[2] Bulletin Soc. Centr. de Méd. Vétér., tome ii, p. 218, 219.

to their illustrious countryman; a result which would in all probability have been different, had they rigidly tested the validity of the claims of his doctrine,[1] and have taken due notice of the positive observations of Wepfer, Haller, and Rudolphi, and of the recorded cases in which vomiting occurred when extrinsic pressure was removed from the stomach, either by palsy or destruction of the abdominal muscles.

That the energetic concurrence of the nervous power, and of the action of the expiratory muscles of the abdominal wall, takes part in the act of vomiting, as stated in M. Mignon's fourth proposition, all will grant; but that it is fourth in the order of succession we deny, for it is well known that purely nervous phenomena are the first signs of a disposition to vomit; and that it is fourth in the order of importance we deny, on the ground of the experimental truth, that, whereas the condition of the stomach is unimportant, a participation of the nervous system is indispensable, before the movements necessary to vomiting can be effected. So unimportant, indeed, is the condition of the stomach, that vomiting may occur without it, as proved by M. Magendie's experiment of substituting a pig's bladder for a dog's stomach; and it is not a little surprising that the reporter, who seems to have laid so much stress on it, should not have discovered that his idea of "excessive dilatation of the stomach being the condition, *par excellence*, of vomiting in the horse", was opposed to the result of an experiment which he invoked in defence of his views.

Now that we have subverted three out of the four propositions on which Mignon's theory is founded, we should be at a loss to guess what might be his idea as to the reasons why the horse rarely vomits, if, in a discussion which ensued on his report,[2] he had not unequivocally avowed assent to Girard's *mechanical* doctrine, the fallacy of which we have already demonstrated.

Thus we have completed a critical and historical account of the opinions emitted on the subject; and, having proved that all observers have attributed the rarity of vomiting in the horse to mechanical causes *which do not exist*, it remains for us to determine whether there are any impediments to it in the nervous system of the horse. With this object in view, let us inquire into the action of emetics in the horse.

III. WHAT IS THE ACTION OF EMETICS ON THE HORSE? It is so well known among veterinary pathologists, that the horse is not acted on by emetics as is the dog, that, while they frequently prescribe them in the diseases of the latter, they never do so in those of the former. The most celebrated writers on veterinary therapeutics, generally exclude emetics from the list of medicines available for the relief of the diseases of the horse, and refuse to the emetic, *par excel-*

[1] Sound critics have long since decided that Magendie's conclusions on vomiting are not warranted by his experiments, which, at utmost, prove that vomiting may occur by the extrinsic pressure of the diaphragm and abdominal muscles, without the active co-operation of the stomach. They certainly do not disprove, that under ordinary circumstances, the stomach aids in the ejection of its contents by the œsophagus.

[2] Les vraies raisons, les seules bonnes et importantes raisons de l'impossibilité du vomissement, indiquées et décrites d'abord par Bertin, ont été parfaitement développés par M. Girard, dans son mémoire de 1810, et surtout dans sa notice de 1841.

lence,—the potassio-tartrate of antimony,—any emetic virtue in the equine species, when administered internally.[1] Were this the sum total of our information on the point at issue, our inquiry would be at an end; but since it has been alleged by several French experimentalists, that the horse makes efforts to vomit when potassio-tartrate of antimony is injected into its veins, it is imperative that we should test the empirical grounds of such allegation.

We find it stated by M. Dupuy that he has succeeded in producing attempts to vomit in the horse, by injecting tartar emetic into the crural or jugular veins, in doses varying from six grains[2] to two drachms.[3] The director and professor of the Veterinary School at Lyons, in their account[4] of the action of tartarized antimony, thus express themselves: "Injected into the veins of the large herbivora, in doses of from nine to eighteen grains, tartar emetic occasions vomiting, or the phenomena which accompany that act of evacuation." They give no evidence to prove this statement.

Finally, we have to quote an experiment which was performed by MM. Leblanc and Mignon:[5] "Thirty-six grains of tartar emetic injected into one of the jugular veins of a horse,—a longitudinal opening about seven inches long, made on the side of the linea alba,—exploration of the stomach with the finger,—contractions of the viscus insensible,—contraction of the abdominal muscles alternating with that of the diaphragm, the latter taking place during inspiration; at this moment the right crus of the diaphragm is forcibly extended, but the œsophagus is only moderately compressed between its fleshy lips,—the intestines forcibly escape through the wound in the abdomen,—

[1] "In the horse vomition cannot be excited." Introductory Lecture on Chemistry, by Mr. Morton, *Veterinarian*, vol. xiv, p. 767. Le cheval et nos herbivores ruminants sont des animaux chez lesquels nos émétiques sont sans puissance. Bouchardat, *Formulaire Vétérinaire*, p. 258. Of the action of potassio-tartrate of antimony in the horse, Mr. Percival says, "I give it in doses of a drachm and two drachms...Of little palpable efficacy given internally." Percival, *On Form and Action in the Horse and the Effects of Medicine in Horses*, p. 92. Nimrod (*Veterin.* vol. x, p. 415) states that the potassio-tartrate of antimony has always been considered among sportsmen as a cleanser of a foul habit in horses, in doses of from one to three drachms. In a discussion which took place at the Royal Academy of Medicine of Paris on the subject of vomiting, M. Renault thus expressed himself (*Bulletin Soc. Centr. Vétér.* tom. ii, p. 219). "I made a crucial incision into the abdomen of horses to which I had previously *given* tartar emetic. On introducing the hand into the wound, I felt the stomach forcibly contracting, and its contractions became visible on its being made to protrude through the abdominal walls. These contractions did not cease until the stomach was excessively dilated, and then only did vomiting occur. I concluded from these experiments that the muscular coat is an obstacle to vomiting." After the undeniable proof we possess of the potassio-tartrate of antimony not acting as an emetic when administered to the horse, the result of M. Renault's experiments affords much surprise, which is raised into incredulity on reflecting that the stomach could not be pressed upon by the abdominal walls, because crucially divided. That they were not reunited by suture, is evident from his statement that he continued to observe the state of the stomach. It would appear fair to argue, that if a stomach were itself inactive, and not compressed by extrinsic force, its contents could not be evacuated.

[2] Veterinarian, vol. iii, p. 529. [3] Ibid. viii, p. 575.

[4] Dictionnaire Général de Médecine et de Chirurgie Vétérinaires, et des sciences qui s'y rattachent, par MM. Lecoy, Rey, Tisserant, Tabourin.

[5] Bulletin cit. p. 242.

eight minutes after this injection of tartar emetic, appearance of some nausea, or violent and simultaneous contraction of lower abdominal muscles and diaphragm. The whole intestine is removed in order to see the stomach well; the viscus follows, like a pendulum, the alternate movements of contraction and relaxation of the diaphragm; the stomach, which is tolerably distended, is the seat of slow, continuous contractions, which always commence at the same point, the pylorus. The contraction and relaxation of the lower abdominal muscles and diaphragm take place gradually and slowly. This partition yields little by little, but uninterruptedly, to the abdominal muscles; these act similarly with regard to the diaphragm, so that if we form a material idea of these respiratory powers, we can compare them to two machines always equidistant, and directed in the same course, each describing the half of a double cone, whose limited base of reciprocal excursion would be in the middle. When one of them is at the summit, the other is at the base, and *vice versâ;* consequently, the action of the diaphragm increases in proportion as that of the abdominal muscles diminishes. These forces are therefore essentially respiratory. The one—the diaphragm—is active during inspiration; the other—the force generated by the abdominal muscles—is expiratory. Renewed, sudden, violent, and simultaneous contractions of the diaphragm and abdominal muscles occur; the latter even appear to augment their action in proportion as the diaphragm relaxes; it is the nausea, or the manifestation of the effort. No vomiting has occurred; finally, the animal expires."

From this account it appears that, on seeing violent efforts of the abdominal muscles and diaphragm of a horse, into whose veins tartar emetic had been injected, and from whose abdomen all the intestines had been removed through a large opening, MM. Leblanc and Mignon inferred that the emetic was producing its specific action by exciting efforts to evacuate the stomach through the œsophagus. No vomiting occurred, and therefore the experiment, *primâ facie*, establishes,—firstly, that the horse makes efforts to vomit when potassio-tartrate of antimony is injected into the veins; secondly, that as those efforts are not followed by vomiting, there must be some mechanical impediment to the escape of matters through the cardia. Both these conclusions are erroneous; and they are so for the following reasons. The description given of the movements of the abdominal muscles and diaphragm, is not such as to prove beyond doubt that they were efforts to vomit. Confessing adhesion to the doctrine that the diaphragm takes an active part in vomiting, we deny that it does so by virtue of an *inspiratory movement*, and that, as it *relaxes*, the abdominal muscles make a sudden effort, as in forcible expiration. The act of vomiting is one *sui generis*, and not a respiratory act.[1] Let any one try to simu-

[1] Since the terms in which M. Mignon has related his experiment may leave some doubt as to his idea of the nature of the movements in vomiting, it may be cleared up by his ulterior exposition of that idea. "Ce phenomène (le vomissement) commence aux derniers termes de *l'inspiration* pour finir dans *l'expiration*. C'est dans ce dernier temps ou pendant le relâchement du diaphragme qu'a lieu le rejet des matières sans aucun doute; mais c'est dans l'inspiration, ou par le fait d'une violente et brusque contraction du diaphragm, aidé de l'action des muscles abdominaux, que l'estomac verse son trop-plein dans l'œsophage."—*Op. cit.*, p. 244.

late the effort to vomit, or study the real effort in his own person, and he will feel that the glottis having been closed after a deep inspiration, the diaphragm and abdominal muscles act simultaneously, so as to press the stomach between them. To this it may be objected that, for the diaphragm to descend while the glottis is closed, the air in the chest should be rarefied, which is an unlikely occurrence. The fallacy of this objection is demonstrable by experiment, as Dr. Sharpey has long since shown in his lectures on anatomy and physiology in University College. A bandage being closely passed round the lower part of the chest, it is easy to press down the diaphragm at will; on doing so, the bandage becomes loose, evidently owing to drawing in of the cartilages of the lower ribs by the descending diaphragm. Thus the augmentation of the thoracic cavity in its long axis, is compensated by its decrease in the transverse; and it becomes obvious that the diaphragm may descend when the glottis is closed, without enlarging the chest, and therefore without rarifying the contained air. That, however, the witnessed efforts were in reality the pangs of death, is rendered more than probable by the terms in which the narrative of the experiment ends,—"*enfin, l'animal expire.*"[1]

But conceding, for the sake of argument, that the movements observed were efforts to vomit, we maintain that it is not just to conclude that, because they were not followed by vomiting, there must have been some mechanical obstacle to the escape of matters through the cardia. As the experiment was performed, it was impossible for the stomach to be pressed upon by either the abdominal muscles or diaphragm, and therefore there was no chance of its contents being evacuated through the œsophagus. Protected, as the horse's stomach is, by the far-back projecting ribs, it can only receive pressure from the abdominal muscles, indirectly, through the intestines. When these are removed, the viscus cannot be pressed upon by those muscles, and the pressure of the diaphragm on it is very slight, because the stomach undulates like a pendulum, backwards and forwards, in the empty cavity, without meeting with the least resistance.

The evidence which we have adduced, and which is all that we have been able to collect, in favour of the doctrine that efforts to vomit can be excited in the horse by injecting tartar emetic into its veins, may be thus briefly summed up:—

1. M. Lecoq and his colleagues (the veterinary professors of Lyons) have expressed an opinion in the affirmative, without publishing the grounds of their belief.

2. The experiment of MM. Leblanc and Mignon, which professes to prove the doctrine, appears to have been conducted with so little regard to the exclusion of sources of error, that it would be unwarrantable to draw any positive inferences from it.

3. As we are not acquainted with the details of the experiments which formed the basis of M. Dupuy's statement in support of the above doctrine, they need confirmation; a need which becomes imperative, on reflection that the horse is not susceptible of emesis by the introduction of any medicinal substance into the stomach, and that as our knowledge of the action of emetics in the dog proves, that when introduced into the veins or into the stomach their effects differ in degree and not in kind, there is strong ground for the belief that the horse is unsusceptible of the specific action of emetics, even when directly injected into its circulating system.

[1] As the performance of such experiments in horses renders it indispensable that they should be thrown on the ground and secured, great caution should be observed in interpreting the muscular efforts; for when a horse is cast he struggles violently for liberty, even before a bold stroke of the knife has made a gash seven inches long into its belly, and all the bowels have been removed.

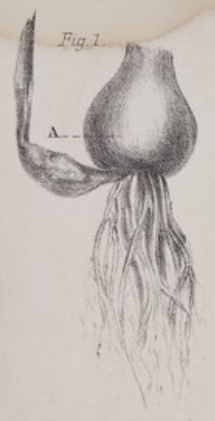
In order to settle the question, I determined to appeal to experiment, and procured for the purpose a horse and a mule, both of sound constitution. I have injected into their jugular veins solutions of tartar emetic, in 5, 30, and 50 grain doses, but have never seen efforts to vomit; to avoid misunderstanding I may say, that I have never seen any such thing as the animals thus experimented on take a deep inspiration, fix the chest, and make sudden and forcible exertion with the abdominal muscles. Inasmuch, however, as I noticed some preternatural phenomena of muscular action in two of the experiments, a detailed account of them is rendered necessary. Twenty minutes after injecting into the left jugular vein of a horse thirty grains of tartar emetic, dissolved in three ounces of water, the muscles generally became rigid; but there was no movement caused by any of them. It was static, not dynamic contraction. After this, I noticed a good deal of twitching of the muscles of the fore and hind limbs, and of those of the lower part of the neck; but the abdominal muscles acted evenly, though with more frequency than usual, as the respiration became a good deal accelerated. At the end of three hours the twitching had almost ceased, and had done so completely; and the animal had resumed eating in a little less than four hours.[1] Lest it should be feared that I misconstrued the phenomena of muscular contraction, I may say that almost every five minutes, and sometimes oftener, I took notes of the state of the animal; and did not note such words as *spasm* or *effort*, but avoided the danger of erring in judgment by describing in periphrases all I saw. It is important to notice that in the same animal, on a subsequent day, I injected 30 grains of tartar emetic in $1\frac{1}{2}$ oz. of water, without producing any sensible effect whatever. I injected 50 grains of tartar emetic, dissolved in $1\frac{1}{2}$ oz. of tepid water, into the jugular of a mule, with the following result. Fourteen minutes after the injection, the animal did not present any change; but, in a few minutes more, left off eating and stood still. In the ensuing thirty-seven minutes, the muscular system was several times subject to momentary rigidity, which, with the exception of slight elevation and extension of the head, was unaccompanied by any movement. This muscular rigidity simulated the tetanic state, and contrasted strongly with the state of the muscles of the same animal while voiding fæces and urine; in an hour and fourteen minutes the mule re-

[1] A few minutes after I had injected the tartar emetic, the animal commenced purging, and continued doing so for an hour and a half. To determine whether this phenomenon was due to the tartar emetic or to the water, I injected three ounces of tepid water into the jugular vein of a mule—it had no effect; but purging was occasioned in fourteen minutes by injecting into the same vein, after the lapse of an hour, six ounces of tepid water. In none of my subsequent experiments was this purging produced,—a fact which needs further experience for its explanation.

sumed eating, and presented no other symptoms. If I had been a little less cautious in the avoidance of fallacy, it is very likely I should, in observing these muscular phenomena in two of the experiments, have succeeded in seeing attempts to vomit; but they assuredly were not such, for they did not in any degree resemble the efforts made by the same animals to evacuate the bladder and rectum, or the efforts made by the dog and man to empty the stomach through the œsophagus.

Considering the vague manner in which M. Dupuy alludes to his experiments, and, on the other hand, confident of the fairness with which my own have been conducted and recounted, I feel myself justified in opposing my conclusions to his, and in stating that all the attempts hitherto made to excite efforts to vomit in the horse by emetics have failed. This unsusceptibility to emetic action, and the very rare manifestation of the phenomena of vomiting by the horse, must obviously be regarded as cause and effect, and, consequently, as the answer to the question, Why does the horse rarely vomit?

7th February, 1852.


Fig. 1.
A.
June

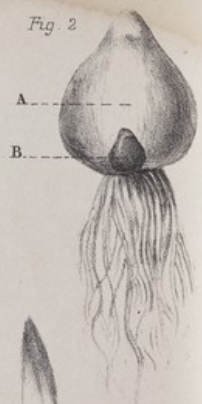
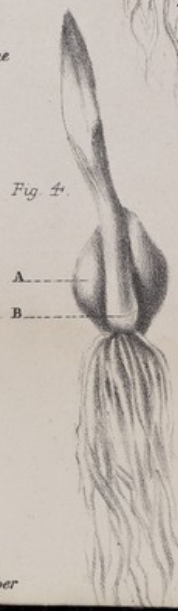
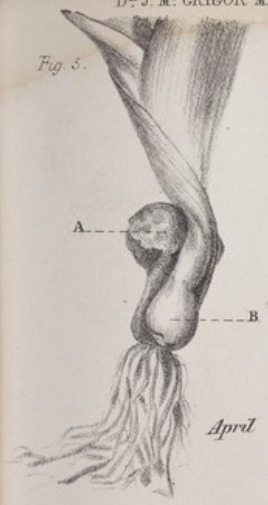
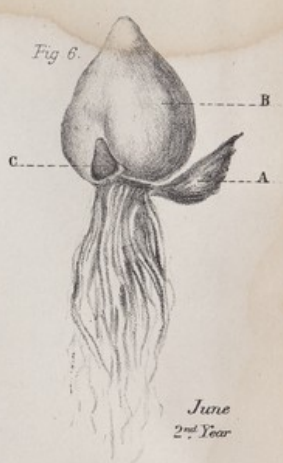
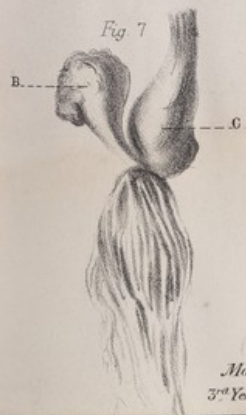

Fig. 2
A.
B.
July

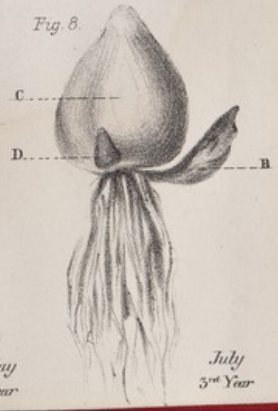

Fig. 3
A.
B.
September


Fig. 4.
A.
B.
February


Fig. 5.
A.
B.
April


Fig. 6.
B.
C.
A.
June
2nd. Year


Fig. 7
B.
C.
May
3rd Year


Fig. 8.
C.
D.
B.
July
3rd. Year

With the Authors Compl.ts

ON THE

NATURAL HISTORY, PHYSIOLOGICAL ACTIONS,

AND

THERAPEUTIC USES,

OF

COLCHICUM AUTUMNALE,

CHIEFLY WITH

REFERENCE TO THE GROWTH OF THE PLANT, AND
THE CHEMICAL CHANGES IT PRODUCES
ON THE BLOOD AND URINE.

BY

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EDINBURGH:
SUTHERLAND AND KNOX, GEORGE STREET.

MDCCCLII.

[FROM THE MONTHLY JOURNAL OF MEDICAL SCIENCE, FOR 1851-52.]

MURRAY AND GIBB, PRINTERS, EDINBURGH.

ON COLCHICUM AUTUMNALE.[1]

It appears probable that this drug is identical with that known to Dioscorides,[2] Paulus Ægineta,[3] Alexander Trallianus,[4] and other ancient authors, under the names of "Κολχικον" and "Ἑρμοδακτυλος;" but it is equally probable that its use in medicine did not become established till so late a period as 1763, when it was introduced, with many other remedies, by Baron Stoërk,[5] of Vienna. Subsequently it has been examined with much attention by Sir Everard Home,[6] Sir Charles Scudamore,[7] Dr Haden,[8] and Mr Want.[9]

In the following inquiry, I propose to direct attention,—first, to the botanical and chemical histories of the drug, chiefly with reference to the growth and preservation of the bulb, and to the nature of the active principle obtained from it; and secondly, to its physiological and therapeutic actions.

BOTANICAL HISTORY.

Colchicum belongs to the endogenous natural order *Melanthaceæ* of Lindley, and *Colchicaceæ* of De Candolle, of which order the following are the principal distinguishing characters:—

They are in general bulbous, or fibrous-rooted plants. In some the flowers are half-subterranean, like the *Crocus*; in others they

[1] Being an abstract of an Inaugural Dissertation, delivered to the Medical Faculty of the University of Edinburgh, August 1851.
[2] Dioscorides—Materia Medica, lib. iv., cc. 84, 85.
[3] Paulus—Materia Medica, lib. vii., c. 3.
[4] Alexander—Medici Libri, lib. xi.
[5] Stoërk de Colchico, 1763.
[6] Sir E. Home—Philosoph. Transac., 1816-17, part 2.
[7] Sir C. Scudamore on Gout and Rheumatism, 1819.
[8] Haden on Colchicum, 1820.
[9] Want—Lond. Med. and Phys. Jour., vol. xxxii.

form spikes, or simple branching herbaceous stems, as in the genus *Veratrum*. The Calyx and Corolla form together a perianth, generally consisting of six pieces, which are usually petaloid. The ovary is three-celled, with many seeds.

They belong to Linnæus' class and order *Hexandria Trigynia*. The *Melanthaceæ* approach in character most nearly to the *Liliaceæ*, but are distinguished from that order by their tripartite fruit, and anthers turned outwards.

These plants are common at the Cape of Good Hope, Asia, and North America.

"This order seems to be confined within no geographical limits; it is, however, far more abundant in northern countries than elsewhere."[1]

In their properties, these plants are almost always poisonous in all their parts, and act generally as *narcotico-acrids*.

The principal genera of this order which furnish medicinal plants, and whose active properties have been most fully investigated, are COLCHICUM, VERATRUM, and ASAGRAEA, to the former of which, however, our attention is at present limited.

There are three well-known and distinctly-defined species of COLCHICUM,—namely, *Colchicum autumnale*, *C. montanum*, and *C. variegatum*.

Colchicum Illyricum has been supposed by some to be a distinct species, but more generally it is considered to be identical with *C. variegatum*. To this species the drug known as HERMODACTYL has been referred.

Kunth[2] believes that the *Colchicum montanum* of Allioni and the *C. autumnale* χ of Bertolini are identical. He thus describes this species:—The tube of the perigone is five or six times longer than the limb; the calycine segments lanceolate; stamens equally inserted; bulb unifloral; leaves succeeding the flowers, linear lanceolate, obtuse, and attenuated towards their base. *C. variegatum* he regards as a distinct species, and thus describes it:—Leaves oblongo-lanceolate, channelled, and undulated at their margin; the lanceolate segments of the corolla acute.

COLCHICUM AUTUMNALE is the only species officinal in this country. In vernacular language it is termed *Autumn Crocus*, or *Meadow Saffron*. It is indigenous to Britain, occurring frequently in Suffolk, Oxfordshire, and Wiltshire, in England; but in Scotland, although found in many situations, it must be regarded rather as an introduced than a native plant, the only Scotch station assigned to it being at Alloa.[3]

The leaves of COLCHICUM are broad, lanceolate, and erect.

[1] Lindley's Vegetable Kingdom.
[2] Kunth—Enumeratio Plantarum, tome iv., 1843.
[3] Lightfoot—Flora Scotica.

The flowers arise from the bulb by a long tube, which is surrounded at the base by a membranous sheath.

The stamens are inserted in the oblongo-ovate segments of the pale purple perianth.

The ovary is at the base of the bulb, and its long filiform style runs up the whole length of the tube.

In its mode of inflorescence, COLCHICUM AUTUMNALE somewhat resembles *Tussilago Farfara*, or common *Colt's-foot*, inasmuch as the flowers and leaves occur at different seasons,—the difference being, that in the *Colt's-foot* the leaves immediately succeed the flowers, whilst in the COLCHICUM several months elapse between the flowering and leaf-bearing seasons.

The manner of the propagation of COLCHICUM AUTUMNALE is interesting, not merely in a botanical point of view, but also as regards the medicinal qualities of the corm or bulb at different periods of its growth.

It is well known to be capable of being propagated in two different ways,—namely, by seeds and bulbs. Dr Christison,[1] however, has mentioned a third,—viz., the throwing off of a number of small immature bulbs from the parent plant. The manner in which perfect plants grow from seeds and immature bulbs has not been determined. It has not, indeed, been accurately ascertained if these immature bulbs ever produced perfect plants at all; but it seems probable that their immature separation from the parent renders them incapable of coming to perfection, and they are perhaps to be regarded more as a kind of abortion, than as the fruits of a legitimate mode of propagation.

I have endeavoured to trace the growth of the plant from seeds, but in vain. The COLCHICUM in Scotland rarely produces fruit, and never ripens its seeds. I have never succeeded in attempts to grow any of the seeds procured from the druggists' shops.

The propagation of the plant, by its more ordinary method of forming a single new bulb, has been better ascertained; and, in order to enable the growth of a single bulb to be traced from its origin to its termination, I shall in my description refer to the accompanying diagram,[2] where the individual bulbs are lettered, and can be easily followed through all the stages of their progress.

We shall suppose the time at which the observation is commenced to be *June*, and that we have in view a nearly full-sized bulb, which we shall call A. At this time it is as large as an apricot, firm, amylaceous, and extremely bitter, and having attached to it the shrivelled remains of the old bulb, and the leaves, now yellow and decayed.

At the end of *June*, or commencement of *July*, a small bulb will be observed to have become developed upon the side of the corm

[1] Dispensatory.
[2] The figures in the diagram are reduced one-third.

A, at its lower part. This small bulb we shall call B. At this time it is a little larger than a grain of wheat, and lies in a small fissure on the side of the parent bulb, a little above the origin of the radicles. It increases slowly and gradually in size till the beginning of *August*, when it appears as a mere dilatation of the flower-stalk, which it then commences to put up.

In *September* the flower is in full perfection, the long tube of the perianth of which, has raised the six-partite limb to the height of from six to eight inches above the ground. The flower remains for two or three weeks, and then dies down; and nothing of the plant is seen above the surface till the beginning of *February*, when the leaf-stalk commences to rise.

If at this time the plant be taken up, A and B will still be found to be united, but B will be observed to have increased little in size since autumn, being but still hardly larger in diameter than the leaf-stalk.

The bulb B thus grows little during the autumn, but in winter it increases rapidly in size; in *April* it is like a large hazel-nut, and from that time it increases still more, till the end of *June* or beginning of *July*, when it is, as Dr Christison states, as large as an apricot.

In *April* the leaf-stalk is found perfected by a fine group of dark green leaves, generally three in number, and having within their sheath the capsules which ought to ripen their fruit in the course of summer.

In *May* the old bulb will be found dry and withered, and with very little starch; and in *July*, if the plant be taken up, three bulbs will be found,—A, now reduced to the form of a membrane, bearing no resemblance to a bulb at all; B, now arrived at full growth; and a new member of the series, C, the progeny of B.

This third bulb, C, it is unnecessary for us to trace further; it follows a course precisely similar to that through which B has passed, and which has just been described.

The history is now complete, so far as regards the rotation of flowers, leaves, and fruits; but the lifetime of B has not yet expired; for if we take up the plant in *May* of the third year, we still find the shrivelled remains of B, and C large, firm, and amylaceous, now bearing leaves, having flowered during the previous autumn. We may go on still further, and if in *July* the plant be examined, B will be found to have almost disappeared, C large, amylaceous, and extremely bitter, and at its base a new bulb D, of very minute size, which in the ensuing autumn will produce the flower.

Thus we have traced the growth of COLCHICUM from its infancy, through maturity, to decay.

It must be evident, from the foregoing observations, that the plant is essentially biennial, but it has been thought by some to be triennial. "It sees a part of three successive years, but only outlives two revolutions of each season." (Christison.)

Of peculiarities in the growth of COLCHICUM, I may mention one, which has struck me forcibly on account of its frequent occurrence.

In *February* and *August*, instead of one leaf-stalk and one flower-stalk making their appearance at their respective periods, I have often remarked that two have occurred, one on either side of the parent bulb. I believe this to be one of the effects of cultivation, as I have nowhere seen it remarked in descriptions of the plant by botanical authors.

Having observed, in cases where the leaf-stalk was accidentally removed from the parent bulb, that a new bulb was thrown out from the top part of the old one, I made the following experiment, in order to ascertain, if possible, the cause of this peculiarity.

Two bulbs were taken up on 1st *November;* from these the leaf-stalks and their bulbs were detached, and the parent bulbs replanted. Three weeks afterwards a small leaf-stalk was observed to have been given off from the top part of each bulb; and on another occasion, on which the same experiment was tried, two of these leaf-stalks made their appearance. In both of these instances they were found to proceed from very minute bulbs, not larger than barley-corns.

I believe that there is always one of these adventitious bulbs at the top of each parent bulb, but that they never germinate, where the plants are indigenous, unless the proper leaf-stalk and its bulb be removed. When they do so, however, they always remain attached to the parent bulb, and are perfected in much the same time as the normal bulb of the leaf-stalk.

Dr Christison has mentioned that the full size of a COLCHICUM bulb is that of a small apricot. This I believe to be perfectly correct, in places where the plants are indigenous; but I have frequently procured specimens from the Botanic Garden here, where they were cultivated for the purpose of examination, of the size of large apples; and in *October* 1849, I procured one which weighed nine and a half ounces.

I have lately received from the North-Western Provinces of India two specimens of COLCHICUM bulbs, which respectively bear the names of "SORINJAN TULK," or bitter *Sorinjan*, and "SORINJAN SHEERUN," or sweet *Sorinjan*. Both of these were brought from Bombay, and I believe are identical with those mentioned by Dr Royle,[1] and thus described by Dr Pereira:—"The SORINJAN SHEERUN resembles the cormus of colchicum autumnale. The cormi are flattened, cordate, hollowed or grooved on one side, convex on the other; they have been deprived of their coats, are externally dirty-yellow or brownish, internally white, easily broken, farinaceous, odourless, tasteless, or nearly so, and worm-eaten."[2]

[1] Royle's Materia Medica.

[2] Pereira's Materia Medica.

This description entirely coincides with the specimens in my possession. They are so easily broken, that when I received them nearly one-half was reduced to powder.

Geoffroy[1] has correctly pointed out how they may be distinguished from the cormi of COLCHICUM AUTUMNALE. "They are not rugose, are white internally, are moderately hard, easily broken, and form a whitish powder; whereas the dried cormi of COLCHICUM AUTUMNALE are rugose, softer, and have a reddish or grayish tint, both internally and externally."

The SORINJAN TULK appears to me to resemble the bulb of COLCHICUM *variegatum*; they are much smaller than the preceding, and possess considerable bitterness; they are not so easily broken. Pereira states that they are marked by longitudinal stripes, indicating a laminated structure. I have failed to discover this in the specimens in my possession; and I presume if the true HERMODACTYL possesses a laminated structure, it cannot be considered as a true cormus, and therefore cannot belong to the colchicum tribe. The Hakeims use both kinds in *rheumatism*, and in many nervous disorders, in doses of two to four grains of the powder three times daily.

I also received from the same source a few seeds, which were said to be those of COLCHICUM *Illyricum*. They were of the size of, and altogether not unlike, *Lupin* seeds. I attempted to grow them, but in vain. They are used in the same diseases as the SORINJAN, and are said to be diuretic.

PHARMACOLOGY.

Every part of the plant, excepting the leaves, has been used in medicine. The flowers have been used galenically, and have been supposed to be that part of the plant used in the preparation of the *Eau médicinale d'Husson*. In most modern pharmacopœias, as in those of the British Empire and of France, the seeds and corms alone are officinal.

As found in commerce, the seeds of COLCHICUM are rough, small, and almost spherical, imparting to the mouth a bitterness and acrimony which surpasses that of the bulb. They are about the size of millet seeds, and have a dark brown colour.

The slices of the bulb are grayish-white, somewhat kidney-shaped, and have a dark-brown covering externally. When in good preservation, they are dry and easily fractured. They should not be thicker than a half-crown. Their taste is extremely bitter, and somewhat acrid.

There has been much difference of opinion as to the time of year in which the cormus should be taken up for medicinal use. According to my own observations, the middle of JULY is the most

[1] Geoffroy—Traité de Matière Médicale.

fit time for this purpose. The bulb is then of its greatest size, it is firm, amylaceous, and extremely bitter. The *bitterness* is the character which affords the best criterion of its activity.

According to some, the amount of *Starch* and firmness in the bulb are the proper guides; but the amount of active principle does not necessarily bear any corresponding ratio to that of the starch, in proof of which I may observe, that the bulb, when more spongy and watery, and less amylaceous, as in *April*, possesses almost as much bitterness as when nearly mature,—a circumstance remarked by Dr Christison, and which I have confirmed by repeated observation.

I feel confident that we can place no reliance in a test of goodness proposed by Dr A. T. Thomson.

In 1820 Dr Thomson[1] published a paper showing the tincture of *Guaiacum* to be a test for *Gluten*, and pointing out its applicability as a test for the goodness of COLCHICUM. His manner of procedure was as follows:—Ten grains of the bulb were rubbed in a mortar, with sixteen minims of distilled vinegar, and immediately afterwards sixteen minims of the *tincture of guaiacum* were added. A beautiful cœrulean-blue colour was produced with those specimens which, according to Dr Thomson, were good.

I have several times made, with specimens which I knew to be good from their bitterness, similar experiments, and obtained no cœrulean-blue colour, but only a dirty brown.

In the same year (1820) Mr Battley[2] published similar experiments which he had made, but with entirely different success from Dr Thomson.

In order to ascertain what the true nature of this re-action might be, I expressed the juice from several bulbs, and having filtered away the starch, applied the test to the fluid; a beautiful blue colour was immediately produced. It was evident from this that the *gluten* was not the part acted on. The blue liquid was then heated well, the albumen coagulated, and the blue colour remained with the coagulum, whilst the remaining fluid was free from colour. On raising the heat to 212°, the blue colour entirely disappeared. On the other hand, the test was applied to the starch collected on the filter, but without effect. The experiment was varied in the following manner:—

The fluid was first filtered to separate *starch*, then boiled and filtered to separate *albumen*. On the test being applied both to the filtered fluid, and to the *albumen* on the filter, no blue colour was obtained.

From these experiments I drew the following conclusions:—

I. That albumen is the principle acted on.

II. That a heat above 180° destroys this action.

[1] Thomson—London Medical Repository, vol. xiv.

[2] Battley—London Medical Repository, vol. xiv.

III. That the value of this test is to prove that the bulbs have been dried at a temperature not higher than 180°.

During the summer of 1849 I endeavoured, by another process, to determine the medicinal value of the drug at different seasons, by ascertaining the amount of alkaloid contained in the bulb at monthly periods, and thus to determine the fittest time for gathering for medicinal use.

Having expressed the juice of several bulbs, and separated the *albumen* and *starch*, I attempted by precipitation in a graduated test-tube with *tincture of galls*, to determine by the bulk of the precipitate the amount of active principle. This, however, failed, on account of the precipitate partly floating to the top, and partly remaining at the bottom. This I believed was occasioned by the fluids being of different densities. A similar experiment was tried with an alcoholic solution of the bulb, but with little better success, as the small quantity of active matter procured from a necessarily small amount of bulbs gave an almost imperceptible precipitate.

At whatever period the bulbs are taken up, they should be sliced into small pieces about the thickness of a half-crown, the outer membrane having been previously peeled off. They should be then spread out upon trays to dry, either at ordinary temperatures, or at any rate not exceeding that of 150° F., after which they must be carefully preserved from moisture. (Christison.)

Mr Houlton [1] recommends that the bulb should be stripped of its dry coating, carefully deprived of the bud or young bulb, and then dried whole.

CHEMICAL HISTORY.

Vegetable chemistry has of late years made great and rapid progress, and in no way more practically useful than in the discovery and examination of the organic bases which constitute the active principles of many vegetables used as medicines.

It is hardly necessary to do more than allude to *quina* and *morphia*, as examples of the valuable contributions which chemistry has made of late years to the resources of the medical practitioner.

It happens, however, in many instances that we may be quite aware of the existence, in a medicine, of an active principle, and yet, on the one hand, from the difficulty of obtaining it pure, or on the other, from the activity of the crude drug in small doses, it may, in a practical point of view, be of little consequence to ascertain the precise chemical and other qualities of the active ingredient. Yet assuredly the more we can investigate these active principles the

[1] Houlton—Pharmaceutical Journal, vol. iv.

more precise will be our knowledge of the properties of the original drug.

The active principle of COLCHICUM AUTUMNALE was, on the authority of Pelletier and Caventou,[1] supposed to be identical with that of Veratrum album,—namely, VERATRIA. Geiger and Hesse,[2] however, discovered in COLCHICUM an alkaloid differing from veratria, and which was named by them COLCHICIA.

The following was their process for preparing it:—

The seeds were bruised and exhausted by digesting them in *rectified spirit*, acidulated by *sulphuric acid*. The excess of acid was removed by the addition of *hydrate of lime*, and the fluid filtered to separate the *sulphate of lime* which was deposited. Any excess of *lime* which might remain in the spirituous fluid was removed by the careful addition of one or two drops of *sulphuric acid*. The fluid was then filtered and distilled to recover the alcohol, and the watery residue of the distillation was mixed with an excess of *carbonate of potass*. The precipitate which fell was dried between folds of bibulous paper, and then was taken up in *absolute alcohol*, the alcoholic solution decolorised by *animal charcoal*, and evaporated for crystallisation at a gentle temperature.

By this process, it is said, COLCHICIA may be obtained also from the flowers and corms.

During the winter of 1849, and again during that of 1850, I endeavoured to procure Geiger and Hesse's COLCHICIA by this method, but without success, although I followed their process exactly. I was particularly cautious in the application of heat; for it is well known that many of the vegetable bases, such as those from *Hyoscyamus* and *Stramonium*, are very easily destroyed by an undue elevation of temperature, which may probably account for those and other similar bases not having been accurately examined and analysed. COLCHICIA is perhaps as easily destructible as *hyoscyamia* and *daturia*.

I have not been able to find that any chemists have procured crystals of COLCHICIA, except Geiger and Hesse. My account of its properties, therefore, rests solely on their authority.

"COLCHICIA crystallises from its alcoholic solution, when that is mixed with water, in colourless prisms and needles. If the alcoholic or ethereal solution be evaporated, the COLCHICIA remains in the condition of a transparent varnish-looking substance."

It was in this state alone that I procured anything which could be considered to be COLCHICIA. I am very sceptical with regard to the crystalline nature of COLCHICIA at all; for having dissolved this matter successively in *alcohol*, *ether*, and *water*, and having left

[1] Pelletier and Caventou—Journal de Pharmacie, tome vi.
[2] Geiger and Hesse—Geiger's Pharmacie, tome i. Annalen der Pharmacie, tome vii. Journal de Chimie, tome x.

the solutions to spontaneous evaporation, no trace of crystallisation ever appeared, but the bitter matter was invariably deposited in the state of a brown resinous-looking mass.

This brown mass was without smell, and possessed considerable bitterness, the bitter taste being generally followed by a slight sense of irritation in the throat, but not with the intense acrimony of VERATRIA.

"COLCHICIA in its hydrated condition has but a feeble alkaline re-action, but neutralises acids completely, and forms with them salts, which are in part crystallisable, and which have a bitter, acid, and slightly acrid taste.

"COLCHICIA dissolves pretty easily in water. It is very soluble in rectified alcohol and ether.

"It strikes a yellow colour with solution of *chloride of platinum*, but does not form an insoluble *platino-chloride* with it. *Tincture of galls* causes a white precipitate, and with *tincture of iodine* it becomes rapidly turbid, producing a russet-brown colour.

"It is persistent in the air, melts easily at a gentle heat, and is destroyed by a higher temperature." (Geiger and Hesse.)

The alkaloid VERATRIA, discovered by Pelletier and Caventou, in the root of *Veratrum album*, and in the seeds of *Helonias* or *Asagræa officinalis*, is prepared much more easily. I found no difficulty in procuring it by the process of the Edinburgh Pharmacopœia, which is nearly the same as that described by Couerbe,[1] and is as follows :—

"Grind the seeds of *Cevadilla* in a coffee-mill, and form them into a thick paste with *rectified spirit*. Pack this firmly in a percolator, and pass *rectified spirit* through it till the spirit ceases to be coloured. Concentrate the spirituous solutions by distillation so long as no deposit forms, and pour the residuum while hot into twelve times its volume of cold water. Filter through calico, and wash the residuum on the filter so long as the washings precipitate with *ammonia*. Unite the filtered liquid with the washings, and add an excess of ammonia. Collect the precipitate on a filter, wash it slightly with cold water, and dry it, first by imbibition with filtering paper, and then in the vapour bath. A small additional quantity may be got by concentrating the filtered ammoniacal fluid, and allowing it to cool."

The Pharmacopœia, I think, has erred in not ordering the immense volume of water, into which the hot alcoholic solution is thrown, to be evaporated down. In one of my operations the quantity of water amounted to twenty-four pints; and it was found quite impossible to precipitate this immense quantity by *ammonia*. It was therefore evaporated down to four pints, when an abundant precipitate was obtained.

The quantity of impure VERATRIA which was obtained from

[1] Couerbe—Annales de Chimie et de Physique, tome lii.

fifty-six ounces of seeds was *fifty grains*; but Couerbe states that *a drachm* of VERATRIA may be obtained from *sixteen ounces* of seeds; but, as Dr Christison remarks, "the product to be so large must be very impure."

The impure *Veratria* which I procured was taken up in very weak *hydrochloric acid*, decolorised with *animal charcoal*, and re-precipitated by *ammonia*. It was then nearly pure white, and weighed *twenty-seven grains*.

That VERATRIA and COLCHICIA (if Geiger and Hesse's account of the latter be correct) are in no respect identical, is obvious from their great difference in properties, viz.,—

(1.) VERATRIA is entirely incrystallisable.	(1.) COLCHICIA crystallises in delicate acicular prisms.
(2.) VERATRIA possesses a powerful persistent acridity of taste.	(2.) COLCHICIA, though extremely bitter, possesses none of this.
(3.) VERATRIA, when it comes into contact with the nostrils, excites most violent sneezing. So powerful indeed is this property of VERATRIA, that when engaged in working with the seeds of *Cevadilla*, I have frequently been obliged to desist, on account of the sneezing induced.	(3.) COLCHICIA possesses no sternutatory effects at all.
(4.) VERATRIA is almost entirely insoluble in *water*. It is soluble in *alcohol* and *ether*.	(4.) COLCHICIA is very soluble in *water*, *alcohol*, and *ether*.
(5.) VERATRIA, by the action of *nitric acid*, becomes first red and then yellow. *Sulphuric acid* produces first a yellow, then a blood-red, and lastly a fine violet colour.	(5.) On COLCHICIA *nitric acid* first produces a bright violet, then an indigo-blue colour, quickly passing into green and yellow. *Sulphuric acid* colours it yellowish-brown.

These comparative characters are sufficient to establish the complete dissimilarity of these two bases.

COLCHICIA has not yet been analysed, and therefore no formula of its constitution exists.

Geiger and Hesse, although they did not analyse their COLCHICIA, ascertained its physiological properties, and compared them with those of VERATRIA.

The following experiments are related by them :—

One-tenth of a grain of COLCHICIA dissolved in weak alcohol was administered to a cat eight weeks old. There formed immediately much froth at the mouth. At the end of about an hour there were abundant liquid dejections, and then followed, after an interval, several attacks of vomiting. The gait of the animal became staggering, it fell, rolled from side to side, uttered a plaintive cry, and appeared agitated by convulsive movements. These symptoms augmented more and more, and death took place in twelve hours.	There was given, for comparison, one-twentieth of a grain of VERATRIA to a cat a little younger. The poison showed itself with much greater intensity. The animal staggered, moved convulsively, and died in ten minutes.

On opening the body, the stomach and intestinal canal were found violently inflamed, and congested throughout their entire course.

On inspection, no inflammation was found, except at the inferior part of the œsophagus, which part of the digestive tube was not inflamed in the cat poisoned by COLCHICIA.

It appears, from these experiments, that the poisonous properties of COLCHICIA are essentially those of the *irritant* class, whilst, from the rapid action of VERATRIA, and the absence of post-mortem appearances, it seems to have produced death by an action on the *nervous system*.

No other important principle has been announced as existing in COLCHICIA, except the alkaloid discovered by Geiger and Hesse. It was the substance taken by Pelletier and Caventon for VERATRIA; and, according to their statement, it was combined with *gallic acid*.

The following is their analysis of the corms:—

(1.) Fatty matter, composed of { Olein. Stearine. Volatile acid.
(2.) Supergallate of VERATRIA (*Colchicia.*)
(3.) Yellow colouring matter.
(4.) Gum. (5.) Starch. (6.) Inulin. (7.) Lignin.[1]

Stoltze[2] also analysed the bulb of COLCHICUM, and found that it contained in

	March.	October.
Volatile acrid matter,	a trace.	rather more.
Soft resin,	0·04	0·06
Crystallisable sugar,	0·41	1·12
Incrystallisable sugar,	5·91	2·72
Bitter extractive		2·17
Difficultly soluble extractive,	1·30	0·52
Gum,	0·81	1·65
Starch,	7·46	10·12
Lignin,	2·32	1·61
Extractive soluble in potass,	0·61	0·52
Water,	81·04	80·31
	99·90	100·80

We find by this analysis that in *March* the *starch* is diminished 3 per cent., and the bitter extract greatly increased.

Another analysis was performed by Melandri and Moretti.[3]

The *seeds* have only been analysed qualitatively by Buchner,[4] but his notice of the constituents is not worthy of remark.

[1] Pelletier and Caventou—Journal de Pharmacie, tome vi.
[2] Stoltze—Thomson's Organic Chemistry.
[3] Melandri and Moretti—Bulletin de Pharmacie, tome ii.
[4] Buchner's Repertorium, xliii.

PHARMACY.

Much discussion has been held respecting the comparative value of the different preparations of COLCHICUM, and especially as to whether the seeds or corms yields the most eligible forms for its administration.

"It might be reasonably expected," says Dr Barlow, "from the virtues of COLCHICUM being found to reside in the seeds as well as in the root, that the former would yield a medicine of greater uniformity, being in a state of more perfect and determinate maturity, requiring less care in the collection and preservation, and being less liable to have their powers impaired. My experience of the several preparations fully confirms this supposition."[1]

This is upon the whole the opinion most generally entertained by practical physicians.

The employment of the *seeds* in preference to the *root* was first insisted on by Dr Williams of Ipswich.[2]

There are at present seven preparations of the bulb and seeds of COLCHICUM officinal in the three British Pharmacopœias; but of all these there are perhaps only four which can be considered as in use at the present day. These are,—*Acetum Colchici*, E. L. D.; *Extractum Colchici Aceticum*, E. L. D.; *Tinctura Colchici*, E. L.; and *Vinum Colchici*, E. L. The *acetic* forms answer best as *diuretics*, perhaps because they are weaker preparations, and not so apt to act upon the bowels.

The *tincture* and *wine* are the preparations employed for general purposes; but when it is desirable to exhibit COLCHICUM in the form of pill, the *acetic extract* is certainly the most eligible form.

PHYSIOLOGICAL EFFECTS.

It is very difficult to state in a precise and definite manner what the action of COLCHICUM is, or to what class of medicinal agents it ought to be referred.

In medicinal doses it seems to have the property of lowering the heart's action, and to affect most of the secretions of the body; and hence, according to the circumstances under which it is administered, it may produce *diuretic*, *emetico-cathartic*, or *diaphoretic* effects.

It has been regarded by some as an *expectorant;* and by others it is supposed to have a peculiar effect in stimulating the *hepatic system*.

It has also been regarded as having a powerful influence over the *uterus*.

From the marked effects which it produces in GOUT and RHEU-

[1] Cyclopædia of Practical Medicine—Gout.
[2] Williams—London Medical Repository, vol. xiii.

MATISM, it has been regarded as possessing a *specific* action in those diseases; but this may be said to be a mere statement that it acts powerfully and successfully, for it does not appear that it often alleviates those diseases, without producing in a well-marked degree some of its ordinary physiological effects, such as lowering the pulse, causing *diarrhœa*, *diuresis*, or *diaphoresis*.

In large doses there can be no doubt that it is an active *narcotico-acrid* poison; but its action seems to be more due to *acrid* than to *narcotic* properties, as the effect on the brain is generally secondary to that on the intestines.

One of its most remarkable physiological effects was discovered by Chelius of Heidelberg.[1] He found that the URIC ACID contained in the urine of those taking COLCHICUM was nearly doubled in the space of twelve days. In one case the urine before taking COLCHICUM contained 0·069 per mille of URIC ACID; four days after commencing to take the COLCHICUM the proportion was 0·076; on the eighth day, it was 0·091; and on the twelfth day, it was 0·102. Chelius obtained the same results in other instances.

Dr Christison[2] examined the urine of a patient taking COLCHICUM, and he found that in two days the quantity of UREA was nearly doubled. In the urine before taking COLCHICUM there was no deposit of *lithate of ammonia*. Its density 1020. It contained above forty-seven parts of solid matters in the thousand, and of this quantity twenty parts were UREA. The specimens of urine passed on the first and second days after commencing to take COLCHICUM were exactly alike. They were very turbid, and their turbidity disappeared with a gentle heat; the deposit was evidently *lithate of ammonia*. The density of the first was 1033·5, and that of the second was 1034, which are both very unusually high for urine not *diabetic*. As they were obviously identical in their nature, Dr Christison only analysed the second. It contained seventy-nine parts of solid matters in a thousand, and of this quantity thirty-five were UREA. Dr Christison suspected that the quantity of urea was even greater, for not having added an excess of *nitric acid*, some of the *nitrate of urea* might have remained in solution.

Through the kindness of Dr Halliday Douglas, I had an opportunity of examining the effect of COLCHICUM on the urine of a sailor, who was a patient in the Royal Infirmary. He was under treatment for secondary syphilis, but was otherwise healthy. I was permitted to give him a few doses of COLCHICUM, in order that I might ascertain the physiological action of that agent on

[1] Chelius—Archives Générales de Médecine, tome xviii. Chelius—Annales Cliniques de Heidelberg, 3me vol. *Vide* paper by Dr Lewins—Edinburgh Medical and Surgical Journal, vol. lvi.

[2] Christison—Lewins, op. cit.

the kidneys, but before doing so I examined his urine. The density was 1025. It contained no deposit, nor was it affected by *heat* or *nitric acid*. It contained :—

Total solids, ...	27·500
Water, ...	972·500
Urea, ...	12·360
Uric acid, ...	0·231
Inorganic salts, ...	7·436
Organic matter, ...	7·423
Total, ...	1000·000

Here it will be perceived that both the UREA and URIC ACID were slightly deficient, if we compare it with the standard of healthy urine, as given by Becquerel.[1] Density 1018·9 Contains :—

Total solids, ...	31·185
Water, ...	968·815
Urea, ...	13·333
Uric acid, ...	0·391
Inorganic salts, ...	7·695
Organic matter, ...	9·261
Total, ...	1000·000

On the third day, after commencing to take COLCHICUM, the urine was examined. It possessed a slight turbidity, which, however, was dissipated by heat. Density 1030. It contained :—

Total solids, ...	29·650
Water, ...	970·350
Urea, ...	15·500
Uric acid, ...	0·491
Inorganic salts, ...	6·350
Organic matter, ...	7·209
Total, ...	1000·000

Here, it will be observed, the UREA was increased by *one-fourth*, the URIC ACID nearly *doubled*, and the inorganic salts and inseparable organic matters were considerably decreased.

The urine was again examined on the sixth day after commencing to take the COLCHICUM, with the following results. Turbidity rather increased. Density 1034. It contained :—

Total solids, ...	33·460
Water, ...	966.540
Urea, ...	18·341
Uric acid, ...	0·750
Inorganic salts, ...	7·436
Organic matter, ...	6·933
Total, ...	1000·000

[1] Becquerel—Semeiotique des Urines.

Here, then, the physiological action of COLCHICUM in increasing the UREA and URIC ACID was well marked.

Having obtained these results from this case (which are only corroborations of many others), it was not considered justifiable to proceed further with the administration of COLCHICUM with this patient.

It has been supposed that under the use of COLCHICUM a remarkable change takes place in the system,—namely, that the URIC ACID becomes converted into UREA; but this has not at all been substantiated, and from the above cases of Chelius, and the analyses which I have just noticed, we must be led to suppose that no such change occurs, but that an increase in both those principles is the result.

Dr Graves[1] states, that the beneficial action of COLCHICUM is not owing to its producing a more rapid excretion of *lithates* through the kidneys, but to the remarkable property the plant possesses of altogether putting a stop to the morbid formation of *lithates*.

Dr Gairdner[2] says, that he has always found that the increase of UREA was accompanied by a corresponding diminution of the *urates* in the urine. But, from the above experiments, I am inclined to believe that both of these suppositions are erroneous.

With a view to ascertain the power of COLCHICUM as a *sedative* I made the following experiments,—in the first, my pulse being 87; in the second, 84. On both occasions twenty minims of *Tinctura Colchici* were taken.

At 8 P.M.	...	...	Pulse 87	At 6 P.M.	...	...	Pulse 84
9 „	...	...	„ 87	7 „	...	...	„ 84
10 „	...	...	„ 80	8 „	...	...	„ 78
11 „	...	...	„ 75	9 „	...	...	„ 72
11½ „	...	...	„ 70	10 „	...	...	„ 66
12	...	...	„ 65	11 „	...	...	„ 60
12½ A.M.	...	...	„ 65	12	...	...	„ 62

No other physiological action was manifested, except slight nausea.

POISONOUS ACTION.

In large doses, COLCHICUM proves a violent *irritant* poison. The symptoms occasioned by it are,—"Severe vomiting and urgent diarrhœa, with a sense of burning in the throat, excessive colic, and heat in the abdomen; great depression of the circulation, and sometimes suppression of urine.

"Sometimes no other symptoms exist, and death takes place from *exhaustion*, the effect of *inflammation* of the bowels. Sometimes death is preceded by *headache, delirium, stupor*, and *insensibility*, denoting an action upon the *nervous system*." (Christison.)

[1] Graves—London Medical Gazette, vol. vii.
[2] Gairdner on Gout. London, 1849.

POISONOUS ACTION ON ANIMALS.

Orfila[1] states, that he has frequently given two or three bulbs to dogs in *June*, without producing any sensible effects; this induced him to believe that climate and the season of the year have great influence on their deleterious properties. This, however, is the season of the year when the bulbs are supposed to possess their greatest activity.

Mr Want states, "that cattle are affected by it only at the spring of the year, when the seed-vessel is fully mature."

"It happens that the seed, if swallowed, adheres to the coat of the stomach, producing at the several points of its adhesion spots of inflammation, which occasion the death of the beast."

"It is a curious fact, that they are affected by the recent plant only; for when dried and made into hay it loses its deleterious property, and is then eaten by them with impunity."[2]

Sir Everard Home[3] injected 160 drops of the vinous infusion of COLCHICUM into the jugular vein of a dog. The animal immediately lost all power of voluntary motion; the breathing became extremely slow, and the pulse was hardly to be felt.

In ten minutes the pulse was 84; inspirations natural (40 in a minute). In twenty minutes the pulse was 60; inspirations 30; and a tremulous motion had taken place in the hind legs. In an hour the pulse was 115, and irregular. The animal was capable of sitting up, but was in a state of violent tremor; the inspirations could not be counted. In one and a half hours, the tremor had gone off; pulse the same; the animal made ineffectual attempts to vomit, and continued to do so for ten minutes, with great langour; inspirations, 54. In two hours the pulse was 150, and very weak; the animal had voided an ounce and a half of urine; had vomited twice, each time bringing up a quantity of mucus, tinged with bile, and had two liquid stools. In three hours had vomited again, and had another stool; pulse too weak to be counted. In four hours continued extremely languid; and in five hours vomited some bloody mucus, and expired.

Autopsy.—The *stomach* contained mucus tinged with blood, and its internal surface was *inflamed*. The *duodenum* had its internal surface universally *inflamed*. The same appearance was met with

[1] Orfila—Toxicologie, vol. ii.
[2] Want—London Med. and Phys. Journ., vol. xxxii.; Annals of Philosophy, vol. iv.
[3] Philosophical Transactions, vol. xvi., part 2.

in the *jejunum* and *ilium*, and also in the *colon*, where it was more strongly marked than in the *ilium*.

Another case, with somewhat similar results, is recorded by Sir Charles Scudamore.[1] He injected 160 drops of the vinous infusion of COLCHICUM into the jugular vein of a strong dog. For the first fifteen minutes he did not seem to suffer the least inconvenience. In an hour and a quarter he was sitting on his hind legs, the eyes were bright, and there did not appear any remarkable change. In five hours he was still capable of sitting up, but appeared much dejected; and there was some difficulty of breathing, attended with an occasional husky cough. His pulse was 113; the inspirations were 56. In five hours and a quarter he vomited some bloody mucus, and expired.

Dissection.—The *stomach* was in a state of *gangrene*. The *duodenum*, *jejunum*, and *ilium*, were in a high state of *inflammation*, approaching to *gangrene*. About two ounces of highly offensive grumous blood were found in the stomach; there was also blood in the duodenum, but not offensive. The *colon*, *cæcum*, and *rectum* were much *inflamed*; with here and there deep rose-coloured spots, of the size of a pea. The bladder was nearly full of urine, of a deep saffron colour; the gall-bladder was much distended with bile. There was an effusion of bile upon the liver.

Scudamore made an experiment also with the expressed juice of COLCHICUM.

At forty minutes past 1 o'clock P.M., 120 drops of the expressed juice of the fresh roots of COLCHICUM were injected into the jugular vein of a young terrier. Immediate signs of great debility were produced; he passed a natural alvine discharge.

In about two minutes he vomited half an ounce of frothy bile, of a gamboge colour. Rising upon his legs he staggered as if intoxicated, and immediately fell. In five minutes the respirations were 52. In seven the eyes were fixed, the pupils contracted; the extremities were stiff, and drawn up.

In ten minutes he was upon his legs, passed a scanty bilious discharge, walked about, and soon again made great efforts, without effect, to relieve himself, uttering at the time a cry of distress. He ran into a dark part of the room. In lying down he had some rigors, and there was a profuse salivation.

Three o'clock.—He was on his legs, with his back raised, and showing signs of pain in the bowels. The inspirations were 42. He seemed capable of walking, but was very languid.

At *four o'clock* the lassitude had increased, and he supported himself with difficulty on his legs; a copious discharge of mucus and saliva was proceeding from the mouth.

[1] Scudamore on Gout and Rheumatism, 1819.

A little before *five o'clock* he appeared to be suffering much, was moaning continually, and took no notice on being disturbed.

At *six o'clock* he was still much distressed on his inspirations, being only 12 in a minute; the eyes were fixed, the teeth firmly closed, and he appeared to be dying.

At *seven o'clock* there was no perceptible change, except that his inspirations had increased to 14 in a minute.

Nine o'clock.—He respired only 8 times in the minute with much difficulty. His moaning was much fainter.

Ten o'clock.—He was found dead, and was quite cold.

Dissection.—The *stomach* was highly *inflamed*, containing about an ounce of dense mucus, mixed with grumous blood; the *duodenum*, *jejunum*, and *ilium*, were highly *inflamed*; the inflammation decreasing in the *colon*, *cæcum*, and *rectum*.

The following experiment with *Eau Médicinale*, whose principal constituent is supposed to be some preparation of COLCHICUM, probably the *flowers*, is narrated by Scudamore:—

Sixty minims of Eau Médicinale, with the sediment which it forms shaken up, were given to a very strong rough terrier, at half-past ten A.M.

Two o'clock.—He was lying down, and looked lethargic.

Half-past Four.—Pulse 96, vibrating and intermitting every five beats.

Eight o'clock.—Pulse was softer; he had vomited some frothy mucus, and appeared very languid.

Next morning.—Pulse 164, and irregular. The dog had recovered his strength.

Ten o'clock.—A further dose of 160 drops was given.

Half-past Two.—He looked dejected; the pulse was 104, and very irregular.

Four o'clock.—Had brought up some opaque, viscid mucus.

Six o'clock.—Had vomited a quantity of frothy slime mixed with blood, and appeared altogether very ill. Rigors; pulse 80, and small, with intermissions after every five or six beats.

Half-past Nine.—Seemed dull and languid; pulse 106.

On the following morning, at ten o'clock, he was found extended on the ground; had voided a quantity of urine. He was quite insensible, and now and then stretching out his limbs. His inspirations were 6 in a minute.

At *one o'clock* he did not seem to suffer.

Half-past Two.—He was stretching himself out, as if in the act of dying. There were slight convulsions of one of the legs. The pulsation of the heart was not to be felt; and no distinct respiration could be observed.

At *three o'clock* he had expired.

Dissection.—The *stomach* was highly *inflamed*, and contained a dark-brown fluid. The marks of *inflammation* increased in the *duo-*

denum, and through the *jejunum;* decreased in the *ilium*, and increased again in the *colon*, which appeared in a state of general ecchymosis, from venous blood extravasated under the mucous membrane. The *cæcum* and *rectum* were highly *inflamed*.

Various other experiments with the different preparations of COLCHICUM are noticed in Scudamore's work on gout, but these it is unnecessary to detail.

Dr R. Lewins,[1] in an essay on COLCHICUM, describes several experiments which he made on the lower animals with different preparations of that plant.

On the 15th December, 70 minims of the *Vinum Seminum Colchici* were administered to a middle-sized dog, without any immediate sensible effect.

A short time afterwards it devoured a large quantity of meat very greedily, and continued gnawing bones for some time with great avidity.

At *seven* P.M., nearly four hours after he had swallowed the COLCHICUM, he ate another large quantity of beef, and lapped barley-broth with great eagerness. He was visited for the last time that night about *nine* P.M., when he appeared in his usual state of health.

Next morning, at *half-past eight*, sixteen hours after the COLCHICUM had been given, he seemed weak, low, and very sick. He remained prostrate on some straw in the corner of the room, and could not be induced to move. During the night he had been most severely purged, and in every part of the room were egesta from the stomach, in some places mixed with a fluid of a dark brown colour, in other places mixed with food; and near where he was lying there were large watery evacuations, intimately mixed with a fluid resembling blood. No attempts were made to vomit during the visits on this day, nor did he express by outward symptoms any indication of suffering acute pain. The eyes were hollow, and had a dull glazed appearance. On presenting food of the same kind that he devoured greedily the previous afternoon, he would not touch it. The prostration of strength and insensibility to external impressions became greater and greater throughout the day.

On visiting him next morning, at a little after *eight o'clock*, he was quite dead, cold, and stiff. The food which had been placed before him yesterday morning had never been touched.

Dissection.—The body, examined at *half-past eleven*, presented the following appearances:—The *intestines* were found very much *contracted* and *vascular*. The internal surface of the *stomach* presented rather more rugæ than natural, and was lined with mucus; its cavity was nearly full of dark brown coloured bile, and the

[1] Lewins—Edin. Med. and Surg. Journ., vol. lvi.

pyloric orifice was contracted. The *duodenum* was much injected, and coated with mucus; the whole course of the *jejunum*, and upper part of the *ilium*, were of the most intense red colour. On the lower part of the *ilium* were observed a number of dark streaks, slightly raised, running in a longitudinal manner. The *large intestines* were likewise very *vascular*, although not so vivid as in the small intestines. In the *ilium* a large tapeworm, measuring considerably upwards of a foot, was found dead. There was not the slightest appearance of food or fœcal matter in any part of the intestinal canal; a large quantity of bloody serosity, mixed with thin mucus, lined the parietes in almost their whole extent. The *gall-bladder* was full of dark-brown bile, and the *urinary bladder* was distended with natural coloured urine. All the other organs were perfectly normal.

Dr Lewins found that, notwithstanding the powerful effects which COLCHICUM produced upon men and dogs, it acted with very different degrees of energy upon other classes of animals, and with the view of ascertaining in what respect this action is different, he made the following experiments:—

On 7th December, half a drachm of the *Vinum Seminum Colchici* was administered to a small rabbit. It commenced eating immediately afterwards; and on the following day nothing unusual was observed, except that it appeared to have passed a larger quantity of urine than natural.

On the 8th, at *six* P.M., twenty-four hours after the last dose, one drachm was administered, and with the exception of a *diuretic* effect, no particular change was observed.

On the 9th, no perceptible effect having resulted from yesterday's dose, at *nine* P.M., twenty-seven hours after the last experiment, two drachms more were swallowed, and no evident change was observed in the animal.

On the 10th, at *six* P.M., twenty-one hours after the last dose, three drachms more were given. After one drachm had been introduced, the rabbit began to squeak, and struggle violently; and on being let loose ran about as if in pain. The same effect followed after the exhibition of the second dose; but after the third drachm it began to eat, and appeared quite as lively as usual.

Thus to a very small animal, in the course of four days, *six drachms and a half* were given (more than six times the quantity which proved fatal, with the most violent symptoms, to a dog), with little effect except acting upon the kidneys.

Another case is mentioned, where *seventeen drachms* were given in *six* days, without any material change being produced on the animal. An experiment was also made on a cold-blooded animal,—viz., a frog. In this case, in the course of *six* days, 350 *minims* were administered, with comparatively very slight effects

being produced, and which might very well be attributed to the quantity of wine swallowed.

From the results of these investigations, Dr Lewins says, it appears that we are warranted in concluding, that the action of COLCHICUM is most decided upon carnivorous and omnivorous animals, whilst its action on herbivorous, graminivorous, and cold-blooded animals is comparatively feeble.

POISONOUS ACTION ON MAN.

THE following cases of poisoning by the various preparations of COLCHICUM are illustrative of the fact which has been pointed out, that its action in this respect is due more to *acrid* than to *narcotic* properties, inasmuch as the symptoms of the latter, when they appear at all, are generally subsequent to those of the former. In all the cases—*colic, vomiting*, and *purging, slow and almost imperceptible pulse*, and great *prostration of strength*, are represented as having occurred, whilst only in three was there any distinct manifestation of an action upon the *nervous system*. In one case only does the action on the nervous system *precede* the symptoms of acrid poisoning, and in this, *convulsions*, followed by complete *opisthotonos* and *paralysis*, were present. In three cases, the *pupils* were remarked to be *dilated;* in one, *contracted;* in the majority, *suppression of urine* existed; but in one, *diuresis* was present from the time of poisoning to that of death—a period of six weeks.

The post-mortem appearances are in general *redness* and *inflammation* of the intestinal canal; but in the cases mentioned by Chevallier and Caffe, no morbid appearances existed.

The following case, illustrative of the poisonous effects of the *seeds* of COLCHICUM, is narrated by Mr Fereday, of Dudley:[1]—

I.—David Cole, æt. 44, a stout muscular man, feeling pains in his bowels, to which he was subject, on the morning of 8th March, about six o'clock, swallowed, believing it to be rum, about two ounces of wine of the seeds of COLCHICUM.

He immediately discovered his error, but knowing its effects in small doses, conceived it would be followed by vomiting and purging sufficient to avert mischief. He sought no medical aid till four in the afternoon, when he was first seen. He was sitting on a chair, his elbows on his knees. He said that he felt no inconvenience for an hour and a half after taking the dose, when pains in the bowels came on; but that he continued his work until eleven o'clock, when pains in his stomach and bowels, retching, and copious vomiting of a yellowish fluid, compelled him to desist.

Four o'clock p.m.—He describes the pain in the epigastrium as agonising, and says it is like a knife piercing him. The retching is incessant and extremely violent, but no fluid is evacuated; there is tenesmus; a small quantity of fœcal matter has passed. No tenderness on pressure, either in the epigastrium or abdomen. The appearance of the tongue is natural; the pulse small, slow, and feeble; breathing not much affected; the feet cold; his countenance is anxious; features sharp; his cheeks, lips, and palpebræ purple. On attempting to walk, says he thinks he shall lose the use of his limbs.

A mustard emetic was given, followed by copious draughts of warm water and gruel. These were soon returned, with apparently no admixture. Cathartic medicine was given, and immediately returned. Was put to bed; warm bricks were applied to the feet, and hot flannels to the stomach. To take forty drops of laudanum immediately; gruel and coffee plenteously.

Nine o'clock p.m.—The retching, vomiting, and pain in the stomach continue with undiminished violence; the fluid vomited contains a sediment like coffee grounds; he complains greatly of thirst; has made little water. Twenty drops of laudanum every two hours; a blister to the epigastrium; sinapisms to the feet; an enema every hour.

9th March, six o'clock a.m.—Has passed a sleepless night; the symptoms remain unaltered; the eyes are sunk; feet warmer; skin generally natural; no perspiration; pulse scarcely to be felt; respiration hurried; great thirst; no urine. Enemata returned without fœcal matter; camphor, calomel, and opium every three hours; an effervescing draught with brandy every hour.

Eight o'clock p.m.—The retching and pains continued until four o'clock, when the bowels were much distended. Has since had copious liquid stools, dark coloured, and very offensive, and expresses himself better. Makes a few drops only of urine; loses his sight for a minute or two after getting out of bed to the night chair; the pulse is scarcely perceptible, and occasionally intermits; he is perfectly sensible, but talks with effort; calls continually for water. Aromatic confection, carbonate of ammonia, and camphor mixture, with brandy, every hour.

10th March.—In the course of the night his stools passed involuntarily, and in great numbers, his weakness increased, and he died a few minutes before five o'clock this morning, perfectly sensible to the last moment.

Sectio Cadaveris.—The face, neck, upper and front part of the thorax, insides of the arms, front of each forearm, and insides of the thighs, were covered with patches of a purple efflorescence, as were also the integuments of the scrotum and penis. The muscles of the forearm were very rigid, and their fibres contracted into hard knobs. The great omentum, instead of covering the front of the intestines, was turned up between the stomach and convex surface of the liver behind, and the diaphragm in front, from the efforts of vomiting. There was increased redness in a portion of the peritoneum covering the jejunum. The stomach and bowels were coated with a thick, tenacious, but colourless mucus. On a portion of the mucous membrane of the stomach, near the cardiac orifice, and corresponding to its great arch, was a patch of redness, about the size of a half-crown piece; its secretion here did not vary in tenacity, quantity, or colour, from that of any other portion of the membrane. Upon dividing it at this part, its section presented nothing beyond its usual appearance; there was no pulpiness, no thickening, but a small quantity of blood was effused between it and the muscular coat, giving the reddened internal appearance. Careful examination of that portion of the reddened peritoneum covering the jejunum, demonstrated the like hemorrhagic condition of the blood-vessels. Blood was effused between the peritoneal and muscular coats; but the mucous membrane corresponding to this portion was perfectly healthy, at least it was perfectly free from inflammation. No other trace of inflammation was observed in other portions of the abdominal viscera. The gall-bladder was distended with healthy bile, the urinary bladder was contracted and empty.

[1] Fereday—London Medical Gazette, vol. x.

II.—Case of poisoning by a decoction of the *seeds* of COLCHICUM, by Dr Newbrandt.[1]

Caspar B., of Aesthausen, æt. 52, of a sanguine temperament, drank, by mistake, on the night of the 18th February 1830, some of a decoction made with a large spoonful of colchicum seeds and three pints of water; he had, in the night, more than fifteen stools and vomitings. When Dr Newbrandt saw him next day, he was in a disturbed state. The stools and vomitings were less frequent; the patient, although weak, did not complain of any pain, and could raise himself; the abdomen was not distended, and it contracted spasmodically on being touched; the pulse was small and frequent; the stools, which were very fœtid, contained small whitish membranes. The patient was made to drink a great quantity of warm water containing butter. This drink provoked vomiting and stools. Immediately after, coffee was ordered, and a strong infusion of marsh-mallow with lemon-juice. Next morning, the 20th, at eight o'clock, the physician found his patient in the following state:—

Face pale; respiration precipitate; eyes sunken; pupils much dilated; tongue covered with a whitish matter, and could be put out only with difficulty; region of the stomach rather painful; breath, face, and extremities cold; pulse very frequent, scarcely perceptible; no thirst; stools more frequent since last evening, and containing matters of a light blue colour. The patient took with pleasure some mucilaginous soups and coffee. Although he replied correctly to questions addressed to him, his intellectual faculties seemed to be confused. Death at ten o'clock.

Sectio Cadaveris, 23 hours after death.—Countenance unaltered; pupils much dilated; eyes sunken; the abdomen scarcely more swelled than during life, was of an extraordinary hardness, and showed peculiar stains, more numerous in the cavity of the stomach, and at the sides, towards the back; they were violet, greenish-blue, not circumscribed. The muscles were of a deep blue when dried in the air. The trachea, towards the bifurcation, was inflamed. The lungs collapsed, small, pale, and soft to the touch, containing much coagulated blood; on their surface were large black, violet, and brownish spots. The œsophagus was brownish-red at its opening into the stomach. The cardia was of a violet-black colour. The stomach, at its exterior surface, was of a light violet, and much deeper at the interior; the veins of the stomach and other intestines were greatly distended with perfectly black blood. The liver had a violet tint at its concave surface. The gall-bladder was bulky, and full of green bile. The large and small intestines were hardly inflamed without, and showed only a few red, brownish spots within. The other organs presented nothing abnormal.

III.—In the same journal as the above, a fatal case of poisoning by the *leaves* of COLCHICUM is mentioned.

IV.—Blumhardt[2] relates a case of poisoning caused by an infusion of a large tablespoonful of the *seeds*.

In three quarters of an hour the man was seized with griping, and then profuse diarrhœa and vomiting. Next morning, twelve hours after the poison was taken, the physician found him still affected with vomiting and purging, but not with pain. He seemed indeed to suffer so little, and to improve so much under the use of emollients, that he was thought to be fairly recovering; but next day the pulse was almost imperceptible; the countenance and extremities were cold; the voice hoarse; the breathing hurried; the eyes sunk; pupils dilated; the epigastrium tender; and the forehead affected with pain. He died at twelve the same day.

[1] Newbrandt—Medicinisches Correspondenz-Blatt.—The Chemist, vol. i., 1840.

[2] Blumhardt—Repertorium für die Pharma

V.—M. Ollivier[1] met with two cases of death within twenty-four hours, in consequence of a tincture being taken, which contained the active part of forty-eight grains of the dry *bulb*; and a third case of death in three days, caused by three doses of a watery decoction, made each time with forty-six grains of the bruised *bulb* collected in *July*. Severe purging and prostration followed each dose. There were no symptoms of any affection of the brain.

VI.—A case is mentioned by Chevallier[2] of fatal poisoning from the intentional administration of a vinous infusion of the dry *bulb*, in which death took place in three days, preceded by violent burning in the intestines, great thirst, frequent vomiting of mucus, and intense suffering. The dissection revealed no morbid appearance at all.

VII.—Three American soldiers, who drank by mistake a large quantity of colchicum wine, prepared from the *bulb*, died with symptoms of burning pain, urgent thirst, and frequent vomiting of mucus. One of them, who took eighteen ounces, and died in two days, presented the leading symptoms of malignant cholera,—viz., frequent vomiting; copious rice-water stools; cramps of the abdominal muscles, and flexion of the extremities; coldness of the skin, tongue, and breath; blueness of the nails; dull sunken eyes, contracted pupils, and collapse of the features. The two others had at first similar symptoms, which passed into those of chronic dysentery, and proved fatal in a few weeks.[3]

VIII.—M. Caffe[4] relates the case of a young lady, who, in order to destroy herself, took five ounces of the wine of colchicum. She was soon seized with acute pain in the stomach, then with frequent vomiting; general coldness and paleness; a sense of tightness of the chest, and oppression of breathing; a slow thready pulse, and extreme prostration; and subsequently, with severe and constant cramps in the soles of the feet. In eleven hours she had less frequent efforts to vomit, but was excessively exhausted. In twenty hours the pulse was imperceptible, and in two hours more she died.

There was no suppression of urine; no purging; no diminution of sensibility; no delirium; no convulsions; no change in the state of her pupils.

About a twelvemonth afterwards, her sister destroyed herself by taking the same preparation, of which she took the same quantity, and she died with exactly the same symptoms in twenty-eight hours.[5]

On examination, in neither of these cases was there any inflammation detected.

IX.—Mr Taylor[6] records the case of a gentleman, who, in November 1839, swallowed, by mistake, one ounce and a half of wine of colchicum. He was immediately seized with severe pain in the abdomen, other symptoms of irritation came on, and he died in seven hours. No post-mortem examination was required by the coroner!

In another instance, in which an ounce was taken, death occurred in thirty-nine hours.[7]

Mr Taylor also mentions an interesting case of poisoning by the medicinal administration of colchicum, communicated to him by Mr Mann. Three and a half drachms of the wine of colchicum were taken in divided doses, and

[1] Ollivier—Journal de Chimie Médicale, 1839.

[2] Chevallier—Journal de Chimie Médicale, viii. Second Series. 1832.

[3] Repertorium für die Pharmacie, lxxi.

[4] Caffe—Annales d'Hygiène Publique, xvi.

[5] Annales d'Hygiène Publique, xii.

[6] Taylor's Medical Jurisprudence, p. 268.

[7] Schneider's Annalen, i., 232.

caused death on the fourth day. There was no inflammation of the mucous membrane, but simply extravasation of blood into the mucous follicles.[1]

X.—One ounce and a half of the vinous tincture of colchicum was by mistake givenone evening to a feeble man, æt. 56, labouring under chronic rheumatism. No complaint was offered for at least one hour after, but then retching came on, with acute pains referred to the stomach, to which vomiting and purging soon supervened. This state continued the whole succeeding night, and a great part of the day following, when the alvine evacuations ceased; but the most distressing nausea continued, with frequent retching. The stools were in the course of the night often involuntary, but not bloody. Excessive thirst came on the day after the accident, and continued till death, with severe pains of the stomach and bowels, which occasioned fomentations to be employed. In the evening the patient seemed nearly exhausted, delirium appeared, the pulse was scarcely perceptible. He lived, however, through the second night, but died the following morning. On dissection, there was no appearance of inflammation of the bowels, but redness of the stomach.[2]

XI.—M. Leroy des Barres[3] has reported to the Academy of Medicine the following case of poisoning by COLCHICUM:—

A female, æt. 57, suffered from pain in the epigastrium. Her medical attendant prescribed for her thirty grammes (an ounce) of tincture of colchicum, of which she was to take a teaspoonful night and morning in a simple julep of taraxacum. Before commencing this treatment, she was directed to take a purgative draught, composed of syrup of buckthorn and sulphate of soda. By some unfortunate mistake, the whole of the colchicum was swallowed instead of this draught. In five minutes she felt severe pain in the stomach and bowels, and there was great anxiety. In this state she was seen by M. des Barres. Her face was pale, the features contracted, and the eyes sunk. The pain in the abdomen was very severe. She vomited once a glairy mucus-like matter, and this was followed by several liquid dark-coloured stools, accompanied by violent colic. The patient complained of a sense of suffocation and strangulation. The pulse was weak, and only fifty in the minute. The extremities were cold, vision was not affected, and the intellect was clear. Small doses of tartar-emetic were administered. These brought away a yellowish-coloured fluid, having a spirituous odour, like that of the tincture of colchicum. Vomiting was promoted by draughts of warm water, and the patient then took the iodureted water recommended by M. Bouchardât; this appeared to relieve the cramps in the stomach and the colic pains. In about an hour the dose of iodureted water was repeated. In two hours the patient still continued to suffer from incessant vomiting, with cramps in the muscles of the legs and arms. The extremities were cold, and the lips and arms had a livid hue. Frictions were employed, stimulating poultices were applied to the abdomen, and sinapisms to the feet. The patient still continued to take occasional doses of the iodureted water. Thirteen hours after the poison had been swallowed, the woman was suffering from great prostration of strength, vomiting, diarrhœa, cramps in the limbs, twitchings of the tendons, great agitation, and severe pain in the abdomen; the pulse was 65; only a few drops of urine had been passed. On the following day the symptoms continued, the pulse having risen to 90. The heat of the body was more equally distributed, the tongue was dry, thirst intense, and there was entire suppression of urine. The cramps and convulsive motions of the limbs, with the feeling of strangulation, had disappeared. Leeches were

[1] Taylor—Op. cit.
[2] Edin. Med. and Surg. Journ., vol. xiv.
[3] Leroy des Barres—Gazette Médicale de Paris, 1848.—Journal de Médecine de Bourdeaux, 1848.—Monthly Journal of Medical Science, 1849.—London Medical Gazette, March 1849.

applied to the abdomen, and emollient medicines prescribed. In the afternoon the patient had considerably improved, and urine was freely secreted. On the fifth day the fever had disappeared, but there was still some diarrhœa. In the course of a week or ten days the patient had entirely recovered.

In this case the employment of M. Bouchardât's antidote seems to have been of no use whatever.

XII.—Dr Bleifus[1] has related the following case of poisoning by the *leaves* of COLCHICUM:—

A man gathered the leaves in the beginning of May, and after cooking them, ate about two ounces for supper. In six hours he was seized with violent colic, vomiting, and purging. In fifteen hours, when his physician first saw him, the countenance was ghastly, as in malignant cholera, the pupils dilated, and, scarcely contractile, but the mind entire. He complained of rheumatic pains in the neck, and burning pain in the pit of the stomach. He had frequent vomiting and purging, spasms of the abdominal muscles, coldness of the skin, a small, slow, wiry pulse, cramps of the fingers and calves of the legs. Coffee and lemon juice allayed the vomiting, and a temporary amendment ensued; but early on the third morning he became worse, and soon after the narrator of the case found him dying.

XIII.—The *flowers* are not less poisonous than the leaves, bulbs, and seeds. A case is mentioned in "Geiger's Journal," of poisoning with a decoction of some handfuls of the flowers, where death occurred in twenty four hours, under incessant colic, vomiting, and purging. In this case the stomach and duodenum only were inflamed.[2]

Another case is mentioned by Garibel, of a servant who died from the effects of poisoning by the *flowers*, which were given to cure intermittent fever.[3]

XIV.—The following case of poisoning by the *bulb* of COLCHICUM, taken for the purpose of procuring abortion, is recorded by Mr Dillon.[4]

Susan Laing, was about 30 years of age, and of good health and constitution. She was about two months gone in pregnancy with a bastard child, and having read in a newspaper that a woman had caused abortion by taking meadow saffron, she determined on getting rid of her burthen by a similar measure. She accordingly procured some, and having made an infusion, took it on an empty stomach, early on the morning of the 10th March 1827. Mr Dillon was called to her about four o'clock in the afternoon of the 11th, and on inquiry learnt that she had miscarried the preceding evening. He found her in a very hopeless state, her extremities were quite cold, and the whole of her body, particularly the hands, feet, and face, livid; the expiration was hurried, and the pulse could not be felt at the carotids, and but faintly at the heart. Notwithstanding, the sensorium was undisturbed, and she gave a clear account of what she had done, her motives for so doing, and the effects the poison had on her. She said, that in about half an hour after she had taken it, her stomach became sick, griping came on, and a violent purging, which continued with great severity. She had had no medical assistance, and was so tormented with pain and purging, that she had no sleep during the night. Mr Dillon administered large draughts of brandy and spices, but to no effect, as she died two hours after.

The body was opened next day, and all the viscera were found perfectly sound, with the exception, that the mucous membrane of the stomach and bowels were dreadfully inflamed throughout.

[1] Bleifus—Repertorium für die Pharmacie, lxix.
[2] Magazin für Pharmacie, xxx.
[3] Garibel—Histoire des Plantes des Environs d'Aix.—Stephenson and Churchill's Medical Botany, vol. ii.
[4] Dillon—Stephenson and Churchill's Medical Botany, vol. ii.—Burnett's Medical Botany, vol. ii.—Beck's Medical Jurisprudence.

XV.—The last case which I shall mention is one recorded by Dr Scilling,[1] in which the effects upon the nervous system are well marked, and *precede* the symptoms characteristic of irritant poisoning. In this case *diuresis* was present from the commencement till the time of death.

A boy, six years old, who, on the 27th June 1836, had eaten of the seeds and leaves of COLCHICUM, was attacked the same night with convulsions, which soon assumed the appearance of opisthotonos. He slept for some time, but soon after another similar attack ensued, and after this had ceased, spontaneous vomiting set in. Tartrate of antimony, ipecacuan, and copper were administered. The patient lay upon his back, and rolled about his head. He passed his urine involuntarily, and his pulse became thready and tremulous. On the 28th and 29th he seemed somewhat recovered. *3d July.*—The elbow and knee joints of the left side swollen, hot, and painful; hemiplegia of right side; perfect loss of hearing; grinding of the teeth and gastricismus. *4th July.*—The right side perfectly paralytic; abatement of the other symptoms. *5th July.*—Convulsion of the whole of the left side; pulse 180. *6th July.*—The patient ate something for the first time; continual diuresis. *18th July.*—Every quarter of an hour a quantity of urine is passed; pulse 180-185, smaller and harder. *24th July.*—Convulsions and loss of feeling on left side; the pulse thready; the breathing has for some days been intermittent. On the night of the *8th August*, the convulsions disappeared; the pulse almost imperceptible. *14th August.*—Violent convulsions, in which, however, the paralytic side did not partake. At three in the morning the patient died. A dissection was not allowed. The diuresis in this case, which began within twenty-four hours after taking the poison, and only ceased at death, is well worthy of observation.

Many other cases of poisoning by COLCHICUM are on record. Too much space, however, has already been occupied in the enumeration of several, the symptoms of which fully establish the fact previously stated, that in general its action is due more to *irritant* than *narcotic* properties; and that when the symptoms of both occur, the *latter* are generally subsequent to the *former*, the only case on record of an action on the nervous system preceding the irritant effects being the one which I have last adduced.

Treatment of Cases of Poisoning.—M. Bouchardât[2] has recommended the use of iodurettted water in such cases. It is a weak preparation of the *Liquor Iodidi Potassii compositus* of the London Pharmacopœia, and is prepared by dissolving six grains of iodide of potassium and three grains of iodine in a pint of water.[3]

Not having been able to procure M. Bouchardât's original paper on the subject, I am ignorant of the principle on which he recommends its use. In the eleventh case of poisoning which I have recorded, and in which the iodurettted water was employed, it seems to have been of no use whatever.

The proper treatment in cases of poisoning by COLCHICUM consists in promoting evacuations upwards and downwards to expel the remains of the poison, and then uniting large opiates with

[1] Scilling—Medizin. Annalen.—Monthly Journal of Medical Science, 1842.
[2] Bouchardât—Gazette Médicale de Paris, Janvier 1837.
[3] London Medical Gazette, 13th April 1849—*Note.*

counter-irritation of the abdomen, or the application of leeches. (Christison.)

THERAPEUTIC ACTION OF COLCHICUM.

I.—As a *Diuretic.* In 1763, Baron Stoërk,[1] of Vienna, first introduced COLCHICUM as a diuretic; and in a book which he published shortly afterwards, numerous cases cured by the use of COLCHICUM were recorded by him.

He relates two cases of *Dropsy* succeeding to *Scarlatina*, which were completely cured in fourteen days; and a case of *Asthma* and *Ascites* in an old man, both of which diseases were entirely removed in a week. The preparation which he used was the *Oxymel Colchici;* and he directs a drachm to be taken for a dose, this quantity being gradually increased to two ounces daily.

He briefly states the physiological action of COLCHICUM, according to his own observations, in the following sentence:—"It dissolves phlegm, and increases expectoration and urine."

It were needless to occupy space by enumerating more of the cases which were published by Stoërk; but having, in justice to him as the original introducer of COLCHICUM, noticed some of them, I may state, that although considered a great physician, the Baron cannot be regarded as a correct reporter of his own cases; for his contemporary Haen[2] says, that "out of thirty-six cases of cancer reported by Stoërk to have been cured by the use of hemlock, it was found on inquiry, that thirty of them had died, and that the remaining six still laboured under the disease."

Thus, although we can place little reliance upon Stoërk's cases, there can be no doubt, from the subsequent confirmation of experience, that there was good foundation for much that he stated. Notwithstanding the exaggerations imputed to him, Stoërk has at all events the merit of bringing into notice many medicines which have since found places in our pharmacopœias, and which, in many instances, have proved valuable additions to the Materia Medica.

M. Planchon[3] mentions several cases occurring in his practice, in which asthma, hydrothorax, ascites, and anasarca were completely removed by the use of COLCHICUM.

At the present time COLCHICUM, I believe, is little employed as a *diuretic*, as there is hardly any mention of it made in the works of authors of the present day.

Dr Mason Good[4] says that it is useful in dropsy, and that it

[1] Stoërk, de Colchico, 1763.
[2] Haen—Epistola de Cicuta.
[3] Planchon—Journal de Médecine, 1765.
[4] Good—Study of Medicine.

ought to rank next to *Squill* as a diuretic in that disease. He exaggerates the acrid properties of the drug, however, in the following passage:—"Even while cutting the roots, the acrid vapour that escapes irritates the nostrils and fauces; and the substance held in the fingers, or applied to the tip of the tongue, so completely exhausts the sensorial power, that a numbness or torpitude is produced in either organ, and continues for a long time afterwards." It is needless to say that there is not the least foundation for such averments.

Dr Craigie,[1] one of the few who mention its use in dropsies, states that it is uncertain in its effects.

That it has diuretic properties, however, is undoubted; and its power of causing increased secretion of urea seems to point it out as a proper stimulant to the kidneys, in cases of suppression of urine, when a fatal result from accumulation of urea in the blood is always to be apprehended.

In the case of a boy, who had almost complete suppression of urine for three days, the whole quantity passed in that time not exceeding an ounce in all, and where digitalis, spiritus etheris nitrici, acetate of potass, and diluents, had produced no effect, Dr Douglas Maclagan used the *Acetum Colchici* with complete success.

"In the acute states of dropsy it is best given with mercurials in powder; but in asthenic cases, it is most advantageously combined with the warmer diuretics, with tonic infusions, with preparations containing camphor or ammonia, or with large doses of the alkaline subcarbonates, particularly in the gouty or rheumatic diathesis." (Copland.)[2]

In *Dropsy* succeeding to *Scarlatina* I have frequently found Colchicum of much service, especially in cases where the urine is much suppressed, and where comatose symptoms are present. The accession of *Coma* may easily be ascribed to the accumulation of *Urea* in the blood; and the power which it has been shown that Colchicum possesses of replacing the *Urea* in natural, and often superabundant, amount in the urine, seems to point it out as a useful remedy in this and other diseases in which *Suppression of Urine* and *Coma* co-exist. In a case of scarlatina which I attended along with my friend Dr A. Christison, now of the H.E.I.C. Service, and where the urine was totally suppressed, and the symptoms of coma were present, the *Acetic Extract of Colchicum* was used with complete success. Diluents and ordinary diuretics were freely employed when the case was first seen, with the effect of causing a slight secretion of urine of low specific gravity. Having suggested that Colchicum might

[1] Craigie—Practice of Physic.
[2] Copland—Dictionary of Practica Medicine—*Dropsy*.

be found of some service, and being anxious to observe its effects, both as a diuretic and as an eliminator of urea, I examined the urine before its exhibition. The results were as follows:—

Urine examined on 10th April.

Total solids,	35·795
Urea,	2·427
Uric acid,	a trace.
Inorganic salts,	13·510
Organic matter and water,	969·573
Albumen,	14·490
Total,	1000·000

The *Acetic Extract of Colchicum* was ordered on the 11th April, and the other medicines discontinued. On the 12th, the comatose symptoms were considerably abated; urine, of a normal density, was passed in tolerable quantity, and was examined again on the 13th, two days after the exhibition of Colchicum. It contained:—

Total solids,	30·659
Urea,	7·500
Uric acid,	0·480
Inorganic salts,	8·718
Organic matter and water,	975·359
Albumen,	7·943
Total,	1000·000

On the evening of the 14th, the comatose symptoms disappeared; urine, of normal density, was passed in proper quantity; the dropsical effusion and anasarca completely gone. On the 15th, considerable diarrhœa had set in; the Colchicum was stopped, and the urine again examined. It contained:—

Total solids,	27·972
Urea,	13·573
Uric acid,	0·814
Inorganic salts,	7·431
Organic matter and water,	978·182
Total,	1000·000

The analyses of the urine in this case will show the powerful influence which Colchicum possesses in altering the renal secretion, and of how much service, as a remedy, it may be in cases of threatened poisoning by *Urea* in the blood. I believe, in all cases where *Albumen* and *Urea* appear to be vicarious, and where *Coma* supervenes, evidently from the accumulation of the latter principle in the blood, that Colchicum will prove to others of as great service as it has already done in the small experience I have had of it.

Two other cases of a similar nature, in which the urine was examined at regular intervals, were treated in the same manner as that which I have related, with precisely the same result.

II.—As a *Sedative*. In 1820, Mr Haden[1] published a small pamphlet upon COLCHICUM, and especially takes notice of its sedative effects on inflammation.

Mr Haden states, "that in pure inflammation, if it be given so as to produce full purgative effects, COLCHICUM will be found to bring the pulse nearly to its natural state, from being either quick, or hard, or slow and full; but this action may also be produced before purging has taken place. Fevers and inflammations so removed never require the use of tonics during convalescence: the patients, indeed, generally appear to be as well as though they had never been the subjects of disease; and although it sometimes happens that a recurrence of symptoms takes place, it is in a much milder degree, and the new disorder is always immediately removed in the course of a few hours by a very little of the same treatment."

Mr Haden found that the *Tincture of Colchicum* often did not produce purgative effects until forty-eight hours had elapsed, and then it was frequently very violent in its action; with the combination of a saline aperient, however, he found that the beneficial sedative effects of Colchicum followed more quickly and were equally decided as when Colchicum was given alone. Having repeatedly found that very violent effects were produced by the *tincture*, he chiefly made use of the *powder* of the *bulbs*, which he extensively employed in a variety of diseases, the principal of which were Acute Rheumatism, Inflammatory Fevers, Inflammation of Lungs, Pleura, and Bronchi, and in Puerperal Fever. He gives numerous cases in which Colchicum was administered in the form of powder, combined generally with a saline aperient.

The following is a case of *Acute Rheumatism*:—

"A stout labourer was suddenly seized with rigors, after working for several hours in the river Derwent; violent fever followed; and the next day he was confined to bed, being incapable of moving his limbs in the slightest degree. Five grains of powdered Colchicum, with a scruple of Sulphate of Potass, were given four times a-day; and two days afterwards he was found walking in the streets, and was soon quite well. Five doses of the medicine were taken."

The next two cases are of *Lumbago*, treated in the same manner:—

"Two patients, about the same age, were unable to rise from their beds, on account of Lumbago. One of them, a gentleman, in addition to taking Colchicum, drank plentifully of warm fluids, took the warm bath twice a-day, and kept himself covered by the bed-clothes; he was quite free from pain in twenty-four hours, and was up and quite well in four days;—whilst the other, a lady, although she remained in bed, and the disease left her perfectly well in a week, yet did not use the warm bath, nor any other of the means used by the other patient. In neither of these cases were tonics required."

[1] Haden on Colchicum. 1820.

These last cases do not prove so much in favour of COLCHICUM as in favour of the warm bath and diaphoretic treatment. In the former of the two cases, where these were used along with Colchicum, the relief was rapid, but by no means unusually so; in the latter, where no warm bath was used, the disease lasted longer under the use of Colchicum alone.

It is remarkable that Mr Haden should have been so successful in his use of the powder of the bulb,—a preparation seldom used by other practitioners; but he seems to have employed it, not as being more active, but as being less irregular, than the tincture in its action. His statements, if confirmed, would lead us to esteem it as a remedy of remarkable antiphlogistic powers; but few practitioners are inclined to believe that it will ever supersede the use of the lancet, and other safer antiphlogistic remedies, in cases of pure inflammation.

He gives one case of *Pneumonia*, in which Colchicum was used with perfect success, instead of blood-letting; but nothing appears in his narrative to show that it has any advantage over tartrate of antimony, which is more manageable, and less likely to produce unpleasant effects.

Mr Haden has, towards the end of his work, expressed his opinion of the action of COLCHICUM in the following terms:—"The sensible effects of Colchicum would appear to be, to control the action of the heart and arteries, and indeed often to reduce that action below that of the standard of health. This effect is often produced long before its other sensible effects are apparent; but when continued long enough, and generally before its remedial virtues are decidedly obtained, purging takes place. Sickness and vomiting accompany the purging in some instances, whilst in others the secretion from the kidneys or from the skin is increased, sometimes without the former symptoms being perceived."

We thus find from Mr Haden's observations, that he considered the action of Colchicum essentially sedative, whilst the purgative and diuretic effects were only incidental accompaniments. This statement of the powers of Colchicum as a sedative is certainly over-rated, and its influence in this respect has not been realised to such an extent in the present day.

Dr Copland[1] says, that the kinds of inflammation in which Colchicum may be of essential service are those cases in which a torpid and obstructed state of the liver is present, and then Colchicum, combined with deobstruent purgatives, is of much use. "In cases attended by very acute pain, or by the effusion of fluids from the inflamed part, it will also be of service, when judiciously combined with other means; but its action should be carefully watched, as in some constitutions it produces most depressing and even injurious effects."

[1] Copland—Dictionary of Practical Medicine: *Inflammation—Heart.*

"The sedative influence of Colchicum on the circulation is also shown in cases of preternatural action of the heart, whether from functional or organic causes."

Dr Lewins[1] says,—"In all inflammatory affections of the chest, and perhaps of the brain, or its investing membranes, I am convinced that bleeding may frequently be, to a certain extent, superseded by the use of Colchicum. In many diseases of the heart and large arteries it is a most valuable medicine."

This, however, I believe to be an exaggeration of the extent to which Colchicum is applicable in inflammatory diseases.

In *Fever*. The same author has recorded six cases of Continued Fever, in which COLCHICUM was used with complete success. In all of these considerable physiological action was manifested, such as vomiting, purging, and reduction of the pulse. These, however, are, generally speaking, very unsafe results in fever.

Dr Tait[2] has noticed thirty-five cases of *Scarlatina*, successfully treated with COLCHICUM. In a few cases vomiting was induced, and a considerable quantity of bile evacuated. In most cases diarrhœa followed its use, but the most apparent phenomena were great reduction of the pulse in frequency and force, and subsidence of the palpitation of the heart, which in young subjects was often apparent to the eye. Dr Tait's cases were all of the purely inflammatory type.

Dr Lewins,[3] jun., has seen cases of *Scarlatina* effectually cured by the administration of COLCHICUM. He says,—"In every instance where Colchicum was employed, the malady very speedily proceeded to a very favourable termination; while other cases, apparently similar in their character, in which this medicine was not administered, proved by no means so satisfactory,—several having terminated fatally, and the sequelæ of others being exceedingly troublesome."

III.—As a *Diaphoretic*. COLCHICUM is now never employed merely to produce diaphoresis, although there can be little doubt that its influence in this respect is by no means inconsiderable, sweating being very often mentioned as having supervened in cases which have been treated by COLCHICUM.

When COLCHICUM is used in combination with OPIUM, sweating is frequently produced, and is often copious. The effect here is not to be ascribed solely to the Colchicum, nor is it always proportionate to the dose of Opium, but is probably due partially to both. Probably its good effects in some cases of *Rheumatism* are partially due to this diaphoretic action, and the combination of Colchicum and Opium, appears in many respects to resemble Dover's Powder in its physiological and therapeutic action.

[1] Lewins—Edinburgh Medical and Surgical Journal, xlvii.
[2] Tait—Lancet, 1837-38, vol. i.
[3] R. Lewins—Edin. Med. and Surg. Journal, vol. lvi.

The two diseases in which COLCHICUM is now more generally employed, are GOUT and RHEUMATISM. Its effects in these two diseases have not been decidedly referred to any one of its physiological actions, but have often been considered as being of a specific character. Instead, therefore, of attempting to refer it to any of the above divisions, it will be better to give its action in Gout and Rheumatism a separate consideration.

IV.—In *Gout* and *Rheumatism*. If we are to believe that the *Eau médicinale* has for its basis some preparation of COLCHICUM, we must assign to Dr Jones, of London, the merit of having first employed the drug in this country for the cure of Gout and Rheumatism. On the other hand, if we are to believe that the Eau médicinale does not contain COLCHICUM, to Sir Everard Home this merit belongs, he having been the first to use COLCHICUM in this country under its own proper designation. As I believe, however, that the former of these opinions is the correct one, it demands a notice of the introduction of this nostrum into Britain, and its subsequent use there.

In 1810, Dr Edwin Jones,[1] of London, first introduced the *Eau médicinale* into this country from France; and in a small pamphlet which he published in that year, he relates several cases in which he used the new drug with great benefit. He states the following to be its mode of action:—"In four or five hours after taking the remedy, the patient begins, however severe the paroxysm may be, to experience a diminution of pain; he generally falls into a quiet sleep, and awakes in the morning nearly or quite free from suffering, and often begins to enjoy some returning use of the affected limb; he then commonly feels considerable nausea, sometimes accompanied by vomiting, and generally followed by bilious stools; in the meantime the paroxysm diminishes, and on the third, or even the second day, nothing remains but a slight swelling or stiffness of the parts, which soon goes off, leaving the patient in his usual state of health. These are the common effects," says Dr Jones, "but there are others no less singular and deserving attention. Together with a diminution of pain, there is an abatement of fever and irritation; the pulse is often reduced twenty strokes in a minute, and in many instances considerably more; at the same time a moderate diaphoresis usually takes place. It very frequently also acts as a powerful diuretic, and its operation in this way sometimes lasts for several days."

Though the paroxysm was removed in the majority of his cases in the above manner, Dr Jones observed that the time in which this was effected varied under different circumstances. Sometimes a patient gets rid of a sharp fit the next day; in others, several days may be necessary. "In its action on the bowels, the Eau médi-

[1] Jones on Gout. 1810.

cinale is extremely capricious; it usually operates in the way above-described, sometimes it produces no evacuation at all; in others it proves powerfully emetic and cathartic; and in a few cases has acted with considerable violence." These variations in action did not appear to depend on the relative strength of the patients, as Dr Jones observed that several weak and delicate persons took full doses, without experiencing any disturbance, whilst in some robust habits it acted powerfully both by vomiting and by stool, although only half a dose was taken. This appears rather to be referable to some peculiarity of constitution. It was equally uncertain in the time required to produce these effects. It generally began to operate in eight or ten hours after being taken, sometimes not till twenty-four or forty-eight hours had elapsed, and in some rare cases not till after three days. Dr Jones concludes his remarks on the action of this medicine by stating, that "for the most part the Eau médicinale, even where it has been more violent than was expected, has not been followed by any ill consequences."

In 1815, Mr Want[1] published his conviction of the identity of the basis of the *Eau médicinale* and COLCHICUM. He successively used the menstrua of proof-spirit, wine, and water, and succeeded in forming a preparation with Spanish wine, differing in no respect from the Eau médicinale. I believe Mr Want to have been the first person to draw attention to the probable identity of these two medicines.

The publication of this paper by Mr Want paved the way for a controversy between him and Dr Sutton,[2] the latter of whom very much doubted that Mr Want had discovered the plant whose active properties were the basis of the French nostrum, and he endeavoured to show that Mr Want had only reported cases cured by a medicine well known at the time, and that the power of this remedy merely consisted in its cathartic properties, and stated that many purgatives then known would have the same effect; but Mr Want disproved this in a subsequent paper, in which he published a case of Gout, cured by Colchicum, in which no purging had been induced.

In 1816, Sir Everard Home[3] and Sir Charles Scudamore[4] both wrote upon Gout, the former advocating the identity of the Eau médicinale with Colchicum, the latter denying it. Sir E. Home quotes himself as a case in which the Eau médicinale had acted powerfully and effectually in restraining the gouty paroxysm.

Under the influence of a violent fit of Gout, Sir Everard took sixty drops of the *Eau médicinale*. The pain was intense, and he felt chill; in two hours he became rather hot and thirsty: in three

[1] Want—Medical and Physical Journal, vol. xxxii.
[2] Sutton on Gout.
[3] Home—Philosophical Transactions, 1816-17, Part 2.
[4] Scudamore on Gout and Gravel. 1816.

hours the pain was considerably diminished; in seven hours he had a confirmed motion of the bowels; nausea came on, and the pulse, naturally at 80, lowered to 60, and intermitted. In ten hours the nausea had gone off, he remained languid, his pulse was 70; he had some appetite. The following morning the pulse was 80, and he was quite well.

Sixty drops of the *Vinum Colchici* were given to a man labouring under Gout, and whose paroxysms were generally of three or four weeks' duration. His age was sixty. When the medicine was exhibited, his pulse was 115. In half an hour he had slight nausea, which soon went off. In five hours a profuse perspiration came on, and the pain of the Gout entirely ceased, leaving a soreness in the part affected. In twelve hours his bowels were gently moved, his pulse 105, and irregular. In fourteen hours his bowels were again acted on. In nineteen hours his pulse was 92, and natural. In forty-eight hours he was quite well, and continued so.

From his observations on the use of Colchicum in Gout, Sir Everard Home concluded, that the effects of the remedy always were to reduce the pulse ten or twenty beats in a minute, and that this effect generally took place about twelve hours after the administration of the medicine.

Sir Charles Scudamore denies that the basis of the *Eau médicinale* and COLCHICUM are identical, and he gives his opinion, that the Eau médicinale is a dangerous remedy, and one which has been much too highly praised by Mr Want; he, however, does not deny that it has some influence over Gout, as he states that on the first trial of the medicine the paroxysm is removed, and that as if by a charm, and that relief is often obtained without any sensible effects upon the stomach or excreting organs, and that the curative power of the remedy lessens gradually on repetition, and with many persons becomes entirely lost. The capricious action of this medicine seems to him to be the chief objection to its use. He states a case in which the patient had taken several bottles in a few weeks, without any effect being produced; and another, where the contents of a single bottle so paralysed the stomach, that for many days it was scarcely sensible to the strongest stimulants; the patient was recovered with much difficulty, and remained for a long time in a state of serious debility. He says,—"When the Eau médicinale does not immediately debilitate by the violence of its effects, it often leaves behind an impaired condition of the nervous system, as, that the head is affected with frequent giddiness; the stomach with weakened digestive power, and frequent sensations of sinking and vacuity; the limbs, and especially the parts affected in the paroxysm, suffer for many weeks with trembling, numbness, and coldness, and very commonly with tedious œdema; these symptoms appearing variously in different individuals. It tends also to render the bowels inactive, to diminish the alimentary secretions, and ma-

terially to weaken the functions of the liver. In the general character of the medicine, it may with truth be said, that sooner or later, in proportion as it is freely employed, it leads to a broken state of health."

Mr Ring[1] relates a case of poisoning by the *Eau médicinale*, in a person who had been accustomed to use it for the relief of Gout. Half a bottle was taken; it operated violently as an emetic, cathartic, and sudorific. Next day the Gout had disappeared, but a violent pain had seized the patient in the pit of the stomach; this increased during the night, when it became excessive, alternately affecting the stomach and bowels. On the second day the pain gradually abated; but in the evening bilious vomiting came on; and on the third day he died.

These effects observed in the *Eau médicinale* show it to have been a preparation very analogous to COLCHICUM in its general effects; but its uncertainty and violence correspond more with the accounts which are generally given of the action of *Veratrum Album*, and it is not improbable that it may have contained the active part of both these drugs.

In modern practice, however, the *Eau médicinale* has been completely superseded by the ordinary officinal preparations of Colchicum, and the little bottles in which it was sold are rarely met with, except as preparations in a museum.

There can be no doubt, from the results of modern experience, of the value of COLCHICUM as a remedy in cases of Gout; but, as I have had no opportunity of treating cases of that disease, it cannot be expected that I should offer remarks on the practical employment of any medicine for its cure. I must therefore rest satisfied with briefly reporting the experience of practical men.

1. In *Gout*. It appears probable, that COLCHICUM, in the form of *Hermodactyl*, was employed even in ancient times in the cure of Gout—now it is in very general use.

Its action is very frequently said to be specific, but, as has been previously remarked, this expression seems to imply nothing more than that its effects are energetic and marked; for the term "specific," as applied to a medicine, in strict propriety means one which cures a disease, without producing any distinct or obvious physiological action, in virtue of which it may be supposed to lead to the cure of the disorder; but it appears, from the concurrent testimony of many practical men, that Colchicum seldom cures the paroxysm of Gout, without producing some distinct physiological action. To use the words of Dr Barlow,[2]—"A full dose of this medicine purges copiously, allays pain, and lowers the pulse; its operation seems to

[1] Ring—Edin. Med. and Surg. Journal, vol. vii.
[2] Barlow—Cyclopædia of Practical Medicine—*Gout*.

combine the several advantages of blood-letting, purging, and the production of sedative action."

Dr Christison[1] has stated, that COLCHICUM seldom acts therapeutically before producing a slight degree of that physiological action indicated by diarrhœa, colic pains, and frontal headache, which, in a higher degree, constitutes it a poison.

Dr Gairdner,[2] however, states, that he believes the action of COLCHICUM to be *specific*, and denies the necessity of its producing any physiological effects before relief is obtained. To use his own words,—"COLCHICUM never more effectually relieves the patient than when it acts silently and peacefully, without producing any evacuation whatever, or in any way disturbing the patient's comfort and ease."

The rarity of the occurrence of Gout here has prevented me from deciding, by means of statistics or otherwise, whether or not it is necessary that some physiological action should accompany the therapeutic effect of this drug. I have found means of doing so, however, with regard to Rheumatism, to the results of which inquiry I shall immediately refer.

Dr Robertson[3] states, that while COLCHICUM acts freely on the bowels, and especially on the duodenum, it possesses a specific action on the white fibrous tissues; it is to this property, he says, that COLCHICUM owes its power over Gout, Rheumatism, and certain affections of the heart, and every disease involving febrile or inflammatory excitements, quite independently of its other effects on the system.

Dr Todd[4] states with regard to Gout, what Dr M'Leod[5] does with regard to Rheumatism, that whenever nausea or purging are induced during the administration of Colchicum in those diseases, the dose of the medicine should be diminished, or altogether abandoned.

It has frequently been objected to the use of COLCHICUM, that it confirms the Gouty constitution in the systems of those who have used it. As Gout generally occurs in persons who have the disease confirmed in their constitutions, either by having inherited it, or by having acquired it by full living, &c., Colchicum has got the credit of confirming the Gout in their systems, when that is in reality due to the inherited or acquired Gouty habit.

With regard to this, Dr Holland[6] writes,—"A suspicion has existed that, though capable of relieving the present paroxysm, Colchicum renders the attacks of the disorder more frequent. On my experience, I believe this opinion to be justified only where the

[1] Christison's Dispensatory.
[2] Gairdner on Gout. 1849.
[3] Robertson on Gout.
[4] Todd on Gout and Rheumatism.
[5] M'Leod on Rheumatism.
[6] Holland's Medical Notes and Reflections. 1840.

medicine has been used imperfectly, or without other precautions, which are more or less essential to its success."

On the other hand, Dr Copland[1] says,—"COLCHICUM, when used with the view of preventing or suddenly curing the paroxysm, and without reference to the removal of the morbid condition of which it is the external manifestation, is liable to many objections. The consequences of having frequent recourse to it vary in different constitutions, and with the habits and mode of living of the patient, but they commonly are a much more frequent return of the fit, or of the symptoms indicating its approach; impaired nervous power; debility of the digestive organs; torpor and irregularity of the biliary functions, and of the bowels; headaches, and a variety of symptoms referable to the encephalon." Besides this, he has met with instances of Hypochondriasis, Melancholia, Mental Delusions amounting to Insanity, Paralysis, and Angina Pectoris, evidently arising from this.

Were all physicians agreed upon this point, COLCHICUM would require to be considered in a far different light from that in which it is at present. Few men have met with such untoward results in the use of this medicine as Dr Copland, and we should believe, at least hope, that they are considerably exaggerated.

With regard to the application of COLCHICUM for the relief of the morbid conditions of which Gout consists, besides its use for the mere alleviation or prevention of the paroxysm, Dr Robertson[2] states, that "the action of Colchicum must be said to be more decided and greater on the local manifestation of Gout, and the inflammatory character of its paroxysms, than on the constitutional condition on which Gout depends, and of which the localised ailment is only a form and development."

Dr Holland,[3] however, is more favourable to its further application. He says:—"I can scarcely doubt the expediency of carrying the employment of COLCHICUM beyond the mere relief to the local inflammation of the disease. The remedy, with due care, may be made preventive as well as curative of Gout, and, according to my experience, with no less safety to the patient.

"We may reasonably, then, if this view be just, extend to its use as a medicine the remark before made,—that too exclusive attention is given to the external part of the disease, and the value of the remedy, in the constitutional form of the disorder, too little regarded. Larger experience is making a gradual change in this respect; but there is still a tardiness and timidity in its application beyond the mere fit of Gout, which is not warranted by any ascertained risk."

I offer no further remarks here, both on account of the various opinions of high testimony I have adduced, and also from the fact

[1] Copland—op. cit.: Treatment of Gout.
[2] Robertson—op. cit.
[3] Holland—op. cit.

previously stated, that the rare occurrence of Gout in this place has prevented my making observations on the nature and treatment of the disease.

Besides its actions before stated, COLCHICUM appears to act both as a diuretic and diaphoretic. Its principal action appears, however, to be, lowering the pulse and relieving pain, and therefore its chief object is that of a sedative and anodyne.

Bearing in mind, however, the tendency to the formation of *Lithic Acid* in Gouty subjects, and the effect of Colchicum in altering the renal secretion, it seems more than likely that its effects are partly due to changes which it induces in the chemical quality of the blood, and the secretions derived from it.

All the preparations of COLCHICUM have been employed in Gout and Rheumatism, and have been administered in various ways. Dr Watson[1] recommends forty to sixty minims of the *Vinum Colchici* in a saline draught at bed-time, and a half drachm more in a warm black dose the next morning. More commonly, however, the simple tincture of the seeds is employed. This is recommended by Dr Barlow[2] as being more uniform in strength, and more certain in operation.

It may be administered in full doses (*i. e.*, twenty to thirty minims), and repeated at intervals till the pain has abated, or till some of its physiological actions, such as purging or diuresis, manifest themselves.

2. In *Rheumatism*. COLCHICUM has been employed upon much the same principle as in Gout, and from the similarity of the two diseases we would be led to expect much the same results from its use. It does not appear that its curative powers are in general manifested, until it has fairly produced some of its constitutional effects; and when these are fairly shown, as, for example, when it produces some amount of irritation of the bowels, the disease frequently yields with great rapidity. In Dr Watson's words,—"The preparations of COLCHICUM have sometimes, whether venesection has been premised or not, an almost magical effect in quelling the disease. Frequently when most successful (though that is by no means a necessary condition of their success), they exercise some marked influence upon the stomach and bowels. Colchicum is very apt to occasion deadly nausea and vomiting, griping and diarrhœa, and when these consequences ensue from its use, the inflammation of the joints often subsides entirely. At any rate, if the Rheumatism does not give way, when the stomach and bowels become thus affected, you may be certain that to push the Colchicum further would be useless."

In an interesting memoir, Dr E. Monneret[3] gives the details of

[1] Watson's Practice of Physic.
[2] Barlow—op. cit.
[3] Monneret—Archives Générales de Médecine, Mars 1844. London Medical Gazette, vol. xxxiv.

treatment of twenty-five cases of *Articular Rheumatism* treated by COLCHICUM. The greater number of his patients took from one to four drachms of the tincture, in one, two, or four divided doses, in the twenty-four hours. One drachm was the smallest dose of the tincture ever administered, and several of his patients took it for seven, some for ten, and others for thirteen, days. In eight of the patients the diminution, or even total disappearance, of the symptoms of Rheumatism coincided with the exhibition of Colchicum. In these cases, the disease was either of some days' duration, and was scarcely accompanied by febrile symptoms, and then ended in twelve or fourteen days, or it was completely chronic.

In either case, the powerful effects produced by the Colchicum on the bowels sufficed to suspend or expel the disease; the improvement always coincided with the diarrhœa. In most instances, diarrhœa was the prevailing feature, although in some cases retching and vomiting, without any purging, were induced. The discharges were almost always bilious, or evidently mixed with bile. The motions were usually passed with acute suffering, violent colic pains, tenesmus, and scalding of the anus. Vomiting was scarcely induced by a smaller dose of the tincture than from two to four drachms in a draught.

In order to corroborate the idea that COLCHICUM in general produces some physiological effect, before its therapeutical action is manifested, I have prepared the following table of cases of *Rheumatism* treated by Colchicum, in which will be seen the effects of this medicine in seventeen cases.

This must necessarily be more or less imperfect, from the incomplete manner in which the cases have been reported; still it is sufficiently correct to show, that the physiological effects of COLCHICUM are the almost certain accompaniments of its therapeutic action; and it would thus appear that in *Acute Rheumatism*, COLCHICUM produces its good effects, partly by its *evacuant*, and partly by its *sedative* influence.

Beyond this, however, I believe that Colchicum exerts a great influence in the treatment of this disease by the power it has of altering the renal secretion.

In *Acute Rheumatism*, Dr Garrod[1] states, that the blood contains no more *Uric Acid* than in health; this being a very minute quantity. I am inclined to believe, however, that in all cases of *Acute Rheumatism*, both *Urea* and *Uric Acid* are present in the blood in increased quantity.

[1] Garrod—Lond. Med.-Chir. Trans., vol. xxxi.

TABLE OF CASES OF RHEUMATISM TREATED WITH COLCHICUM.

No.	Age.	Sex.	Variety, and parts affected by Rheumatism.	Pulse before treatment.	Previous duration of disease.	Duration of treatment.	Treatment.	Physiological action.	Pulse after treatment.
1	58	M	Articular. Lower extremities.	78	12 Days.[a]	10 Days.	Colchicum Powder.	Diarrhœa, nausea.	Natural
2	40	M	Muscular, and Articular of knees.	60	5 Months.[a]	17 Days.	Colchicum and Dover's Powder.	Griping, nausea.	Natural
3	29	F	Articular. Upper extremities.	—	3 Weeks.[a]	16 Days.	Colchicum Powder.	Diarrhœa. ...	—
4	70	M	Articular.	80	6 Months.[a]	11 Days.	Colch., Morph., Iodid. Potass.	Nausea, diarrhœa.	Natural
5	32	M	Articular.	100	4 Days.[a]	4 Days.	Colchicum, Morphia. ...	...Diarrhœa. ...	65
6	17	F	Articular.	96	14 Days.[a]	2 Months.	Colchicum, Morphia. ...	...Diaphoresis, nausea	80
7	20	F	Articular. Ankles and wrists.	100	8 Days.[a]	4 Days.	Colchicum, Morphia. ...	...Profuse diaphoresis	86
8	22	F	Articular. Knee and wrist.	120	8 Days.[b]	13 Days.	Colchicum, Magnesia.	...Diarrhœa. ...	60
9	—	M	Articular. Wrists. ...	72	21 Days.[b]	12 Days.	Colchicum, Magnesia.	...Diarrhœa. ...	54
10	18	M	Articular. All joints, more or less.	120	35 Days.[b]	17 Days.	Colchicum, Magnesia.	...Diarrhœa. ...	60
11	—	F	Articular.	—	4 Months[c]	14 Days.	Vin. Colchici, V.S. ...	...Diarrhœa. ...	—
12	—	M	Articular.	—	49 Days.[c]	14 Days.	Vin. Colchici, Leeches.	...Diarrhœa. ...	—
13	—	M	Articular.	—	14 Days.[c]	14 Days.	Vin. Colchici, Cupping.	...Diarrhœa. ...	—
14	—	M	Rheumatic Fever. ...	—	6 Days.[c]	3 Days.	Vin. Colchici, Opium.	...Diarrhœa. ...	—
15	28	F	Articular. Elbows, wrists, hand.	—	3 Months.[d]	14 Days.	Vin. and Acet. Extr. Colchici.	Vomiting. ...	—
16	30	M	Articular. All large joints.	—	12 Days.[e]	6 Days.	Tinct. Colchici. ...	...Profuse diaphoresis	—
17	30	M	Articular, Metastatic. Knees, elbows. ...	130	14 Days.[f]	18 Days.	Tinct. Colchici, Morphia.	...Diarrhœa, diaphor.	100

[a] From the Journals of Royal Infirmary, Edinburgh.
[b] Tweedie—London Med. Gaz., vol. viii.
[c] Elliotson—London Med. Gaz., vol. viii.
[d] Seymour—London Med. Gaz., vol. xviii.
[e] Dublin Medical Transactions, 1830.
[f] Personal observation.

In the cases which I am about to relate, and which fell under my own observation, I shall endeavour to prove that such is the case, and to show, from the analysis of blood and urine, which I made both before and after the exhibition of Colchicum, that the remedial agency of this medicine is due, partly at least, to its power of eliminating Urea and Uric Acid from the blood, and increasing their quantity in the urine.

The first case was that of a girl, under Dr Wright's care, in the Royal Infirmary. On the 13th of October a small quantity of blood was subtracted, analysed, and found to contain :—

In 1000 parts of blood, ...	0·507 Urea.
" "	·864 Uric Acid.

The urine was examined at the same time. It contained :—

Total Solids, ...	28·568
Water, ...	971·432
Urea, ...	10·496
Uric acid, ...	·257
Inorganic salts, ...	7·461
Organic matter, ...	10·354
Total, ...	1000·000

Colchicum, in combination with Muriate of Morphia, was then administered.

The urine was again examined on the 18th October, being the fifth day. It was found to contain :—

Total solids, ...	31·459
Water, ...	968·541
Urea, ...	12·312
Uric acid, ...	·421
Inorganic salts, ...	8·231
Organic matter, ...	10·495
Total, ...	1000·000

The urine was again examined on the 22d October, or ninth day. It contained :—

Total solids, ...	35·613
Water, ...	964·387
Urea, ...	13·964
Uric acid, ...	·598
Inorganic salts, ...	9·401
Organic matter, ...	11·630
Total, ...	1000·000

After twelve days constant use of the Colchicum, a small quantity of blood was procured for examination. Now, however, not the slightest trace either of Urea or Uric Acid could be detected in so large a quantity as 3500 grains.

The urine was examined at the same time, and was found to contain :—

Total solids, ...	34·554
Water, ...	965·446
Urea, ...	14·561
Uric acid, ...	·737
Inorganic salts, ...	9·649
Organic matter, ...	9·607
Total, ...	1000·000

The Colchicum being still continued, the urine was again examined on the eighteenth day, and found to contain :—

Total solids, ...	38·128
Water	961·872
Urea, ...	17·635
Uric acid, ...	1·034
Inorganic salts, ...	9·999
Organic matter, ...	9·460
Total, ...	1000·000

The next case to which I shall refer was treated by myself in the New Town Dispensary. I had only an opportunity of examining the blood in this case once,—namely, before Colchicum was taken. It then contained :—

In 1000 parts, ...	1·416 Urea.
"	·691 Uric acid.

Before taking Colchicum, the urine contained :—

Total solids, ...	23·479
Water, ...	976·521
Urea, ...	6·353
Uric acid, ...	·097
Inorganic salts, ...	7·333
Organic matter, ...	9·691
Total, ...	1000·000

The urine was again examined on the fourth, ninth, and thirteenth days respectively, and contained :—

On 4th Day.

Total solids, ...	24·538
Water, ...	975·462
Urea, ...	9·103
Uric acid, ...	·231
Inorganic salts, ...	8·693
Organic matter, ...	6·511
Total, ...	1000·000

On 9th Day.

Total solids,	...	26·322
Water,	973·678	
Urea,	12·961	
Uric acid,	·497	
Inorganic salts,	9·400	
Organic matter,	3·444	
Total,	1000·000	

On 13th Day.

Total solids,	...	27·466
Water,	972·534	
Urea,	16·824	
Uric acid,	·936	
Inorganic salts,	7·203	
Organic matter,	2·503	
Total,	1000·000	

These are the analyses of two very favourable cases, in which it will be seen, that the UREA and URIC ACID are increased in proportion to the time that the medicine is continued. I have selected these two cases from many others, on account of their showing the increase more gradually. In all the cases, however, in which I have analysed the urine, the great increase was distinctly marked, although perhaps not in so regular proportion.

One other case I would wish to mention, in which the *Urea* and *Uric Acid*, although they increased after a few days' use of the medicine, did not continue to do so subsequently in the same proportion.

The analyses were made before taking Colchicum, and on the third, sixth, and tenth days after its exhibition :—

Before taking Colchicum.

Total solids,	...	25·636
Water,	974·364	
Urea,	7·634	
Uric acid,	·129	
Inorganic salts,	8·421	
Organic matter,	9·402	
Total,	1000·000	

On 3d Day.

Total solids,	...	27·479
Water,	972·521	
Urea,	11·156	
Uric acid,	·300	
Inorganic salts,	8·303	
Organic matter,	7·718	
Total,	1000·000	

On 6th Day.

Total solids,	...	27·907
Water,	972·093	
Urea,	15·060	
Uric acid,	·570	
Inorganic salts,	6·500	
Organic matter,	5·177	
Total,	1000·000	

On 10th Day.

Total solids,	...	28·426
Water,	971·574	
Urea,	15·730	
Uric acid,	·582	
Inorganic salts,	7·351	
Organic matter,	4·763	
Total,	1000·000	

These analyses will show to what extent COLCHICUM will alter the renal secretion, by supplying it, when deficient of its normal constituents—*Urea* and *Uric Acid*—from the blood.

I am inclined from all this to believe, that to this property of Colchicum its remedial action is in a great measure to be referred. Further analyses of the blood may, however, be thought necessary, but neither time nor opportunity have afforded me means for this purpose. The thirteen analyses of the urine recorded above are only a few selected from many others made by me, and were effected according to Becquerel's method.

COLCHICUM has been employed in all forms of the disease, but it appears to me more particularly useful in *Articular Rheumatism*. In Dr Watson's[1] words, "our wishes and our expectations from Colchicum are often doomed to be defeated. I believe that in proportion as the synovial symptoms predominate, or mix themselves distinctly with the fibrous—in proportion as the disease approaches in its characters to Gout—you may expect to be successful with Colchicum. Large doses are not requisite; twenty minims of the *tincture* or the *wine* may be given every six hours, until some relief is obtained; or a grain of the *inspissated juice*, or of the *acetic* extract of Colchicum, every four hours. Under this treatment the disease sometimes vanishes within three or four days; the medicine producing sickness and purging, and the Rheumatism or the Rheumatic Gout rapidly declining. Occasionally the same favourable event takes place, although there has been no disturbance of the stomach and bowels."

The cases in which Colchicum appears to me to be more preeminently useful are those in which the disease attacks the joints, and is of an erratic character—*e. g.*, suddenly disappearing from one

[1] Watson—op. cit.

joint, and as suddenly appearing in another; these, I believe, are the cases in which the heart and other internal organs are chiefly affected, and probably if the active operation of Colchicum is secured early, these formidable secondary diseases are less likely to occur.

Of the use of Colchicum topically, little need be said. It is reported to have been employed with great success by Dr Gumpert,[1] who records the case of a clergyman, who had been confined to his bed for six weeks with Rheumatism; on the fifth day, after using frictions with the tincture of the seeds, he was enabled to leave it.

Dr Laycock[2] has likewise used it with great success in Gout, Rheumatism, and also venereal pains.

V.—*Use of Colchicum in other diseases.*

1. In cutaneous diseases.—*Urticaria.*[3] In a case of this disease, where the urine was of low specific gravity (1010), and was found on examination to be much deficient in Urea and Uric Acid, Colchicum was employed with complete success. The urine before taking Colchicum was of a pale straw colour, transparent, and left no deposit on standing. It was analysed according to Becquerel's method, and found to contain :—

Urea, ...	6·91
Uric acid, ...	0·05
Inorganic salts, ...	12·03
Organic matter and water, ...	981·01
	1000·00

Colchicum was then administered, and a fortnight after the urine was again examined. Density 1029·9. It was found to contain :—

Urea, ...	20·36
Uric acid, ...	0·50
Inorganic salts, ...	12·72
Organic matter and water, ...	966·42
	1000·00

Here, then, it will be seen that the physiological action of Colchicum was well marked. The *Urea* was more than tripled in its amount, and raised above the normal standard. The increase of *Uric Acid* was in a tenfold ratio, whilst the other organic constituents and water suffered a corresponding diminution, the inorganic salts remaining nearly as before.

In *Prurigo.*[4] Dr Elliotson gives the case of a man, labouring under this disease in its most inveterate form, to whom half a

[1] Gumpert—Revue Médicale—I.
[2] Laycock—London Medical Gazette, vols. xxiii., xxiv.
[3] Monthly Medical Journal. 1846.
[4] Elliotson—Lond. Med.-Chir. Review, vol. vii.

drachm of the Vinum Colchici was administered thrice daily. This the patient took for three weeks; at the end of which time he was completely cured.

Colchicum would thus seem to answer well in some cases of skin diseases where the urine is of low specific gravity.

2. In nervous diseases.—*Hysteria.* Mr Raven[1] details the case of a young woman, who was thrown into strong hysterical convulsions, by seeing the death of a relation. She had been treated by fœtids, volatiles, cathartics, &c., but without relief. She was admitted into hospital under the care of Dr Alderson, who, having lately seen the good effects of Colchicum in severe cases of *Chorea*, was induced to try it on her. Thirty drops of the tincture were taken every eight hours. In a few days the convulsions left her, and did not return. In *Chorea*[2] also Colchicum has been employed with reported benefit. The cases are mentioned of three children having been relieved from this disease in three or four days, by using from ten to twenty minims of the tincture of Colchicum daily.

3. On the *Genito-Urinary System.*—Dr Clutterbuck[3] has seen in Colchicum a peculiar influence over uterine contractions. He administered ten grains of Colchicum in powder to a female in whom the uterus was contracted over the placenta. The dose was repeated, the os uteri dilated, uterine contractions came on, and the placenta was removed in a state of putrefaction.

Dr Metta[4] relates a case of abortion, in which the placenta was retained, and uterine contractions had ceased. Two doses of powdered root of Colchicum, at an interval of half an hour, were prescribed, and soon after uterine contractions came on.

In *Leucorrhœa*, Mr Ritton[5] experienced great benefit from the use of Colchicum in powder. A week or ten days was generally sufficient to complete a cure, the patient being strictly kept from all alcoholic liquors during its exhibition.

In *Gonorrhœa*, Eisenmann,[6] of Berlin, speaks very highly of the results obtained from the use of an opiate wine of Colchicum in this disease. The preparation he uses is composed of three drachms of Colchicum wine and half a drachm of tincture of opium. This is either given in increasing doses, or twenty drops twice daily. The cure is said to be generally complete from the seventh to the fourteenth day.

In *Chordee.*—In a case of *Gonorrhœa*, for which cubebs had been

[1] Raven—London Medical and Physical Journal, vol. xxxvii.
[2] Bibliothèque Médicale, vol. lviii.
[3] Clutterbuck—Mérat et de Lens.—Dictionnaire de Matière Médicale.—Il Filiatre Sebasio, 1843.
[4] Metta—American Journal of the Medical Sciences, vol. viii.
[5] Ritton—Lancet, 1833-34, vol. ii.
[6] Eisenmann—Casper's Wochenschrift, No. xxxv.—Medical Times, 1849.

prescribed, severe chordee supervened. Sir B. Brodie[1] remarked that Colchicum relieved the painful symptoms of chordee better than opium, and had also the effect of restraining sexual desire. He gives a drachm of the wine in one and a half ounces of camphor mixture for a dose.

4. Effects of Colchicum on the *Salivary Glands.*[2] Mention is made of three cases in which profuse ptyalism resulted from the use of *half a drachm* of the tincture of the seeds of Colchicum thrice a-day for some time. In one of these cases, the patient had never been salivated, nor had any mercury ever been taken. They were all cases of ophthalmia.

5. In *Tetanus.*[3] Dr Smith, of Port-au-Prince, employed Colchicum advantageously in tetanus, both traumatic and idiopathic. He gave it in full doses, repeated every half hour, till it produced an emetic or cathartic effect.

6. As an *Anthelmintic* and *Vermifuge.* Dr Pereira[4] states that Chisholm and Baumbach used it successfully in expelling tapeworm; and Mérat et de Lens[5] mention that Bauhin employed it as a vermifuge for pediculi of the head and pubis.

7. As an *Intoxicating agent.*—Chaumeton[6] says that the Turks procure for themselves a kind of ecstatic drunkenness with the Vinum Colchici.

These are merely mentioned as the uses to which Colchicum has been put, as much greater experience than is recorded here would be necessary before we could place much reliance on its power in any of these diseases.

8. In *Cholera Asiatica.*—In 1833, Mr Cotter[7] published a notice of having used Colchicum in Asiatic cholera. His cases are not detailed; and nothing more is remarked, than that he used it with perfect success in all stages of the disease.

No dependence can be placed on statements so vague as this, with regard to the utility of any remedy; nor does it appear easy to understand upon what principles a substance having the physiological action of Colchicum could be expected to be useful, either in the preliminary Diarrhœa or in the stage of Collapse.

The well-known fact, however, that many cholera patients, after having passed from the stage of *Collapse* into that of *Re-action*, continue to be affected with complete *suppression of urine;* that many of these die in a comatose condition, obviously from accumulation of Urea in the blood, naturally point out the employment of *diuretics*, as being indicated in the re-actionary and subsequent stages of the disease, and the power of Colchicum of eliminating that principle, seems to indicate it as the form of diuretic most applicable to the occasion.

[1] Brodie—Lancet, 1838-39, vol. ii.
[2] Dublin Hospital Gazette, 1845.—London Medical Gazette, 1845.
[3] Smith—Wood and Bache's United States Dispensatory.
[4] Pereira—Materia Medica.
[5] Mérat et de Lens—Dictionnaire.
[6] Chaumeton—Flore Médicale, vol. iii.
[7] Cotter—Lancet, 1833-34, vol. ii.

That the non-existence of urea in the urine, and its accumulation in the blood, are at all events pathological conditions of this morbid state, has been abundantly established.

In 1832, Dr O'Shaughnessy[1] published the case of a female, the *serum* of whose blood contained 1·40 per mille of *Urea;* and in 1849, Dr W. Robertson[2] corroborated this fact, by publishing the details of several analyses of blood, taken from the patients in the Cholera Hospital here. In the same year, Dr James Begbie[3] published several analyses of the urine, by which it was distinctly shown that little or no *Urea* is present in that fluid obtained from cholera patients.

During the late epidemic of cholera in this city (1849), I had a favourable opportunity of observing the action of Colchicum as a diuretic in this disease. In the case of a female, in whom a very small quantity of urine, deficient in urea, was voided, the *Acetum Colchici* was prescribed, and its exhibition was followed by the desired effect upon the urine, and with great benefit to the patient. Other cases occurring in the hospital were not so successful, on account of the accession of diarrhœa.

Two great indications in the treatment of this disease being to increase the amount of urine, when that is deficient, which is almost universally the case, and to aid the elaboration of those principles which it is the province of the kidneys to eliminate, the accumulation of which in the blood we may readily suppose to be the cause of the accession of coma, it appears probable that Colchicum might prove of eminent service, and is at all events well worthy of further trial.

9. In *Bright's Disease.*—A consideration of the physiological action of COLCHICUM,—namely, its diuretic action on the kidney, combined with the property of increasing the elimination of Urea, leads me to suggest the probability of its being found useful in some cases of Bright's disease. This I venture to offer as a mere theoretical speculation, which I have had little opportunity of submitting to the test of experience, and which, so far as I can find, has not been adopted in practice by any of the authors who have written on this subject. It is true that Dr Prout[4] mentions the use of Colchicum in this disease, but only where the gouty diathesis is present. Irrespective of this state, I venture to suggest its employment.

The presence of *Urea* in the blood appears to be one of the established phenomena in the advanced stages of Bright's disease.

In 1829, Dr Bostock suggested that *Urea* being deficient in the *Urine* of patients labouring under Bright's disease, might be detected in the *Blood*. He accordingly "sought for it in the serosity of

[1] O'Shaughnessy—Chemical Pathology of Asiatic Cholera, 1832.
[2] W. Robertson—Monthly Medical Journal, 1849.
[3] James W. Begbie on the Urine in Cholera, 1849.
[4] Prout on Stomach and Renal Diseases.

several of Dr Bright's patients, but could detect only a matter possessing peculiar properties, which seemed to approach to those of Urea." [1]

In the same year, Dr Christison[2] first detected this principle in the serum of the blood in several cases of Bright's disease. One case which he relates is as follows:—

The urine in this case, although not greater in quantity than natural, contained only a fifth of the normal proportion of *Urea*. The action of nitric acid on the extract of serum produced a beautiful radiated mass of foliaceous pearly crystals of *Nitrate* of *Urea*.

In 1840, Dr G. Owen Rees[3] published a statement of his analyses of the blood of patients labouring under Bright's disease, showing a diminution in the amount of *Albumen*, and an abnormal quantity of *Urea* in the blood.

The following are his analyses:—

(1.) Serum—Sp. gr. 1015—Contained in 1000 grains,—

Albumen, ...	46·980 grains.
Urea, ...	0·209 —

(2.) Serum—Sp. gr. 1025—Contained in 1000 grains,—

Water, ...	904·20
Albumen, ...	65·00
Extractive and salts, ...	30·30
Urea, ...	0·50
	1000·00

(3.) Serum—Sp. gr. 1029, or natural—Contained in 1000 parts,—

Water, ...	896·6
Albumen, ...	81·6
Extractive and salts, ...	21·3
Urea, ...	0·5
	1000·0

In the first two cases there is a deficiency of *Albumen*; in the last case, although the albumen is normal in quantity, we have morbidly present nearly 0·5 per mille of *Urea*, for in health but a very slight trace of that substance can be detected.

Dr Rees states the largest amount of *Urea* he has found in the blood of Bright's disease is 0·5 per mille, and the smallest 0·209.

Dr Bright[4] states,—"In one very remarkable case, where the albuminous condition of the urine has constantly existed, as far as I know, from frequent experiment, for above three years, the quantity of urea in the blood is very considerable. The results of chemical analysis by Dr Babington were, that the urine

[1] Vide Paper by Dr Christison—Ed. Med. and Surg. Journ., vol. xxxii.
[2] Christison—Op. cit.
[3] Rees—Guy's Hospital Reports, vol. v.
[4] Bright—Guy's Hospital Reports, vol. i., 1836.

did not contain one-third of the *Urea* which it does in health, while about one per cent. of *Albumen* supplied its place. The serum of the blood was remarkably light, in consequence of its deficiency in albumen, having a specific gravity of 1021 instead of 1030; and the quantity of *Albumen* in 1000 grains of serum, amounting, after careful drying, to only 50 grains; whereas from 80 to 100 parts in 1000 is the usual proportion in healthy serum; and it contained fully as much *Urea* as the urine did, the 1000 grains yielding nearly 15 grains of that substance."

The following analyses of the urine in Bright's disease will show the relation which *Urea* and *Albumen* bear to each other and to the other constituents of the urine; but first the analysis of normal urine, as given by Becquerel, must be noticed. Specific gravity 1018·900.

Water, ...	968·315
Urea, ...	13·338
Uric acid, ...	·391
Inorganic salts, ...	7·695
Organic matter, ...	9·261
Total, ...	1000·000

Compare now the following analysis of the urine in Bright's disease (Simon[1]).

	I.	II.
Specific gravity, ...	1014	1022
Solid constituents, ...	33·90	66·50
Water, ...	966·10	933·50
Urea, ...	4·77	10·10
Uric acid, ...	0·40	0·60
Fixed salts, ...	3·04	10·00
Extractive matters, ...	2·40	—
Albumen, ...	18·00	33·60

It will be seen that in the first of these the *Urea* is only a third of its normal quantity, *Uric acid* and *Salts* nearly natural, while there is morbidly present four and a half times the amount of *Urea*, of *Albumen*. In the second, the *Albumen* is in the ratio of three to one of *Urea*.

From these statements it will be seen that in Bright's disease the *Albumen* morbidly excreted in the Urine, and the *Urea*, are correlative and vicarious principles. That in the *Blood*, while the *Albumen* is diminished, *Urea* is morbidly retained; and in the *Urine*, while *Urea* is greatly deficient, *Albumen* is morbidly present.

Might we not then reasonably expect that COLCHICUM (acting, as has been stated) would sometimes act as a favourable auxiliary in the treatment of this disease? The cases I have recorded of its use in the dropsy succeeding to scarlatina, would seem to corroborate

[1] Simon's Chemistry (Syd. Soc. Pub.)

this view. At all events, when *Ascites* or *Anasarca* are present in Bright's disease as intercurrent affections, the use of Colchicum, both as a cathartic and diuretic, seems to me to be indicated; and in those cases where *Coma* supervenes, from accumulation of urea in the blood, I am confident that it will prove of eminent service.

In the observations contained in the foregoing paper, I have endeavoured to give a correct account of the growth of the COLCHICUM plant; and have offered a suggestion as to the probable cause of the appearance of the abnormal or *adventitious* bulbs which are occasionally observed. From a careful consideration of the growth, chiefly with reference to the medicinal qualities of the bulb at different seasons, I am led to believe that the fittest time for gathering for pharmaceutical purposes is the middle of *July*. In the section devoted to the chemical history of the drug, considerable doubt has been expressed concerning the *crystalline* nature of the alkaloid COLCHICIA; that it differs, however, in many respects from VERATRIA, has been abundantly proved by the experiments of Geiger and Hesse. Considerable space has been occupied by the statement of the physiological effects of Colchicum: and particular attention is directed to the powers which it has, as a sedative, diuretic, diaphoretic, and emetico-cathartic, and to the remarkable property which it possesses of altering the renal secretion. The remainder of the paper is devoted to the therapeutic actions of the drug, particularly as a diuretic in Dropsy, a sedative in Inflammatory diseases, and as possessing a powerful control over the paroxysms of Gout and Rheumatism. An attempt has been made to refer its action in the two latter diseases, in great part at least, to the property it possesses of altering the chemical qualities of the *blood* and *urine*; and to prove, by statistics, that in Rheumatism (and it is believed in Gout also) its beneficial effects are seldom apparent until some of its ordinary physiological actions, such as diuresis, purging, or vomiting, manifest themselves. The use of Colchicum in the Dropsy succeeding to Scarlatina has been mentioned, and a suggestion offered, that its employment might be extended to some cases of Bright's disease, and for the relief of comatose symptoms consequent upon *suppression of urine*.[1]

I take this opportunity of returning my best thanks to Professors Christison and Balfour for the kind assistance which I received from them, while engaged in the foregoing investigations.

[1] An outline of this paper was read before the Royal Medical Society in December 1849.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

DE

ARTERIARUM

NOTIONE, STRUCTURA, CONSTITUTIONE CHEMICA ET VITA,

DISQUISITIO

CRITICA, EXPERIMENTIS FULTA,

PRAEMIO ORNATA,

AUCTORE

MAXIMIL. JOH. SIGISM. SCHULTZE.

ACCEDUNT TRES TABULAE LITHOGRAPHICAE.

GRYPHIAE,
IN LIBRARIA C. A. KOCHIANA.
MDCCCL.

SACRUM ESSE VULT

HOC OPUSCULUM

Filius.

Offero Tibi, pater dilectissime, quae jam Tua sunt. Nam Tuae institutioni, qua jam puerum me in rerum naturalium studium introduxisti, Tuis demonstrationibus, quibus microscopium prae ceteris omnibus instrumentis gratissimum ad investigandas res naturales mihi fecisti, Tuis praelectionibus debeo, si quae insunt in hoc opusculo nova aut accuratius observata. Plura eorum experimentorum de arteriarum functione, quae descripsi, jam in praelectionibus Tuis instituisti, et omnino Tu animum auditorum Tuorum ad gravissimas functiones arteriarum earumque constitutionem chemicam saepe ad-

vertisti, quare gratissima mihi erat elaboratio hujus opusculi. Cur Te in hoc commentario non patrem sed praeceptorem solum nominaverim, facile intelliget, qui publici certaminis causa eum conscriptum esse considerat.

Praefatio.

Anno hujus saeculi XLVI gratiosus medicorum hujus universitatis ordo ad publicum certamen medicinae studiosis proposuerat:

„Disquisitionem criticam experimentis fultam praecipuarum quas viri docti proposuerunt sententiarum de arteriarum viribus vitalibus."

Qua de causa in primis anni 1847 mensibus scriptam hanc dissertationem ante Cal. Aug. ejusdem anni facultati gratiosae tradidi.

Postquam Octobri 1847 praemio ornatum erat opusculum, a duobus micrographis acutissimis, *Donders* et *Koelliker*, duo de arteriarum structura commentaria typis vulgata sunt [1]), quorum observationes pro maxima parte cum meis disquisitionibus consentiunt, ita ut in horum clarissimorum virorum auctoritate sicut in patris dilectissimi judicio confirmationem gratissimam repererim. Ex annotationibus, in quibus *Dondersii* et *Koellikeri* observationes adjeci, Lector benevolus facile videat, quibus in partibus horum virorum sententia cum mea congruat.

---

1) Untersuchungen über die Natur der krankhaften Veränderungen. der Arterienwände, die als Ursachen der spontanen Aneurysmen zu betrachten sind, von *Donders* und *Jansen* in dem Archiv für physiologische Heilkunde v. *Griesinger* Bd. VII, 1848. p. 361 und Beiträge zur Kenntniss der glatten Muskelfasern v. *Kölliker* in der Zeitschrift für wissenschaftliche Zoologie v. *Siebold* u. *Kölliker* I. 1848. p. 48.

Index.

		Pag.
Cap. I.	De notione nominis arteriae	1
Cap. II.	De structura arteriarum	4
1)	Descriptiones, quas *Halller, Hunter, Bichat, Meckel, Mascagni* proposuerunt	4 — 6
2)	Sententiae a *Cloquet, Beclard, C. A. S. Schultze, Lauth, Schwann, Eulenberg, E. H. Weber, C. H. Schultz, Purkinje* et *Räuschel* prolatae	7 — 8
3)	Sententiae a *Purkinje* et *Rosenthal, Henle, Donders* et *Mulder* propositae	9 — 10
4)	Quae ipse observavi a) de epithelio arteriarum	10 — 11
	b) de tunica intima	12
	c) de fibris longitudinalibus	13
	d) de tunica media	14 — 16
	e) de tunica externa	16
5)	Annotationes de *Dondersii* et *Koellikeri* commentariis de arteriarum structura post hujus opusculi scriptionem editis	17
Cap. III.	De constitutione chemica tunicarum arterias formantium	19
1)	De substantiis aqua extrahendis ex arteriarum tunicis	21
2)	De fibrarum contractilium substantiis in aqua non solubilibus	21
3)	De fibris elasticis in universum et arteriarum in specie	28
Cap. IV.	De viribus vitalibus in arteriis	33
1)	De prima arteriarum formatione	33
2)	De arteriarum vi in nutritionem	38
3)	De arteriarum vi in secrectionem	41
4)	De arteriarum contractilitate ex expansibilitate, vi in sanguinis motum et in pulsum .	44
Cap. V.	Resultata totius opusculi	55

Scripturo mihi de arteriarum viribus vitalibus necessarium esse videtur, primum definitione concinna confirmare, quae sint arteriae, tum de structura et textura microscopica, quae sint nota exponere, et in primis etiam de chemica constitutione singulorum stratorum inquirere. Itaque capite primo de definitione arteriarum, secundo de structura, tertio de constitutione chemica, quarto de viribus vitalibus disseram.

I. De notione nominis arteriae.

Definitio arteriarum vulgo data haec est, arterias esse ea sanguinis vasa, quae a corde ad omnium organorum peripheriam sanguinem ducant. Sic dicit *Meckel*[1]): „Die Gefässe zerfallen zunächst in: 1) Pulsadern (Arterien), die das Blut vom Herzen ab ebenso in die Substanz desselben führen; und 2) Blutadern (Venen), durch welche es zu ihm zurückgelangt.“ Porro *Hildebrandt*[2]) „Nimmt man nicht sowohl auf die Farbe des Blutes, das in den Gefässen, die zum Kreislauf dienen, enthalten ist, als auf die Beschaffenheit der Wände derselben, und darauf, ob sie das Blut dem Herzen zuführen, oder ob sie dasselbe vom

1) System der vergleichenden Anatomie. Th. 5 p. 8.
2) Handbuch der Anatomie des Menschen, herausgeg. v. *E. H. Weber* 1831, pag. 26.

Herzen wegführen, und also ob sie dem Drucke einer vom Herzen fortgestossenen Flüssigkeit ausgesetzt sind, oder nicht, Rücksicht; so theilt man die Blutgefässe in Arterien und Venen". Etiam *Arnold*[1]) hanc sententiam sequitur. „Die Blutgefässe, vasa sanguifera, die man, je nach dem sie das Blut vom Herzen zu den Theilen des Körpers, oder von diesen wieder zurück zu dem Herzen bringen, als Arterien, arteriae, oder Blutadern, venae, bezeichnet."

Accomodata est haec definitio iis vasis, quae a corde oriuntur. Insunt vero, quum in corpore humano, iisque animalibus, quorum systema vasorum humano simile est, mammalibus, avibus, amphibiis pro parte, tum in piscibus et animalibus evertebratis, quae secundum nostras cognitiones vasorum systemate sunt praedita, talia vasa arteriosa, quae non a corde oriuntur. Systema venae portarum hominis et animalium multorum, talem partem habet, quae, quamquam non a corde incipit, tamen arteriis adnumeranda est. Sanguinem enim a venis mesentericis, gastricis et vena lienali in unum truncum collectum, pars arteriosa venae portarum in vasa capillaria hepatis immitit, ubi sanguis ater seu venosus simili modo purgatur, sicut is, qui in pulmones per arterias pulmonales ad decarbonisandum immittitur. Satis apparet, in hoc vas arteriosum definitionem supra datam non quadrare. — In piscibus et amphibiis nudis, quamdiu branchiis respirant, ea vasa, quae sanguinem oxydatum in totum corpus distribuunt, seu arteriae circulationis magnae non e corde oriuntur, sed e confluentibus branchiarum venis, quae in truncum arteriosum conjunctae aortam conformant, ita ut e corde modo arteriae branchiales oriantur, et in arterias stricte sic dictas, quia non e corde emanant, definitio supra data non cadat.

In molluscis plurimis et pulmonaceis et branchiis praeditis atque in crustaceis decapodibus a corde modo aorta oritur et arteriae pulmonales seu branchiales a venis e corpore revertentibus componuntur, ita ut haec vasa secundum definitionem supra commemoratam excludenda sint ex arteriis.

1) Handbuch der Anatomie des Menschen 1844. pag. 95.

In holothuriis[1]) singulisque iis echinodermis[2]), in quibus de circulatione sanguinis quaedam sunt explorata, similis est systematis vasorum forma, eo solum discrimine, quod in holothuriis cordis loco circulus vasculosus contractilis circa oesophagum invenitur.

Denique in iis animalibus, quae cor non sed systema vasorum habent, satis apparet, definitionem supra commemoratam nullam habere vim.

Quare aliud signum est quaerendum, quod omnibus omnium animalium arteriis pari modo conveniat; tale signum id mihi videtur esse, quod praeceptor meus illustrissimus *Schultze*[3]) in collegiis et in libro suo exposuit, sicut *Tiedemann*[4]) in physiologia sua, nimirum, sanguinem in arteriis a trunco in ramos et ramulos fluere, quo ex signo haec definitio deducenda est:

„Omnia vasa circulationi sanguinis inservientia, in quibus sanguis a trunco in ramos immittitur, arteriae sunt nominanda."

Incipiunt ergo arteriarum trunci aut e corde aut e venis confluentibus in unum truncum, ex quo nova ramificatio arteriosa exoritur. — Ubi vero est finis arteriarum subtilissimarum? Hoc est difficile dictu. Maxima arteriarum pars directe in venas transit, et venae incipiunt ubi sanguis a ramulis in ramos influere incipit; ibique arteriarum est finis. Is locus vero non semper accurate definiri potest, quia vasa capillaria fere omnibus in organis retia formant, in quibus sanguis propter anastomoses multas motum non plane constantem praebet. Quam enim in partem sanguis fortius immittitur, vel attrahitur, in eam fluit,

1) *Tiedemann*, Anatomie der Röhrenholothurie, des pommeranzenfarbenen Seesterns und des Steinigels. Landshut 1816.
2) *Valentin* l'anatomie du genre Echinus. Neuchatel 1842.
3) Systemat. Lehrb. der vergleichd. Anatomie Th. I. 1828. pag. 144—145.
4) Physiologie des Menschen Th. 1. 1830. pag. 185. pp. 310.

quare in hisce vasorum retibus mox huc mox illuc moveri potest. Semper quum dijudicandum sit de vasis quibusdam, num arteriis an venis sint adnumeranda, secundum definitionem supra datam quaestio solvenda est, si omnino discrimen potest fieri. Arteriarum pars secundum observationem nonnullorum virorum[1]) doctorum in vasa serosa transit, id est in vasa, quae ita sunt subtilia, ut sanguinis globulos excipere non possint, sed solo sero sanguinis impleta sint. De hoc vasorum genere in capitibus de nutritione et secretione fusius disseram. Initium horum vasorum facile est definiendum, nam ubi arteria tam subtilis fit, ut sanguinis globuli in eam immitti non possint, ibi vas serosum incipit.

II. De structura arteriarum.

Quae viri docti de arteriarum tunicis exploraverunt, nunc brevibus exponam, et ab *Hallero*, qui post Ludwigium, Monroum, Swietenium, Albinum uberius de arteriarum structura disseruit, incipiam.

Haller quinque distinguit tunicas. Prima est membrana cellulosa adscititia, externa, de qua dicit[2]):

„Aliud autem involucrum constantissimum totam arteriam omnesque ejus surculos undique continet, cellulosa nempe tela: quae ea vasa ad vicinas membranas, ad viscera, ad periosteum, ad musculos religat.“

Usque ad hoc tempus haec tunica idem habet nomen.

Secundam membranam cellulosam propriam his verbis definit[3]): „uti haec cellulosa, quam vocant, arteriae membrana, exteriora versus in vaginas laxatur, ita intus, et qua arteriarum musculosas fibras proxima continet, sensim arctior, densior, magis alba et solidior fit, et cum magna duritate elastica, aegre compressilis et facillime resilitura: vasisque nunc vix

1) *Müller* Physiologie Th. 1. 4te Auflage 1844. pag. 175.
2) Elementa physiologiae corp. hum. 1757. tom. 1, pag. 60.
3) loco citato pag. 62.

conspicuis tingitur, et eadem principem omnino arteriae partem constituit, quae reliquas multum crassitie superet et praeter quam non alia in animalibus frigidi sanguinis reperitur.“

Tertiam membranam arteriarum *Haller* musculosam nominat. Sunt fibrae in circulum flexae, carneae, et formant una cum tunica antecedente tunicam mediam recentiorum. De fibris longitudinalibus, quas jam *Willisius*[1]) descripserat, *Haller* se nunquam eas vidisse dicit his verbis[2]) „Verum anatome et microscopium omnino fibras longitudinem sequentes nunquam demonstravit, aut mihi, aut aliis ante me scriptoribus, quorum auctoritate meam tueor.“

Quarta membrana *Halleri* est cellulosa interior. Inter musculosas fibras intimamque membranam hanc sitam esse demonstrat, praecipue auctoritate *Albini* et *Waltheri*, et in eam effundi flavum eum succum, qui callosus primo, deinde osseus fiat. Microscopica investigatione accuratiore satis confirmatum est, eam non adesse, quare nil amplius de ea.

Quinta denique membrana *Halleri* est intima, usque ad nostrum tempus eodem nomine definita, donec *Henle* hanc tunicam „gefensterte oder gestreifte Haut“ nominavit, qui ope microscopii multis foraminibus perforatam eam invenit, de quo postea uberius.

Post *Hallerum* viri docti tunicam cellulosam externam et tunicam intimam, ut jam dixi, iisdem fere verbis descripserunt ut *Haller*; de tunica media vero dissentiunt multi viri anatomia periti eo modo, ut alteri eam tunicam simplicem ex uno genere fibrarum conformatam describant, alteri e variis fibris compositam. Tali modo *J. Hunter* stratum elasticum et musculosum bene distinguit, his verbis[3]): „Der grösste Theil des Schlagader-

1) Pharmacia rationalis pars 1. pag. 154. Opp. omnia. Lugd. 1681. *Willisius* has fibras dicit nerveas, nec in tunica musculosa decurrentes, sed tunicam intimam constituentes describit.
2) Loco citato pag. 65.
3) Versuche über das Blut, die Entzündung und die Schusswunden; übers. v. *Hebenstreit*. 1795. I. pag. 241.

systems scheint offenbar aus zwei Substanzen zu bestehen, die man besonders an Schlagadern von mittlerer Grösse bemerken kann, wo beide Substanzen gleichförmiger von einander getrennt sind." „So wie man von den Stämmen der Arterien nach ihren Aesten mit der Untersuchung fortgeht, so findet man, dass der Unterschied zwischen der innern und äussern Substanz derselben deutlicher wird; der innere Theil, welcher dunkler aber dabei etwas durchsichtig ist, fängt fast unmerklich in den grossen Gefässen an und wird in den Aesten, sowie sie sich vertheilen, und folglich kleiner werden, immer dicker; dahingegen der äussere weisse Theil nach und nach doch schnelle abnimmt, so wie die Arterien kleiner werden und ihre musculöse Substanz dicker wird",.... „an manchen Orten, besonders wo sich die elastische und Muskelsubstanz der Arterien einander berühren, sind die Fasern von beiden sehr mit einander vermischt und verflochten."

Bichat[1]) contra unum solummodo genus fibrarum in tunica media descripsit, sed primus has fibras circulares novam speciem esse dixit. Multis experimentis, praecipue maceratione et coctione adhibita conclusit, a musculis, a tendinibus et ligamentis fibras arteriarum plane diversas esse. Duritate, fragilitate et elasticitate distinctas eas describit. Fibrarum musculis similiorum nullam facit mentionem.

Meckel[2]) tunicam mediam e fibris circularibus, duris, elasticis, omnibus in arteriis aequalibus constructam describit, fibras longitudinales internas plane negat sicut *Bichat* et *Haller*.

Mascagni[3]) in tunica media vulgo sic dicta duo distinguit strata, alterum, quod dicit elasticum, externum, alterum internum nerveum. In ambobus stratis vero solas fibras elasticas eum vidisse apparet ex descriptione ejus et e tabulis, quare mihi non

1) Anatomie générale. Paris 1801. 3me edition. Paris 1831. Tom. 2. p. 48. ff.
2) Handbuch der menschl. Anatomie. I. 1815. p. 182.
3) Prodromo della grande anatomia. Firenze 1819. p. 61. Tab. XII. Fig. 36—41. Tab. XIII. Fig. 1—6.

satis elucet, quam ob causam distinguat inter ambo strata. Fibras elasticas describit his verbis: „I filamenti di questa seconda membrana (elastica) a occhio nudo ci presentano un andamento arcuato, e come circolare; ma col microscopio al contrario vi si vede un intreccio analogo a quello dei tessuti che noi diciam volgarmente fatti a guisa di stoja."

Delineavit fibras ramificatas, forsan etiam laminas elasticas e fibris retis in modum conjunctis compositas ex arteriis et venis iconibus suo more confectis.

In libris duobus annis post a *Cloquet*[1]) et *Béclard*[2]) editis etiam nullas alias fibras arteriarum commemoratas invenimus ac elasticas; etiam ramificationem et concatenationem fibrarum elasticarum non viderunt. *Béclard* dicit de fibris elasticis: „Les fibres du tissu élastique ont la même disposition, que celles du tissu fibreux blanc," quod naturae omnino non respondet. Verum in eo progressi sunt hi viri, quod congruentiam fibrarum elasticarum in arteriis cum fibris ligamentorum flavorum columnae vertebralis, cum fibris ligamenti nuchae mammalium nonnullorum, cum nonnullis fibris tracheae et bronchorum, itaque cum fere omnibus illis telis fibrosis, quas nostro tempore ad telam elasticam adnumeramus, cognoverint.

Secundum notam quandam, quam *Blandin* in editione sua libri a *Bichat* de anatomia generali compositi adjunxit[3]), *Duméril* primus fuisse videtur, qui genus fibrarum elasticarum constituerit.

Fibrarum duo genera varia in tunica media arteriarum, alterum musculis simile, alterum elasticum distinguit praeceptor illustrissimus *Schultze*[4]) his verbis: „Die Gefässhautfaser, fibra

1) Anatomie de l'homme. Paris 1821. I. p. 35.
2) Additions à l'anatomie générale de *Bichat*. Paris 1821, deutsch v. Cerutti. Leipzig 1823. p. 92. 195.
3) Anatomie générale par *Bichat*. Nouv. edit. par *Beclard* et augmentée d'un grand nombre de notes nouvelles par *Blandin*. Paris 1830. II. p. 54.
4) Lehrb. der vergleichenden Anatomie, Th. I. 1828. pag. 126.

vasorum, ist eine rundliche, kurze, sehr feine, elastische und brüchige Faser, die, unter spitzen Winkeln mit den benachbarten verbunden, platte, bandförmige Bündel bildet, welche die innerste Haut der Blutgefässe theils ringförmig, theils in der Längenrichtung verlaufend, umgeben. Farbe und Durchsichtigkeit der Gefässfaser sind, sowie ihre Lebenseigenschaften in den verschiedenen Thieren und selbst in verschiedenen Theilen desselben Thieres sehr verschieden. Sie ist um so blasser, durchsichtiger, weicher und zusammenziehungsfähiger, überhaupt der Muskelfaser ähnlicher, je unvollkommener das Thier ist, welches man untersucht, und dieselben Eigenschaften hat sie an den Venen und feineren Arterienverzweigungen der vollkommneren Thiere. Die grösseren Arterien der letzteren sind dagegen mit Fasern versehen, welche sich mehr den Sehnenfasern nähern, von diesen jedoch sich wesentlich durch Undurchsichtigkeit, Kürze und netzartige Verbindung zu Bündeln, so wie durch ganz eigenthümliche Mischung unterscheiden."

Quorum duorum generum fibrarum alterum musculis simile viri docti, qui postea de arteriarum tunicis scripserunt, usque ad *Henle* plane omiserunt.

Lauth[1]) *Schwann*[2]) et *Eulenberg*[3]) cognitionem nostram fibrarum elasticarum quidem amplificaverunt, in arteriis vero praeter has fibras elasticas modo paucas fibras cellulosas se invenisse dicunt.

Eodem modo *E. H. Weber*[4]) et *C. H. Schultz*[5]) in eo consentiunt, quod fibras elasticas solas arteriarum tunicam mediam componere dicant.

Purkinje et *Räuschel*[6]) tria genera fibrarum in arteriarum

1) L'institut I. II. Nr. 57. 1834. pag. 191.
2) Encyclopäd. Wörterb. der medic. Wissenschaft. Bd. XIV. 1836. Art.: Gefässe pag. 213.
2) De tela elastica. Diss. inaug. Berol. 1836.
4) Hildebrandt's Anatomie Bd. III. 1831. p. 62.
5) System der Circulation. 1836. p. 220.
6) De arterium et venarum structura. Diss. inaug. 1836. pag. 3. 5.

structura discernunt, fibras elasticas arteriae proprias et duas species fibrarum cellulosarum, „quarum una illis, quae in tendinibus conspiciuntur similis est, altera vero ex fibris mollibus constans, quae constituunt telam cellulosam propriam — —. Haec fibra mollis, quae secundum naturam suam prope accedit ad fibram muscularem, vere habenda est fibra muscularis in tunica musculosa intestinorum etc. „Describit *Räuschel* porro (pag. 11) taeniolas tenuissimas inter strata fibrarum elasticarum positas, quae in sectionibus horizontalibus et perpendicularibus et obliquis appareant et laminae cellulosae illis stratis fibrarum elasticarum interpositae habendae sint. Quas laminas cellulosas Fig. IX. A. B. C. delineavit, ita ut dubitare nequeamus, eum membranam fenestratam *Henlei* quoad situm melius descripsisse quam *Henle*.

Purkinje et *Rosenthal*[1]) paucis annis post hanc descriptionem in eo etiam amplificaverunt, quod in tunica media arteriarum fibras describunt latas, nucleis praeditas, quas elasticas habuerunt, sed distinguendas a fibris elasticis communibus nucleis carentibus. Apparet hos viros fibras contractiles peculiares arteriarum nucleatas jam vidisse.

Henle[2]) disquisitiones microscopicas accuratiores de fibris arteriarum diversis instituit. Eas, quae praeter elasticas insunt et contractiles habendae, accuratius et distinctius nomine „Zellenfasern" descripsit et delineavit, elasticarum vero fibrarum partem a reliquis separavit, et „Kernfasern" nominavit. Tunicae, quas distinguit in arteriis, hae sunt: Stratum intimum ab epithelio formatur, quod *Henle* detexit, et ei, quod tunicas serosas induit, simile invenit. Stratum secundum describit *Henle* membranam pellucidam, tenuissimam, striatam et perforatam, quam „gestreifte oder gefensterte Gefässhaut" nominavit. Qua de membrana in sequentibus uberius disseram. Stratum tertium a fibris

1) *Rosenthal*, de formatione granulosa. Diss. inaug. Wratisl. 1839. pag. 12.
2) Wochenschrift für die gesammte Heilkunde 1840. Nr. 21. p. 329. Allgem. Anatomie 1841. p. 494.

longitudinalibus elasticis formatum describit. Stratum ejus quartum formant fibrae circulares, pallidae, latae, nucleos continentes, fibris organicis musculosis haud dissimiles, multis fibris elasticis tenerrimis (Kernfasern) commixtae.

Hoc stratum jam ab *Hallero* et praecipue ab *Huntero* bene distinctum et musculosum nominatum est, ejusque fibras ope microscopii praeceptor Ill. *Schultze* (ut supra demonstravi) descripsit. Postea vero hae fibrae semper cum cellulosis confusae sunt, donec *Räuschel* et *Henle* hanc confusionem accurata et distincta descriptione solverunt.

Stratum quintum a fibris elasticis solis compositum esse dicit *Henle*, quod versus externam ejus partem fibrae cellulosae strati sexti externi sequantur.

Praeterea inter fibras circulares laminae suae fenestratae partes interdum se invenisse dicit *Henle*.

Quam descriptionem ab *Henle* datam praeter alios viros doctos *Donders* et *Mulder*[1]) nuper probaverunt. Verum de distributione et situ partium illarum laminae fenestratae, quas *Henle* inter fibras circulares arteriarum interdum invenit, accuratius quam *Henle* exposuerunt. Hae laminae enim, quas *Räuschel* jam vidit sed pro tela cellulosa habuit, e fibris elasticis latis ramis inter se conjunctis constitutae sunt, et formant quasi tubulos, qui paribus intervallis inter se remoti, alteri alteros circumdantes, tunicam mediam arteriarum maximarum una cum fibris elasticis et paucis contractilibus componunt, ita ut fibrae illae intervalla inter has laminas expleant, sicut *Räuschel* (l. c. fig. IX. A. B.) delineavit.

Quae ipse de arteriarum tunicis ope microscopii observavi haec sunt: Epithelium, quod tunicam intimam omnium arteriarum induit, constat e cellulis nucleo praeditis, quae cum eo epithelio, quod „Pflasterepithelium“ seu „Schuppenepithelium“ nomi-

1) *Mulder*, Versuch einer physiologischen Chemie. 8te Lieferung 1846. p. 644.

namus, comparari possunt, id vero non plane aequant. Scabendo facile aufertur.

Tres varias formas cellularum epithelialium inveni. Forma primitiva est ea, quam in tab. I. fig. 1. a. delineavi. In cellulis ovalibus, teretibus, pellucidis, vix granulatis nucleus magnus inclusus est cum uno vel pluribus nucleolis. Rarius formam inveni in fig. 1. b. delineatam, ubi cellulas epithelii squamaeformes vides. Sed multo crebriores quam haec ambo genera cellularum sunt cellulae in fibras plus minus productae, quas in fig. 2. ejusd. tab. delineavi. Extrema lineamenta harum cellularum fibris brevibus similium pallidiora sunt, sed nucleus eadem perspicuitate et forma exstat atque in reliquis cellulis epithelialibus. Singulae cellulae caudatae, ut videtur, inter se conjunguntur, ita ut fibrae formentur longiores, quae tum ad membranas coalescunt, in quibus magis minusve distincte singulae cellulae vel fibrae constituentes elucent. (Fig. 3. 4. in tab. I.)

Nucleus paulatim pluribus granulis impletur, et saepe in longitudinem producitur, denique plane evanescit (Fig. 5. 6.). Quibus in membranis epithelialibus paulatim fibrae tenerrimae exoriuntur, quae, quo magis nuclei evanescunt, eo distinctiores fiunt, donec tota membrana ex iis solis composita videtur (Fig. 7).

Haec membrana eandem jam habet intentionem ad convolvendum quam tunicae intimae strata sequentia, quae e fibris permultis elasticis tenerrimis constant. Etiam quod attinet ad chemicam constitutionem haec membrana tenerrime striata similis est stratis tunicae intimae, de qua postea uberius. Nam acido acetico affuso non solvuntur fibrae nec pallidiores fiunt, sed magis dilucide et distincte apparent, sicut fibrae elasticae. Quare verisimile esse mihi videtur, ex hac membrana e cellulis epithelialibus composita, tenerrime striata, tunicae intimae strata e fibris elasticis paulo densioribus constantia paulatim formari.

Quae sententia de transformatione cellularum epithelialium longe abest ab ea, quam *Reichert*[1]) nuper de hac re proposuit,

1) *Müller's* Archiv. 1846. Jahresbericht p. 220.

qui cellulas epitheliales in membranas fenestratas transformari dicit eo, quod cellulae in membranam coalescant, et resorptione nucleorum et parietum, quae cellularum nucleis adhaerent, foramina exoriantur Qua cum sententia consentire non possum. Primum enim nunquam observatum mihi esse videtur, in cellula quadam una cum resorptione nuclei etiam foramen in cellula exoriri in eo loco, ubi nucleus aderat. Tum inter stratum epitheliale et stratum membranarum fenestratarum, sicut in sequentibus demonstrabo, stratum e fibris tenerrimis elasticis compositum interjectum est, qua ex re apparet, epithelium non directe transformari posse in membranam fenestratam. In sequentibus de origine membranarum fenestratarum seu laminarum elasticarum, sicut *Donders* eas nominat, uberius exponam.

Adhuc commemorandum est, inter membranas epitheliales nonnunquam tales inveniri, in quibus nuclei in fibras commutari videntur. Talem membranam ex aorta hominis adulti in fig. 8 tab. I. delineavi.

Transeo ad tunicam vulgo intimam dictam. De ejus structura cum observationibus ab *Henle* propositis non plane consentire possum, quum ejus tunicam fenestratam plerumque non esse intimam epithelio obtectam observaverim. Inveni enim in arteriis majoribus (aorta, carotide, subclavia, iliaca) et hominis et bovis plura strata e fibris tenerrimis, elasticis, ramificatis, longitudinalibus composita, quae illud stratum tenerrime fibrosum, quod in descriptione epithelii commemoravi, sequuntur. Quorum fibrae sunt eo tenuiores, quo propinquiores epithelio sitae sunt, verum tunicam mediam versus earum latitudo paululum augetur, nunquam vero excedunt latitudinem earum fibrarum elasticarum, quas *Henle* „Kernfasern“ nominavit.

Haec strata fibrosa intima magnam habent intentionem ad convolutandum. Delineavi in fig. 9. tab. I. partem tunicae intimae aortae hominis adulti, quam scabendo abstuli. In fig. 10. et 11. fibrae e tunica intima aortae vituli delineatae sunt, in fig. 11. ita ramificatae, ut rete tenerrimum efficiant. Magnitudo est in his figuris quingenties aucta.

In arteriis minoribus (femorali, poplitaea, earumque ramis) non plura talia strata demovere potui, ut in aorta etc., sed unum modo, et id plerumque cum lamina sequenti elastica, sic dicta fenestrata, tam arcte conjunctum inveni, ut alterum ab altera non separare possem. — Sequuntur nempe haec strata tenerrime fibrosa fibrae longitudinales elasticae latae, quae in arteriis majoribus e pluribus stratis, in minoribus (femorali, poplitaea, carotide faciali et cerebrali earumque ramis) ex uno vel duobus solis stratis constant, et in his quidem ramificationibus tam arcte conjunctae sunt, ut laminam forment, in qua lineis longitudinalibus fines fibrarum constituentium indicatae esse solent, et in qua multa foramina inveniuntur, quae sunt interstitia inter fibras constituentes. Hanc laminam *Henle* fenestratam seu striatam nominavit et intimam, epithelio solo obtectam, descripsit. Verum quum in arteriis maximis, ut jam descripsi, plura strata tenerrime fibrosa inveniantur inter epithelium et fibras longitudinales latas (e quibus laminae fenestratae exoriuntur) et hae laminae etiam in arteriis minoribus uno strato fibrarum tenerrimarum longitudinalium obtectae sint, mihi videtur sequi, fibras tenerrimas elasticas pro tunica arteriarum intima epithelium sequente habendas esse, quas fibrae elasticae latae seu laminae fenestratae ex iis exortae excipiunt.

Fibras longitudinales latas ex aorta bovis in fig. 1. tab. II. delineavi. Ita sunt ramificatae, ut jam laminae appropinquentur. Lamina elastica e fibris longitudinalibus constituta ex arteria iliaca communi hominis in fig. 5. tab. II., delineata est. In hac figura sunt fibrae, quae in directione a littera *b* ad *b* currunt, longitudinales, laminam constituentes, fibrae tenerae ab *a* ad *a* currentes arcte conjunctae sunt cum lamina, et sicut in sequentibus demonstrabitur, saepe inveniuntur in laminis elasticis et semper in directione contraria ei fibrarum constituentium. Nec in aorta bovis nec hominis in hac tunica fibrarum longitudinalium unquam tales laminas elasticas e fibris longitudinalibus constitutas invenire potui, sed semper modo fibras latas ramificatas.

Tunica media vulgo sic dicta, ad quam nunc aggredimur, constat e fibris circularibus, quibus modo perpaucae fibrae longitudinales praecipue externam partem arteriae versus admixtae sunt.

Tria sunt genera fibrarum, quae partim commixta partim separata hanc tunicam componunt, fibrae elasticae, fibrae contractiles musculosis organicis similes et fibrae cellulosae, quarum fibrarum tertium genus non magni est momenti in tunica media, sed demum in externa tunica arteriarum praevalet.

Aorta, arteria pulmonalis, carotidis, subclaviae et iliacae partes ortui finitimae nudo oculo monstrant in tunica media fibras flavas, densas, duras, siccas, quae sub miscroscopio fere e solis fibris elasticis latis, valde ramificatis compositae inveniuntur. Carotidis vero pars superior, arteria axillaris, femoralis et harum arteriarum rami nec non arteriae intestinorum continent fibras molles, pallidas, rubicundas, musculosis organicis similes, quae ope microscopii fibrae multo latiores quam elasticae et breviores, pallidae, non ramificatae, sive tenerrime striatae sive moleculis subtilibus obtectae apparent, et nucleos oblongos continent, qui praecipue acido acetico fibris allato, dilucide conspici possunt. Cum his fibris semper conjunctae sunt fibrae elasticae, tenues, interdum ramificatae, ab *Henle* „Kernfasern“ nominatae. Quae ambo genera fibrarum ex arteria femorali hominis adulti in fig. I. tab. III., e carotide bovis in fig. 3. tab. ejusd. delineavi.

Acido acetico fibris admixto celerrime pallidiores fiunt et paene evanescunt, nucleus vero finibus sat distinctis manet et demum longo tempore praeterito pallescit. Nonnullis moleculis subtilibus impletus esse solet. Fibrae elasticae acido acetico non commutantur.

Distinguuntur hae fibrae nucleis praeditae peculiares arteriarum a fibris musculosis organicis praecipue multo majore latitudine et fragilitate. Nam quum eas priusquam microscopio supponeres, acubus perscinderes vel cultro contereres, permultas invenies partes harum fibrarum breves, saepe nucleo praeditas, quarum e finibus non sat distinctis concludendum est, eas fragmenta esse fibrarum longiorum. Talia fragmenta fibrarum contractilium ex aorta hominis commixta cum fibris elasticis latis in fig. 5. tab. III, ex arteria femorali hominis in fig. 2., e carotide bovis in fig. 4 tab. ejusd. delineavi. Nucleus, qui saepe in iis invenitur, modo acido acetico allato tam dilucide apparet.

Verum praeter haec fragmenta semper permultae insunt fibrae cellulis oblongis similes, interdum fere rotundae, omnes nucleo praeditae (Fig. 4. b. c. tab. III.), quas non fragmenta fibrarum esse sed fibras novellas, recens exortas, persuasum habeo. Etiam multi singuli nuclei inter fibras inveniuntur (Fig. 4. a.)

Hae fibrae singulares, quae a musculosis organicis et secundum constitutionem earum chemicam (quod in sequentibus demonstrabo) et secundum formam distinctae sunt, in omnibus arteriis inveniuntur, et fere semper cum fibris illis tenerrimis elasticis conjunctae. Verum non semper cum solis iis, sicut in arteria femorali et carotide earumque ramis, sed etiam alterantes cum fibris latis elasticis et laminis sic dictis fenestratis praecipue in aorta et truncis ejus majoribus et in arteria pulmonali obveniunt. Quibus in arteriis hae partes elasticae quantitate multo superant fibras contractiles, quare evenit, ut a multis viris doctis, qui a structura aortae in eam arteriarum ceterarum conclusionem fecerunt, fibrae elasticae solae arteriarum tunicis propriae descriptae sint.

Fibrae elasticae latae arteriarum distinctae sunt a fibris elasticis ligamenti nuchae et ligamentorum flavorum columnae vertebralis eo, quod multo magis sunt ramis inter se conjunctae, ita ut nonnunquam formam talis laminae, qualem jam apud fibras longitudinales arteriarum descripsi, praebeant. Quae laminae elasticae magni sunt momenti in tunica media arteriarum majorum. Inveniuntur enim in tunica media earum hae laminae aequalibus intervallis inter se remotae et alterantes cum fibris elasticis ita, ut laminae quasi tubulos alteros in alteros immissos forment, quorum intervalla fibris elasticis repleta sunt.

Quae tunicae mediae compositio dilucide conspici potest, si tenerrima segmenta arteriarum in ea directione persecta, quae recta est in arteriae axem, microscopio investigantur. Laminae elasticae in omnibus segmentis sive longitudinalibus, sive transversis, sive obliquis quasi taeniolae pellucidae, parallelae, semper iisdem intervallis inter se remotae conspiciuntur.

In omnibus iis arteriis, in quibus fibrae elasticae latae in tunica media absunt, sed solae fibrae contractiles et elasticae tenues inveniuntur, etiam laminae elasticae plane absunt in tunica media, nempe in arteriis intestinorum abdominalium, in arteriis iliacis, femorali, poplitaea etc., in carotidum ramis et in universum in omnibus arteriis minoribus.

In fig. 3. tab. II. delineavi partem laminae elasticae ex aortae hominis adulti tunica media, in qua dilucide conspiciuntur residua fibrarum constituentium, quae in eadem directione currunt ac fibrae elasticae circulares partem sinistram laminae obtegentes. Aliam laminam elasticam ex aorta abdominali bovis delineavi in fig. 2. tab. II., in qua nonnullae fibrae elasticae latae e superficie laminae exoriri videntur.

Verum etiam laminae existunt in quibus nullum vestigium fibrarum constituentium conspici potest. Talem e tunica media aortae hominis delineavi in fig. 4. tab. II.

Saepe hae laminae obteguntur a fibris tenerrimis elasticis spatiose ramificatis, quae semper in directionem currunt contrariam ei fibrarum constituentium, (itaque in tunica fibrarum circularium longitudinem versus), et quae arcte conjunctae sunt cum lamina. Tales fibras laminam elasticam obtegentes e tunica media aortae delineavi in fig. 6. tab. II.

Versus externam partem arteriae tunica media paulatim in tunicam externam cellulosam transit, ut loco fibrarum contractilium fibrae cellulosae existant, fibrae elasticae vero et laminae elasticae paulatim diminuantur. Tunicam e solis fibris elasticis compositam, qualem *Henle* inter tunicam fibrarum contractilium et tunicam cellulosam in arteriis majoribus interpositam describit, invenire non potui.

Tela cellulosa arterias circumdans non distinguenda est a tela cellulosa aliorum organorum.

Directio fibrarum in tunica externa partim longitudinalis, partim circularis, partim obliqua est, ita ut hujus tunicae partes difficile in certam directionem abrumpi possint. Aorta thoracica bovis crassitudine hujus tunicae praecipue eminet; in qua fibrae elasticae latae et tenues, contractiles et cellulosae omnibus in directionibus connexae sunt.

Annotationes de Dondersii et Köllikerii commentariis de arteriarum tunicarum structura post hujus opusculi scriptionem, ut in prooemio commemoravi, editis.

Donders[1]) in descriptione structurae arteriarum nonnullis in momentis ab antecessoribus dissentit. Tres distinguit arteriarum tunicas, elastico-conjunctivam (externam), elastico-muscularem (mediam) et tunicam stratam elasticam (intimam). Quas inter tunicas non finem esse distinctum, sed alteram in alteram paulatim commutari dicit. In omnibus his tunicis fibras et laminas elasticas inesse, ita vero, ut in externa parte arteriae cum fibris cellulosis commixtae sint, in stratis mediis cum fibris muscularibus, in stratis intimis merae sine aliis fibris commixtis inveniantur.

Tunicam elasticam propriam et tunicam cellulosam externam ab *Henle* distinctas non distinguendas esse demonstrat, quia neque stratum e solis fibris elasticis neque stratum e solis fibris cellulosis compositum adsit. Fibras contractiles in tunica media describit sicut *Henle*, de laminarum elasticarum vero distributione in tunica media, de quibus jam alio loco exposuit[2]), uberius inquisivit. Numeravit eas in sectionibus transversalibus et

1) loc. cit. p. 377 — 402.

2) *Mulder*, Versuch einer physiologischen Chemie. 8. Lief. 1846. p. 644.

invenit in aorta thoracica hominis 45—60 laminas elasticas, in aorta abdominali 20, in arteria iliaca communi 13, in subclavia 15, in carotide communi 23, sed in harum arteriarum ramis et arteriis minoribus nullas. Fibras contractiles proteïnum continere ex eo concludit, quod acido nitrico et ammoniaco allatis flavas eas fieri videat. Attamen ex *Eulenbergi* experimentis concludendum esse putat, eas fibras substantiis in coctione gluten dantibus esse annumerandas. Cujus conclusionis errorem in sequenti capite demonstrabo.

De tunica intima in eo cum *Henle* dissentit, quod laminam fenestratam non intimam ab epithelio solo obtectam esse credat, sed plura strata tenerrime fibrosa intima inveniat, quibus epithelium impositum sit, quae strata simili modo describit ut nos in antecedentibus. De epithelio arteriarum vero sententiam proposuit *Donders* abhorrentem ab ea, quam viri docti de epitheliis in universum et arteriarum in specie proposuerunt usque ad hoc tempus. Epithelium enim non e cellulis formatum sed e dissolutis partibus membranae intimae exortum esse putat, quae paulatim in membranam pellucidam transformetur et tum in epithelium dissolvatur. Re vera arteriarum epithelium, quod attinet ad primam ejus formationem, distinguendum esse videtur a ceteris epitheliis, verum talem ejus originem, qualem *Donders* proponit, nunquam fieri sed epithelium e veris cellulis exoriri, sicut in antecedentibus exposui, persuasum habeo.

Kölliker in commentario de fibris musculosis organicis[1]) fibras arteriarum contractiles directe annumerat fibris musculosis organicis, et, quod ad formam, nullum esse dicrimen inter ambo genera fibrarum et descriptione et delineationibus exponit. Quod sit discrimen inter haec fibrarum genera secundum meas observationes in antecedentibus quoad formam jam exposui, et quod ad constitutionem chemicam, in capite sequenti exponam.

Praeterea in eo dissentit *Koelliker* a nonnullis scriptoribus recentioribus, quod telae cellulosae magnam copiam cum fibris

1) loc. cit. p. 79.

contractilibus in tunica media arteriarum commixtam esse putet, ita ut omnes fibrae elasticae in hac tunica ad telam cellulosam adnumerandae et pro „Kernfasern von Bindegewebe“ habendae sint. Plurimi scriptores etiam *Henle* et *Donders* nullam telam cellulosam in tunica media arteriarum inesse affirmant, et mihi quoque modo in externis stratis tunicae mediae telae cellulosae sic dictae fibrillae inesse videbantur. Repetitis vero nuper disquisitionibus etiam in regionibus profundioribus tunicae mediae diversarum arteriarum fibras cellulosas observavi.

III. De constitutione chemica tunicarum arterias formantium.

Quae viri docti de constitutione chemica arteriarum investigarunt haec sunt:

Berzelius[1]) dicit de tunica media: „Beim Trocknen verliert diese Haut wenig Wasser, wird dabei dunkelbraun-gelb oder zuweilen schwarz, hart und spröde, nimmt aber im Wasser ihr voriges Ansehn und Elasticität wieder an.“ ..... „Die faserige Haut der Arterien ist in Wasser ganz unlöslich. Auch nach mehrstündigem Kochen darinn bleibt sie unverändert, das Wasser löst Nichts auf, und wird nicht von Galläpfelinfusion getrübt. Dies gilt jedoch nur für ein kürzeres Kochen, wird es mehrere Tage lang fortgesetzt, so erleidet sie nach Versuchen von *J. Müller* eine Veränderung in der Zusammensetzung, und das Wasser zieht eine leimartige Substanz aus. Mit concentrirter Essigsäure übergossen wird diese Haut weder erweicht noch aufgelöst, und auch in kochender verdünnter Essigsäure ist sie unlöslich.“

Porro nullum inesse fibrinum nec ullam proteïnum continentem substantiam *Berzelius* dicit, et haec addit:

„Von dem Muskelgewebe unterscheidet sie sich durch den vollkommenen Mangel an Reizbarkeit, indem sie weder durch electrische noch chemische noch mechanische Reizung sich zu-

1) Lehrbuch der Chemie. 3. Aufl. 1840. Bd. IX. p. 111.

sammenzieht; in physischer Hinsicht durch die Trockenheit und Elasticität ihres Gewebes, während sich die Muskelfaser in erweichtem und erschlafftem Zustande befinden; und in chemischer Hinsicht durch ihr von dem Fibrin so ganz abweichendes chemisches Verhalten z. B. ihre leichte Löslichkeit in Salpetersäure."

Eulenberg[1]) fibras arteriarum elasticas sicut eas aliorum organorum in coctione gluten dare demonstravit, quod gluten coctione perlonga paratum *J. Müller*[2]) descripsit. In capite de fibris elasticis uberius de hoc glutine disseram.

Valentin[3]) e solutione tunicae mediae arteriarum in acido acetico per kalium ferrocyanatum substantiam quandam praecipitavit, eodem modo *Retzius*[4]), quod praecipitatum a substantia proteïnica exortum esse, necesse est concludere.

Simon[5]) addit, solutionem tunicae mediae arteriarum, coctione cum aqua per decem horas paratam, acido acetico admixto praecipitatam esse, quod praecipitatum, in acidi acetici majore quantitate rursus solutum, kalio ferrocyanato, hydrargyro bichlorato et aceto plumbico denuo praecipitatum sit.

Denique *Donders*[6]) commemorat, in tunica media arteriarum acido nitrico concentrato et ammoniaco allato, ammoniacum xanthoproteïnicum exortum esse, quod colore ejus flavo facile cognoscere possumus.

Quas disquisitiones virorum doctorum non sufficere ad cognitionem constitutionis chemicae arteriarum, facile quisque intelliget. Quare eas repetendas, amplificandas et augendas mihi proposui, et praecipue ita ut singulas arteriarum substantias disjunctas inquisierim de earum natura accuratiora me proferre operam navavi.

1) De tela elastica. Diss. inaug. Berol. 1836.
2) *Poggendorff's* Ann. 1836. Bd. 38. p. 311.
3) *Müller's* Archiv. 1838. p. 199.
4) *Müller's* Physiologie 4. Aufl. 1844. Th. I. p. 171.
5) Medicinische Chemie Bd. II. p. 522.
6) Holländische Beiträge. Bd. I. p. 67.

Arterias ad disquisitiones sequentes consumtas fere omnes e bovibus recens mactatis excidi. Nam semper eas recentes et integras adipisci potui, id quod in arteriis ex hominum cadaveribus ademtis non semper contigit.

Primum de substantiis aqua extrahendis ex arteriarum tunicis disseram, tum de fibrarum contractilium substantiis in aqua non solutis exponam, denique de fibris elasticis non solum arteriarum sed etiam aliorum organorum quaedam proferam.

1. De substantiis aqua extrahendis ex arteriarum tunicis.

Fibrae recentes tunicae mediae carotidis bovis lotae sunt in aqua destillata primum frigida tum calida temperaturam 40°C. non excedente. Extractum aquosum alcalinam habuit reactionem, coctione non coagulatum nec turbidum factum est, acidi acetici quantitate perparva admixta praecipitatum praebuit largum, quod in majore quantitate acidi ejusdem solubile fuit, per kalium ferrocyanatum vero denuo praecipitatum est. Aqua a praecipitato per acidum aceticum effecto filtrata, vix acida, coctione turbida facta est ita, ut flocculi albidi albuminis coagulati exstarent, quo facto aqua ab his flocculis filtrata kalio ferrocyanato admixto non commutata est.

Minima adhuc pars substantiarum organicarum et salium evaporatione apparuit.

Praecipitatum acido acetico effectum in extracto aquoso fibrarum carotidis caseïnum esse, eo verisimile factum est, quod ab acidi acetici majore quantitate denuo solveretur, e solutione acetica per kalium ferrocyanatum rursus praecipitaretur. Ceterae reactiones hujus solutionis et in acida et in sales et in tunicam mucosam ventriculi vituli, qua ad coagulandum caseïni lactis utimur, hoc probaverunt. Nam in parte quadam hujus extracti aquosi fibrarum carotidis, saccharo lactis admixto et

tunicae mucosae ventriculi vituli parte imposita [1]) magna exstitit septem horis praeteritis copia coaguli, quod adhaesit tunicae ventriculi.

In altera ejusdem extracti parte, cui solum saccharum lactis admixtum est, demum quatuordecim horis praeteritis coagulatio incepit, eo quod saccharum lactis in acidum lacticum commutata est.

Ex omnibus his reactionibus et ex ea quoque, quod in extracto aquoso evaporante cuticula oriatur sicut in omnibus solutionibus caseinum continentibus fit, satis elucet, caseïnum inesse in extracto.

A globulino (caseïno sanguinis secundum *Simon*) in eo differt hoc caseïnum, quod coctione non coagulatur.

Sicut in carotide ita etiam in aliis arteriis bovis non excepta aorta in tunica media caseïnum inveni, nec non in iisdem omnibus arteriis hominis quantitatem haud levem reperi. Id vero praecipue annotandum est, quod in iis arteriis, quae fere e solis fibris contractilibus formatae sunt, multo major inventa sit copia caseïni quam in iis, in quibus fibrae elasticae praevalent, sicut ex analysibus quantitativis sequentibus elucet.

Etiam et tunica cellulosa arteriarum caseïnum aqua extrahitur, vero multo minor quantitas quam e tunica media.

De quantitate substantiarum arterias conformantium haec experimenta feci:

1) Duas portiones fibrarum circularium tunicae mediae carotidis bovis pependi alteram ad exsiccandum alteram ad extrahendum cum aqua.

Ab altera portione 0,430 grm. post exsiccationem in temperatura 120° C restiterunt 0,132 grm. sicca = 30,69 p. C.

Alteram portionem 0,530 grm. aqua frigida et calida extraxi. Fibrae in filtro penso collectae tam diu aqua lotae sunt, quam

1) Secundum *Simon* tunica mucosa ventriculi vituli in solis iis caseïnum continentibus liquoribus coagulationem efficit, in quibus saccharum lactis inest.

diu quaedam ex iis solvebantur. Tum cum filtro exsiccatae sunt et pensae. Inveni 0,100 grm. fibrarum extractarum exsiccatarum.

0,530 grm. fibrarum recentium ad hoc experimentum sumseram. Quae 0,530 continent, sicut ex experimento antecedenti concludendum est, 0,162 grm. substantiae siccae Ex his 0,100 grm. in aqua insolubilia sunt, et 0,062 solubilia. Id est: centum partes fibrarum exsiccatarum continent 38,27 partes substantiarum in aqua solubilium.

2) Ex fibrarum alius carotidis 0,185 grm. restiterunt post exsiccationem 0,053 grm. = 28,65 p. C.

Ejusdem carotidis fibrarum 0,552 grm. aqua sicut antea lota sunt. Restiterunt fibrarum exsiccatarum 0,095 grm. Quum 0,185 grm. fibrarum recentium 0,053 grm. substantiae siccae contineant, 0,552 grm. fibrarum recentium 0,157 grm. substantiae siccae continent. Quibus ex 0,157 grm. sunt in aqua insolubilia 0,095 et solubilia 0,062. Itaque centum partes fibrarum siccarum continent 39,49 partes in aquae solubiles.

3) Eadem experimenta repetii cum carotide bovis tertii. Substantiae siccae fibrarum circularium inveni 27,14 p. C. et ex 100 partibus hujus substantiae siccae 36,80 partes in aqua solubiles fuerunt.

Quo in experimento fibras sola aqua frigida non calida extraxi, quam ob rem quantitatem substantiarum in aqua solubilium minorem quam in experimentis antecedentibus inveni.

Ut quantitatem caseïni in aqua solubilis definiam, extracto in experimento primo parato acidi acetici guttam admiscui, postquam evaporatione quantitatem extracti paululum diminui. Quo facto reactio extracti acida facta est et caseïnum omne praecipitatum. In filtro penso collegi hoc caseinum, exsiccavi et pependi, 0,034 grm. caseïni inerant seu 20,98 p. C. tunicae mediae exsiccatae. Itaque ex 38,27 partibus in aqua solubilibus tunicae mediae carotidis 20,98 partes e caseïno constiterunt.

Extractum aquosum a caseïno filtratum coctione, ut jam supra commemoravi, turbidum fit, eo quod albumen coagulatur. Ut quan-

titatem albuminis definiam, idem extractum, ex quo caseïnum praecipitavi et filtravi, evaporatione diminui, coxi et ab albumine coagulato filtravi, 0,012 grm. albuminis inveni, id est 7,40 p. C. substantiae siccae fibrarum.

Extractum ab albumine filtratum evaporatum est. Continuit 0,016 grm. substantiarum siccarum, quarum 0,004 sales anorganici, igne non volatiles fuerunt. 100 partes igitur fibrarum exsiccatarum continuerunt 2,46 partes salium et 7,43 partes substantiae organicae in aqua solubilis praeter caseïnum et albumen.

Quae substantia organica, ut accuratius definiretur, majore quantitate parata est et sequenti modo tractata. Aether ne minimam quidem partem adipis ex ea solvit. Spiritus rectificatissimus extraxit partem perparvam substantiae cujusdam jus carnis olentis. Quae restiterunt in aqua soluta sunt praeter nonnullos flocculos, qui, quum acido acetico solverentur et kalio ferrocyanato e solutione praecipitarentur, e substantia proteïnum continente constiterunt. Fortasse albuminis seu caseïni residuum erat. Solutio aquosa a flocculis filtrata brunneum habuit colorem, neque acido acetico neque kalio ferrocyanato admixto praecipitatum in ea exortum est, verum acido tannico, plumbo acetico, hydrargyro oxydulato nitrico, argento nitrico allatis praecipitata exstiterunt.

De natura hujus substantiae extractivae uberius inquirere non potui propter minorem ejus quantitatem.

Ad omnia experimenta, quae descripsi, tunicam mediam carotidis bovis adhibui, quae fere e solis fibris contractilibus constat. Firmior tunica media aortae, in qua fibrae elasticae fere solae insunt, multo minorem quantitatem substantiarum in aqua solubilium continet.

Cui probando haec experimenta instituï:

621 grm. fibrarum circularium aortae bovis exsiccata sunt. Restiterunt 0,522 grm. = 32,20 p. C.

1,095 grm. fibrarum ejusdem partis aortae thoracicae aqua extracta sunt. Residuum siccum fibrarum erat ponderis 0,272 grm. A centum partibus fibrarum exsiccatarum aortae igitur

23,13 partes in aqua solutae sunt. Carotidis fibrae circulares continuerunt 38,27 p. C. substantiarum in aqua solubilium.

In extracto aquoso fibrarum aortae inveni albuminis quantitatem fere eadem proportione ad caseïnum ut in fibris carotidis.

Denique ut partium anorganicarum quantitatem in fibris arteriarum definirem, fibrarum circularium carotidis bovis exsiccatarum 0,848 grm. combussi. Restiterunt cineris 0,030 grm., quorum 0,020 in aqua solubilia e chlori, acidi phosphorici et sulphurici conjuctionibus cum natro constiterunt, cetera 0,010 grm. e calcaria phosphorica et carbonica.

Resultata analysis sub Nr. 1 descriptae adhuc componam:

In 100 partibus fibrarum recentium carotidis inerant:

Aquae partes	69,31
Substantiae siccae	30,69
	100,00

100 partes substantiae exsiccatae continent:

Fibrarum partes in aqua non solubiles . .	60,66
Caseïni	20,98
Albuminis	7,40
Massa e compluribus substantiis extractivis organicis composita	7,43
Salium in aqua solubilium . . .	2,46
Salium in aqua insolubilium . .	1,07
	100,00

2. De fibrarum contractilium substantiis in aqua non solubilibus.

Tunica media carotidis bovis, quae a fibris elasticis et fibris contractilibus formatur, aqua destillata primum frigida tum calida extracta est. Cujus tunicae extractae altera pars cum acido acetico longum per tempus coquebatur, altera cum liquore kali hydrici diluto temperatura 50° C. per quadrantem horae digerebatur.

In acido acetico de fibris filtrato kalium ferrocyanatum copiosum effecit praecipitatum, quod e proteïnum continente substantia exortum est. In liquore kali hydrici de fibris filtrato, acido acetico admixto, magna pars ejus substantiae praecipitata est, quam secundum *Mulderi* auctoritatem proteïnum nominamus. Illud praecipitatum enim, postquam filtratum et cum acido nitrico concentrato commixtum et calefactum est, colorem prae se tulit flavum, qui ammoniaco allato etiam multo distinctior exstitit, eo quod ammoniacum xanthoproteïnicum effectum erat.

Fibrae elasticae contractilibus admixtae in his liquoribus non solutae erant, a fibris contractilibus nullum restitit vestigium, quod microscopium demonstravit.

Fibrae tunicae mediae alius carotidis bovinae aqua saepius lavi, tum cum aqua coxi. Decem horis praeteritis aqua a fibris filtrata et acido acetico et kalio ferrocyanato admixto praecipitatum praebuit, quod a substantia proteïnum continente exortum est. Fibrae lotae denuo cum aqua coctae sunt et decima quaque hora aqua ad disquisitionem transfusa easdem praebuit reactiones nempe quod attinet ad proteïnum. Praeterea glutinis copiam perparvam continuit aqua. Postquam per horas octoginta fibrae coctae sunt, sed magna earum pars restitit, et aqua filtrata adhuc substantiam proteïnicam continuit, finem feci coctionis.

Elucet ex his experimentis, inesse in tunica media carotidis substantias proteïnum continentes praeter eas, quas aqua haud fervens ad extrahendum adhibita solvit. Quum vero duo modo genera fibrarum in hac tunica inessent, elasticae et contractiles, contractiles vero solae per acidum aceticum et kali hydricum solutae essent in experimentis supra descriptis, praeterea fibrae elasticae, ut in sequenti capite demonstrabo, e substantia gluten dante constarent; nullum dubium esse potest, quin fibris contractilibus hoc proteïnum variis experimentis demonstratum attribuendum sit. Quo accedit, ut et forma et reactionibus sub microscopio animadversis magna sit demonstrata similitudo inter

fibras contractiles arteriarum et musculosas organicas, quas musculosas e substantia proteïnum dante constare nemini non persuasum est. Sed praeter formarum differentias magnum adhuc discrimen est in eo, quod fibrae arteriarum contractiles magnam caseïni copiam continent, quod in fibris musculosis organicis omnino deest.

Verum non solum in carotide sed omnibus in arteriis et hominis et bovis tales fibras proteïnum continentes inesse, id quod jam e microscopica investigatione verisimile factum, disquisitione chemica probatum est. Aortam contra et truncos maximos arteriarum multo minorem quantitatem harum fibrarum continere, quam arterias minores (brachialis, femoralis, carotidis partes superiores, earumque ramos) etiam e chemicis meis experimentis sat liquet.

Quae sit compositio elementaris harum fibrarum proteïnicarum in arteriis, analysi elementari inquirere non potui; nam fibrae contractiles ab elasticis, quibus commixtae sunt, non ita disjungi possunt sicut opus est ad analysin elementarem.

Talem vero analysin elementarem, qualem *Scherer*[1]) cum fibris tunicae mediae aortae variis commixtis instituit, repetere nolui.

Sed hoc loco pauca de caseïno in arteriis contento addam. Usque ad hoc tempus in nulla alia parte organismi humani seu animalis sani, quam in lacte, caseïni copia tam magna inventa est. Verum substantia caseïno lactis valde similis format pro maxima parte lentem crystallinam et corpuscula sanguinis. Quae substantia, globulinum nominata, num sit propria, a caseïno lactis plane diversa[2]), an caseïni modificatio, commixtione cum substantiis aliis effecta[3]), nondum certe constat. Equidem plures hominis et animalium partes inquisivi, ut caseïnum ex iis aqua extraham, nempe musculos striatos et laeves, telam cel-

1) *Liebig.* Annal. d. Pharm. Bd. 40. p. 1.
2) Quod *Berzelius* et *Lehmann* putant.
3) Ex *Simonis* sententia.

lulosam nonnullarum partium, ligamentum nuchae vituli et bovis, sed, etiamsi minimam caseïni copiam in ligamento nuchae et in tela cellulosa inveni, tamen nunquam copiam, quae sit comparabilis cum ea in arteriis. Venarum vero tunicae, quae, quod ad structuram, arteriis maxime similes sunt (nam a fibris elasticis, cellulosis et contractilibus formantur sicut arteriae) etiam caseïni copiam ei in arteriis similem vero minorem continent, quae eodem modo, ut ex arteriis, aqua extrahi potest.

Facile quis opinari posset, caseïnum arteriarum et venarum esse nil aliud, nisi globulinum sanguinis corpusculorum liquefactorum tunicis vasorum infiltratum. Verum non verisimilis mihi videtur haec caseïni origo, quia in sanguinis corpusculis globulinum tam arcte conjunctum est cum haematino rubro, ut chemicis reagentiis ab eo prorsus disjungi non possit, in vasorum tunicis verum nunquam ne minima quidem pars haematini cum caseïno conjunctum insit. Equidem persuasum habeo, hoc caseïnum esse proprium fibris contractilibus vasorum eodem fortasse modo, quo globubinum fibris lentis crystallinae; quae sententia praecipue eo fulta esse mihi videtur, quod in iis arteriis, quae maximam fibrarum contractilium copiam continent, etiam caseïni maxima copia inventa est, in aorta vero, et in venis, quae omnes multo plures fibras elasticas et cellulosas continent, quam contractiles, caseïni copia multo inest minor.

3. De fibris elasticis in universum et arteriarum in specie.

Fibras elasticas coquendo in gluten commutari *Eulenberg*[1] demonstravit. *J. Müller*[2] de hoc glutine haec dicit: „Der Leim vom elastischen Gewebe nähert sich mehr dem Knorpelleim als dem gewöhnlichen Leim. Er wird nämlich von essigsaurem Blei getrübt, von Essigsäure sehr stark getrübt, von Zusatz von Alaun oder schwefelsaurer Thonerde wird er gefällt

1) De tela elastica. Diss. inaug. Berol. 1836.
2) *Poggendorff's* Ann. 1836. Bd. 38. p. 311.

und von schwefelsaurem Eisenoxyd kaum getrübt. Der Niederschlag von schwefelsaurer Thonerde löste sich in überschüssiger schwefelsaurer Thonerde nicht."

Scriptores recentiores nil novi de hoc glutine proferunt. *Mulder* et *Donders*[1] vero Eulenbergii observationes de commutatione fibrarum elasticarum in gluten falsas esse dicunt, easque a telis coquendo gluten dantibus excipiunt.

Infra demonstrabo, fibras elasticas coquendo longum per tempus protracto praecipue in temperatura 100°C. excedente in gluten commutari, sicut *Eulenberg* recte observavit. Analysi elementari fibrarum elasticarum ligamenti nuchae bovis instituta *Tilanus*[2] invenit.

C	55,65
H	7,41
N	17,74
O	19,20

Quibus omnibus ex numeris hanc construxit formulam quam analysi elementari conjunctis telae elasticae cum chloro probatam invenit: $C_{52}H_{80}N_{14}O_{14}$.

Admodum difficile est naturam glutinis telae elasticae eodem modo distincte definire, quo cetera glutinis genera. Nam post coctionem demum multo longiorem, quam qua ad parandum gluten comune et chondrinum utimur, fibrarum elasticarum prima glutinis vestigia invenimus in decocto, et majorem hujus glutinis quantitatem solum ita parare possumus, ut fibras cum aqua temperaturam 100° C longe excedente coquamus. Verum notum est, gluten ossium et chondrinum coctione perlonga praecipue in temperatura 100°C excedente ita commutari, ut plane alias praebeant reactiones. Idem fieri videtur in coctione fibrarum elasticarum; nam decocta breviore coctione parata alias prae se ferunt reactiones ac decocta earundem fibrarum diutius cocta. Qua ex re multae reactionum varietates exstant, de quibus uberius dicendum erit.

1) *Mulder*, Versuch einer phys. Chemie. Lieferung 7. p. 594.
2) Ebendas. p. 595.

Aliud impedimentum accurate definiendi glutinis telae elasticae est, quod cum fibris elasticis multae fibrae proteïnicae et cellulosae commixtae sint, ex quo fit, ut in solutione glutinis fibrarum elasticarum semper quantitas haud levis substantiae proteinicae et glutinis communis inveniatur, quae vix plane demoveri potest.

Experimenta, quae institui ad definiendum fibrarum elasticarum gluten haec sunt:

Ligamenti nuchae bovis particulae parvae aqua destillata frigida et calida tam diu lotae sunt, quam diu aqua substantias solubiles extraxit. Tum cum aqua coctae. Horis sex praeteritis decoctum transfusum parvam continuit copiam proteïni et glutinis comunis. Fibrae tum lotae denuo coctae sunt per septem horas, quo tempore praeterito decoctum transfusum nullum praebuit proteïnum sed parvam glutinis comunis quantitatem, quod gelatinavit, acido tannico et chromico, alcohole, tinctura jodi, aqua chlori et hydrargyro bichlorato praecipitatum est. Fibrae eaedem lotae et per horas septuaginta sex coctae sunt. Parva substantiae organicae copia inerat in decocto; fibrae microscopio suppositae integrae et non commutatae videbantur. Solutio concentrata non gelatinavit in refrigerando. Has praebuit reactiones: Praecipitatum exstitit admixtis: acido tannico, chromico et oxalico, tinctura jodi, plumbo acetico, cupro sulphurico, ferro oxydulato sulphurico, argento nitrico, hydrargyro oxydulato nitrico.

Nullum praecipitatum admixtis acido acetico, kalio ferrocyanato et ferrocyanico, alumine, hydrargyro bichlorato, ferro sulphurico oxydato, ferro acetico oxydato, alcohole, aqua chlori.

Fibrae denuo lotae per horas triginta quatuor coctae sunt. Decoctum concentratum, subfuscum, gluten olens, non gelatinavit. Has praebuit reactiones: Praecipitatum exstitit cum acido tannico, chromico, oxalico, tinctura jodi, plumbo acetico, cupro sulphurico, ferro oxydulato sulphurico, argento nitrico, hydrargyro oxydulato nitrico, alumine, ferro oxydato sulphurico, ferro oxydato acetico, nullum praecipitatum cum acido acetico, kalio

ferrocyanato et cyanico, hydrargyro bichlorato, aqua chlori, alcohole. Quamquam igitur fibrae elasticae per centum et viginti tres horas coctae essent, tamen minima tantum pars fibrarum elasticarum in gluten commutata erat. Itaque alia methodus, qua celerius gluten pararetur, applicata est. Obtemperans consilio praeceptoris mei dilectissimi Cel. Schulze, qui in elaboratione tota hac chemica consiliis et apparatu magna benignitate me adjuvit, fibras elasticas una cum aqua in tubulos vitreos liquando hermetici clausos inclusi, quos tum in cista aënea, in qua temperatura 150 — 160° C erat, imposui. Quibus in tubulis solutionem fibrarum elasticarum perfectam adeptus sum.

Fibrarum earum, quae jam per horas centum et viginti tres coctae sunt, nonnullas in tubulum vitreum inclusi et modo supra descripto tractavi.

Viginti horis transactis omnes fibrae solutae erant. Residuum parvum e calcaria carbonica et phosphorica constitit. Solutio colore fusco non gelatinavit in refrigerando, verum gluten oluit, et acido tannico, hydrargyro oxydulato nitrico, argento nitrico, acido chromico praecipitata est.

Acidi acetici guttula admixta perparvum exstitit praecipitatum, quod in majore acidi ejusdem quantitate rursus solutum sed kalio ferrocyanato denuo praecipitatum est. Plumbum aceticum et alumen solutioni admixtum eam paululum turbidam effecit. Reagentibus ceteris, quae supra adhibui, nihil in hac solutione mutatum est.

Idem experimentum, quod cum fibris ligamenti nuchae factum modo descripsi, cum fibris tunicae mediae aortae thoracicae bovis institui. Decoctum primum post horas tredecim transfusum magnam continuit copiam glutinis communis et substantiae proteïnicae. Eodem modo in omnibus decoctis postea transfusis magna inerat copia proteïni, gluten vero mox alias praebuit reactiones ac gluten commune, et simile erat glutini e fibris igamenti nuchae. Postquam e decocto substantia proteïnica acido acetico praecipitata est et filtrata, gluten has praebuit reactiones: Praecipitatum est acido tannico et chromico, tinctura

jodi, hydrargyro bichlorato, argento nitrico, ferro oxydulato sulphurico. Nullum praecipitatum exstitit alumine, plumbo acetico, hydrargyro oxydulato nitrico, ferro oxydulato sulphurico et acetico, alcohole, aqua chlori, cupro sulphurico, acido oxalico.

Fibrae jam per horas septuaginta septem coctae in tubulum vitreum cum aqua inclusae sunt, et per triginta horas temperaturae 160°C. expositae. Non omnes fibrae solutae erant. Permagna copia proteïni in solutione subfusca, gluten olente, inerat, sed nonnisi parum glutinis per acidum tannicum praecipitati.

Quod proteïnum cum glutine fibrarum elasticarum commixtum, ut jam ante coctionem e fibris elasticis removerem, fibras ligamenti nuchae et aortae bovis aqua extractas liquori kali hydrici diluto immiscui, et per quartam partem horae in temperatura 60°C digessi. Qua digestione fibras contractiles arteriarum, quae proteïnum continent, et cellulosas, quae coctae gluten commune dant, solvi. Tum fibras elasticas solas, quae restiterunt, aqua lotas coxi. Quum post coctionem per sexaginta horas vix glutinis aliquantulum e fibris solutum esset, eas in tubulos vitreos inclusi, et modo supra descripto tractavi. Triginta horis praeteritis fibrae elasticae ligamenti nuchae et aortae plane solutae erant. Liquor subfuscus non gelatinavit in refrigerando et has praebuit reactiones. Praecipitatus est acido tannico et picrinico nitrico, omnibus vero ceteris reagentiis supra adhibitis nullo modo commutatus est. In solutione fibrarum elasticarum ligamenti nuchae acidum chromicum, hydrargyrum bichloratum et tinctura jodi minimam praecipitati copiam effecerunt, quod in solutione fibrarum elasticarum aortae non exstitit.

Acidum aceticum et kalium ferrocyanatum in ambobus solutionibus minimam adhuc proteïni copiam demonstraverunt.

Ex omnibus his experimentis, ad quae amplificanda et augenda tempus adhuc defecit, haec sequuntur.

1) Gluten e fibris elasticis modo coctione perlonga parari potest. Ut fibrae plane in gluten commutentur, temperatura aquae 100°C. longe excedens adhibenda est.

2) Reactiones, quas gluten fibrarum elasticarum prae se fert, diversae sunt secundum tempus, per quod coctum est. Id enim gluten eodem modo commutari videtur coctione perlonga, quo gluten ossium et chondrinum. Quare definitio accurata reactionum glutinis e fibris elasticis valde difficilis est.

3) Praecipitata, quae creberrime apparuerunt, efficiuntur in glutine fibrarum elasticarum acido tannico et picrinico-nitrico, acido chromico et tinctura jodi. Propinquum igitur esse videtur gluten fibrarum elasticarum magis glutini ossium quam chondrino.

4) Praecipitatum, quod *J. Müller* in glutine fibrarum elasticarum acido acetico et alumine effectum esse dicit, a substantiis proteïnicis commixtis exortum esse mihi videtur. Nam solum iis admixtis hoc praecipitatum efficere potui, quod non e glutine formatum erat sed e substantiis proteïnicis.

5) Gluten fibrarum elasticarum merum modo tum parari potest, quum e fibris elasticis omnes fibrae cellulosae et proteïnicae semper eis admixtae remotae sunt, id quod digestione fibrarum elasticarum cum liquore kali hydrici effici potest.

IV. De viribus vitalibus in arteriis.

Vires vitales arteriarum spectant primum ad earum formationem in embryonibus, in partibus reproducendis et in partibus morbis exortis, tum ad ipsarum partiumque finitimarum nutritionem, tum ad secretionem et resorptionem, denique ad contractilitatem atque expansibilitatem seu vim in sanguinis motum et in pulsum. Quae quatuor genera actionum vitalium tractabo.

I. De prima arteriarum formatione.

Vires in prima formatione vasorum agentes descripturus non redeo ad observationes *Guill. Hunteri*[1]), *J. Hunteri*[2]), *J. F.*

1) Anatomia uteri humani gravidi. London 1774.
2) Versuche über das Blut, übers. v. *Hebenstreit* 1797.

Meckel[1]), *Pander*[2]), *Rathke*[3]), *v. Baer*[4]), et aliorum, sed viro sat praeclaro in Embryologia, *Bischofio*[5]), nitor, qui primus in mammalium foetibus cordis et vasorum originem accuratius investigavit, et in libris, quos de cuniculi et canis ovorum evolutione composuit, observationes suas edidit. In cuniculi ovo decem dierum, quod a ventris latere contemplatus est, in pariete anteriore partis superioris embryonis canalem vidit parum tortum, qui vix determinatus erat a partibus finitimis. Qui canalis primum est rudimentum cordis. Pars ejus superior in capite evanescit, inferior, in duos ramos partita, in vesiculam germinativam abit. Eodem tempore prima conspexit vestigia venae terminalis, et retis vasculosi inter hanc venam et ramos inferiores canalis cordis. Persuasum habet *Bischof*, uno eodemque tempore oriri cor et partem vasorum peripheriam et ambo serius formari quam primordia corporis foetus et systematis nervosi. Quomodo vero oriantur vasa, in primis an recta sit sententia *Schwannii*, vasa formari e cellulis stellatis, id conspicere non potuit. Fortiter vero opponit sententiae a *Doellinger* propositae et a *Reichert*[6]) defensae, vasa esse nil aliud, nisi vias, quas sanguinis corpuscula a corde propulsata in tela adhuc molli sibi fodant. Dicit de *Reicherto* ejusque sententia: „Er hat dabei wohl vergessen, dass, wenn das Terrain, durch welches solche Bahnen gebrochen werden sollen, zu dieser Zeit auch allerdings sehr zart und locker ist, die Kraft, welche diese Bahnung bewirken soll, auch noch sehr schwach ist. Noch mehr aber widerspricht die constante Richtung dieser Blutbah-

1) Handbuch der menschl. Anatomie. 1815.
2) *Christ. Pander*, dissertatio sistens histor. metamorphoseos, quam ovum incubatum prioribus quinque diebus subit. Würzburg 1817.
3) Untersuchungen über die Bildung und Entwickelung des Blennius viriparus und des Flusskrebses.
4) Epist. de ovi mammal. et hom. genesi. Leipzig 1827.
5) Entwickelungsgeschichte des Kaninchencies 1842. p. 120.
6) Entwickelungslehre pag. 143.

nen, die in so vielen Tausenden von Embryonen immer dieselbe ist, dieser Ansicht und zeigt im Gegentheil, dass sie durch ein bestimmteres und sichereres Gesetz gegeben wird, als durch die richtungslose Stosskraft des Herzens.“ Praeterea contra hanc sententiam et eam, vasa capillaria non habere parietes, argumenta gravia a viris doctis proposita sunt, quae jam in capite de structura arteriarum exposui. (Confer *Burdach*, Physiologie IV. pag. 193. *E. H. Weber* in Hildebrandts Anatomie III. pag. 35. *Joh. Müller* bei *Burdach* IV. pag. 197.)

Valentin[1]) dicit, se in avium ovis vasorum originem dilucide conspexisse, quam hoc modo describit: In strato musculoso tunicae vitellariae loci oriuntur liquidi, qui formam ramosam praebent et inter se conjunguntur retiaque formant. Liquor pellucidus commutatur partim in vasorum parietes partim in globulos minimos, qui intra parietes manent, et postea sanguinis globuli fiunt. Cor, cujus originem in ovo incubato *Valentin* simili modo describit, quo *Bischof* ejus primordia in mammalibus, cum his vasorum parietibus sensim conjungitur, et corde mox pulsante circulatio incipit. Quae circulatio, ut ad *Bischofii* descriptionem[2]) revertar, in cuniculi embryone decem dierum hoc modo exstat. E parte superiore canalis cordis duo vasa emanant, arcus aortarum nominata, quae post brevem decursum in truncum aortae conjunguntur. Hic statim iterum sejungitur in duo vasa a *v. Baer* arteriae vertebrales nominata, quae cum vena seu sinu terminali per arterias omphalomesentericas cohaerent, ex quo sinu sanguis per venas omphalomesentericas in partem inferiorem cordis revertitur. Ad hanc arteriarum embryonalium vitam pertinet etiam arteriarum et venarum branchialium et ductus arteriosi formatio et mutatio, ita ut vasa branchialia in embryone humano et reliquorum animalium vertebratorum pulmonibus spirantium omnino evanescant,

1) Entwickelungsgeschichte pag. 287. ff.
2) l. c. pag. 120—122. pag. 152.

ductus arteriosus vero in animalibus calidi sanguinis in ligamentum arteriosum transformetur.

De origine vasorum capillarium in embryonibus et in partibus aut reproductione aut formatione morbosa exortis valde dissentiunt viri docti. Jam supra dixi secundum *Schwannii* sententiam vasa capillaria exoriri e cellulis stellatis, quae inter se conjungantur retiaque forment. Contra hanc sententiam *Platner* [1]) novissimis temporibus dicit: „Bekanntlich hat *Schwann* die Ansicht aufgestellt, dass die Capillaren sich aus Zellen entwickelten, deren sternförmige Fortsätze mit einander verwüchsen. Bei dieser Ansicht stützte sich *Schwann* auf Beobachtungen an den Schwänzen von Froschlarven, wo er helle sternförmige Zellen zwischen den Capillargefässen fand, von denen er glaubte, dass sie sich zu Capillargefässen zu einander verbänden. *Schwann* hat jedoch selbst niemals die wirkliche Bildung von Capillaren aus diesen Zellen beobachtet, auch entging ihm nicht, dass diese Zellen an Froschlarven von jedem Alter vorkommen, während sie doch, wenn seine Ansicht richtig war, bei den älteren entweder gänzlich fehlen, oder nur selten hätten vorkommen müssen. Ich habe im vergangenen Sommer diesem Gegenstande meine Aufmerksamkeit geschenkt und kann auf das bestimmteste versichern, dass sich aus den sternförmigen Zellen niemals Capillargefässe bilden. Nach den Untersuchungen, die ich nicht nur an Froschlarven, sondern namentlich auch an jungen Tritonen von 1—1½ Centimeter Länge anstellte, scheinen überhaupt Capillargefässe unabhängig von bereits vorhandenen Gefässen gar nicht zu entstehen, sondern jedes neue Gefäss ist die Fortsetzung eines bereits vorhandenen, was, wie ich später ersah, auch *Prevost* und *Lebert* beobachteten. Untersucht man die Schwänze von jungen Tritonen, so findet man sehr bald Capillaren, die plötzlich stumpf enden, wie ein Sack. Das Gefäss hört völlig geschlossen auf, und man findet nicht die Spur einer Fortsetzung. An manchen

1) *Müller's* Archiv 1844. pag. 525.

bemerkt man jedoch an dieser Stelle einen langen ganz dünnen Ausläufer, der sich unmerklich verliert, und an anderen sieht man, wie zwei solche Ausläufer sich zu einem gemeinschaftlichen Bogen vereinigen, und wie dieser Bogen allmählig an Durchmesser zunimmt. Dieser Bogen wird unzweifelhaft zu einer neuen Capillargefässschlinge.“

Observationes de hac re valde accurate a *Platnero* institutae videntur; praeterea ejus sententia de origine vasorum capillarium tam accommodata est naturae, ut *Schwannii* sententiam de cellulis stellatis fere devictam habere possimus.

Post Platnerum explicationem recentiorem de vasorum capillarium origine singularem inveni ab *Engel* datam [1]), qui non plane cum Platnero consentit. Dicit enim: „Die Kapillargefässe entwickeln sich nicht als ein Netz von ineinander mündenden Röhren, sondern man bemerkt nur einfache von einem grösseren Gefässe nach verschiedenen Richtungen auslaufende Schläuche, die, indem sie allmälig feiner werden, endlich in der Zellenmasse des Embryonentheils sich verlieren. Diese Gefässschläuche entstehen aus spindelförmigen Zellen, die mit ihren fadenförmigen Fortsätzen auf einander stossen und sich verbinden. Diese fadenförmigen Fortsätze sind natürlich anfangs so fein, dass sie zur Aufnahme von Blutkörperchen sich durchaus nicht eignen, auch wenn man annehmen möchte, dass sie hohl seien.... Bei weiterer Ausbildung werden diese fadenförmigen Verlängerungen allmälig von dem einen Ende her breiter, bis sie den Durchmesser erreichen, den die Zelle in der Gegend ihres Kerns darbietet, worauf dann letzterer an der äusseren Fläche zu sitzen scheint, in der That aber zwischen zwei äusserst feinen Lagen der Gefässhaut eingeschlossen ist. Indem nach und nach Kapillaren, welche von verschiedenen Stämmchen ausgehen, mit ihren fadigen Fortsätzen auf einanderstossen, und sich verbinden, entsteht ein Netz von Kapillaren. Den Ansatz von spindelförmi-

1) Beitrag zur Anatomie der Gefässe. Zeitschrift für die Gesellschaft der Aerzte zu Wien 1847. pag. 153.

gen Zellen an bereits gebildeten Gefässstämmchen bemerkt man in der Regel dort, wo in der Wand des Gefässes ein Kern liegt. Dort erscheint die Gefässwand in eine kleine Spitze ausgezogen, an die sich die spindeltörmige Zelle mit ihrem Faden anlegt, während die Erweiterung oder Durchgängigmachung dieses Fadens von dem breiteren Theile der Zelle allmählig gegen die beiden Enden hin beginnt und fortschreitet."

Quum ipse nullas observationes de hac re facere potuissem, non dijudicare possum de his variis sententiis.

2. De arteriarum vi in nutritionem.

Sanguis arteriosus seu oxygenio praeditus est fluidum nutriens toti corpori, nam ei insunt elementa ad nutritionem omnium organorum. Per parietes vero arteriarum separatur is sanguis a partibus nutriendis. Qui igitur parietes maximi momenti sint in nutritionem necesse est. Sententiae virorum doctorum de hac vi hae sunt:

Nunnulli in primis *Döllinger*[1]) et *Dutrochet*[2]) credunt, nutritionem talem esse, ut sanguinis globuli ex arteriis evadant, et in parenchymate finitimo assimilentur. Nutritionem esse dicunt aggregationem et juxtapositionem materiarum in sanguine jam conformatarum, id quod observatione microscopica confirmatum habebant. Nam sanguinis globulos ex arteriis evadentes et partibus circumdantibus se applicantes et conjungentes se vidisse dicunt. *Dutrochet*[3]) dicit de hac re: „Entre les courbures, que forment les vaisseaux, il existe un tissu fort transparent, dans lequel on distingue beaucoup de granulations de la grosseur des globules sanguins; or, en observant les mouvements du sang j'ai vu plusieurs fois un globule seul s'echapper lateralement du vaisseau sanguin et se mouvoir dans le tissu transparent, avec

1) Was ist Absonderung? etc. Würzburg 1819.
2) Recherches sur l'accroissement et la reproduction des vegetaux. Mem. du Mus. d'hist. naturelle Ann. 4. Tom. 7. pag. 214.
3) Eodem loco.

une lenteur, qui contrastait fortement avec la rapidité du torrent circulatoire, dont ce globule était echappé; bientôt après le globule cessait de se mouvoir, et il demeurait fixé dans le tissu transparent; or, en le comparant aux granulations, que contenait ce même tissu, il était facile de voir, qu'il n'en différait en rien, en sorte qu'il n'était pas douteux, que ces granulations demi-transparentes ne fussent aussi des globules sanguins precédement fixés. Par quelle voie ces globules sortent ils du torrent circulatoire? C'est ce qui n'est pas facile de determiner."

Huic sententiae obstat, quod nullus virorum doctorum recentiorum aliquid hujus modi cum microscopiis maxime excultis videre potuit. Tum perforatae essent arteriae foraminibus, ex quibus sanguinis globuli exire possent, necesse esset. Verum haec foramina nullo modo demonstrare possumus, nec in capillaribus animalium vivorum, nec injectione. Porro qua ratione nutritio varia variorum organorum eo effici potest, quod sanguinis globuli, qui omnibus in partibus systematis arteriosi unam eandemque habent formam, aggregentur? Nisi fortasse fingere volumus, esse tot varia genera sanguinis globulorum, quot organa in corpore, eaque esse praedita tali vi et quasi ingenio, ut semper aptos et convenientes sanguinis globulos sibi eliciant. Naturae repugnantem esse talem explicationem satis apparet.

Prevost et *Dumas* et *M. Edwards* hanc habent sententiam de nutritione. Nucleos sanguinis globulorum esse elementa omnium organorum, eorumque egressu ex arteriis et assimilatione nutritionem effici. Observasse sese putaverunt texturam omnium organorum granulosam, granaque paria esse nucleis globulorum sanguinis. Iisdem argumentis, quibus sententiae antecedenti, etiam huic repugnamus, cui accedit, quod nemo unquam vidit, nucleos e globulis et vasis egressos telae finitimae assimilatos esse.

Vasa serosa hoc loco commemoranda sunt, quae ad nutritionem maximam habere vim dicunt nonnulli viri docti. Jam

Boerhave[1]), *Vieussens*[2]), *Hewson*[3]), *Bichat*[4]), *Döllinger*[5]) vasa descripserunt, quae tam esseut subtilia, ut sanguinis globulos excipere non posssent, sed sero solo impleta essent. Postea saepe sunt negata[6]), verum novissimis temporibus, microscopiis maxime excultis, a multis viris doctis observata sunt.

Praeceptor *Ill. Schultze*[7]) vasa serosa in cute injectione subtili demonstravit. *R. Wagner*[8]) in ranis vidit vasa serosa, de quibus dicit: „Sind es vielleicht obliterirende Gefässe, Ueberbleibsel der ersten Bildung des peripherischen Gefässsystems in Embryo?“

Similia vasa describit *Platner*[9]). *C. H. Schultz*[10]) dicit de his vasis, quae plastica nominat: „Zwischen diesen (den Kapillargefässen) finden sich aber Gefässe von grösserer Feinheit, in welche keine Bläschen dringen, weil ihre Mündung enger ist, als der Durchmesser der Bläschen. Diese Gefässe scheinen leer zu stehen, wenn man sie im Schattenlichte beobachtet; aber bei Beleuchtung mit hellerem Lichte zeigen sie sich von farblosem Plasma ohne Bläschen durchströmt. Wir wollen diese Gefässe plastische Gefässe, (vasa plastica) nennen, wegen ihres Gehaltes und weil sie nicht zur serösen Sekretion, sondern vielmehr zur Ernährung bestimmt sind.“

Inprimis propter verba ultima hunc locum attuli. Maximi esset momenti hoc factum, si affirmaretur. Nunc vero nondum credere possumus, illa vasa plastica nutritioni soli servire. Verisimile est, sicut vasa capillaria ita etiam serosa inservire secretioni, resorptioni et nutritioni.

1) De usu ratocinii mechanici pag. 14.
2) Novum vasorum systema pag. 112.
3) Description of the Lymphatic system. London 1774. pag. 223.
4) Anatomie générale Tom. II. Paris 1818. pag. 1—7..
5) Was ist Absonderung? etc. Würzburg 1819.
6) *Mascagni, Spallanzani, Oesterreicher.*
7) *Müller's* Archiv 1834 Heft I. pag. 30.
8) Physiologie, 3. Aufl. 1845. pag. 167.
9) *Müller's* Archiv 1844 pag. 525.
10) System der Circulation p. 169.

J. Müller[1]) dicit de vasis serosis: „Wenn wir nun behaupten, dass selbst in den durchsichtigeren Häuten noch Blut führende Gefässe vorhanden sind, so ist damit nicht zugleich erwiesen, dass alle Gefässe dieser Theile wirklich auch so stark sind, dass sie die rothen Blutkörperchen aufnehmen; Im Gegentheile ist es wahrscheinlich, dass grade in diesen Theilen die meisten zarten Gefasse nur den flüssigen Theil des Blutes, liquor sanguinis, aufnehmen. Und auch in andern Theilen mag es so feine Kapillargefässe geben, welche für gewöhnlich nur liquor sanguinis aufnehmen und in die Venen abgeben (vasa serosa).

Quomodo vero in his vasis serosis et in vasis capillaribus nutritio fiat, quae in primis sit vis arteriarum in hanc nutritionem, quoad explicare possumus, brevibus exponam.

Vasorum capillarium parietes imbibunt serum sanguinis, quod continet soluta elementa omnia ad nutritionem. Partim ea vi fit, quam endosmosin et exosmosin nominamus, partim chemica quadam et organica attractione. Quo facto partes organi vasis finitimae elementa ad nutritionem necessaria extrahunt, sibi assimilant vita peculiari, nervorum organicorum ope et auxilio.

Primum igitur momentum nutritionis est ea vasorum capillarium natura, ex qua penetrari possint a liquore sanguinis, secundum est connexus virium physicalium chemicarum et vitalium in organis nutriendis, quae substantias ad nutritionem aptas sibi vindicant. Verum etiam in eas organorum partes, quae non sunt in contactu proximo cum vasis capillaribus, permanat serum, iisque quae sunt necessaria ad nutritionem importat.

3. De arteriarum vi in secretionem.

Tres praecipue sunt sententiae, quas viri docti proposuerunt de statu arteriarum in organis secernentibus.

Prima a *Malpighio*[2]), qui primus de glandularum structura subtiliore inquisivit, proposita haec est, in glandulis vasa

1) Physiologie des Menschen. Bd. I., 4te Auflage 1844. pag. 175.
2) Exercitationes de structura viscerum 1665.

finibus clausis sejunctas esse a ductibus secernentibus, nec ullam esse communicationem inter vasa sanguinea et canales glandulae proprios. Secunda a *Ruysch*[1]) proposita, arterias in glandulis apertas habere communicationes cum canalibus secernentibus, et continuis parietibus in eos transire, sententiam Malpighianam fere devicit, et a viris multis in physiologia sat praeclaris probata est. Sic ab *Hallero*[2]), qui eam ita excoluit, ut quinque genera vasorum apertorum describeret, scilicet in ductum excretorium glandularum, in telam cellulosam, in cutis superficiem, in cava interna et in vasa lymphatica.

Tertia sententia a *Mascagni*[3]) relata est, qui quidem canalium secernentium initia clausa esse putavit, arterias vero in omnibus locis, ubi secretio fieret, poris peculiaribus perforatas esse dixit, per quos partes sanguinis transmitterentur.

Quibus e sententiis Malpighiana, vasa capillaria clausa in glandulis juxta posita esse canalibus secernentibus clausis sine ulla aperta communicatione, nostro tempore fere ab omnibus physiologis probata est, et microscopio et injectionibus subtilissimis demonstrata.

Jam si quaerimus de vi arteriarum parietum in secretionem, nonnulla proferenda nobis sunt de partibus secretioni inservientibus in universum. Tres partes in secretione concurrentes distinguere possumus: 1) vasa sanguifera, 2) tunicas secernentes cellulis epithelialibus obtectas (sive in formam tubulorum subtilissimorum redactas, sicut in glandulis, sive cava interna obducentes sicut tunicae serosae), 3) substantiam parenchymatosam interpositam inter vasa sanguifera et tunicam secernentem.

Praecipue ad glandulas haec trium partium dispositio quadrat, in tunicis serosis vero non accurate distinguere possumus inter tunicam secernentem propriam et substantiam parenchymatosam, cui vasa sanguifera imposita sunt.

1) Thesaurus.
2) Elementa physiologica. Lib. II. §. 23.
3) Geschichte und Beschreibung der einsaugenden Gefässe aus dem Lat. v. Ludwig. 1789.

Quum in omnibus locis secernentibus e sanguine in vasis capillaribus incluso secretum parandum sit, necesse est, per parietes vasorum clausos partes liquidas sanguinis penetrare, ut a ductibus secernentibus excipiantur.

Verum secreta peculiaria non sanguinis constitutione diversa in diversis locis secernentibus efficiuntur, sed ex uno eodemque sanguine arterioso per totum corpus fluente varia secreta parantur, solis exceptis hepate et pulmone, quibus ad parandam bilem et exhalationem pulmonalem per venam portam et arteriam pulmonalem sanguis adducitur venosus peculiaris. Etiam non versimile est, tunicarum vel canalium secernentium forma effici discrimen inter varia secreta, quum in diversis animalibus in glandulis, quod ad formam canalium secernentium valde diversis, tamen idem secretum paratur.

Itaque aut soli substantiae parenchymatosae, inter vasa sanguifera et canales secernentes positae, aut, quod multo est verisimilius, omnibus tribus partibus supra memoratis conjunctis attribuenda est vis peculiaris secernendi. Quibus annumerandi sunt etiam nervi, qui certe haud leve momentum afferunt secretioni.

Eorum vi vasa sanguifera instructa sunt facultate solis quibusdam partibus sanguinis transitum concedendi, diversis in diversis locis secernentibus. Nam si omnes sanguinis partes liquidae per parietes vasorum capillarium sicut per tunicas ad filtrandum instructas penetrare possent, quid fieret ex iis substantiis, quae a canalibus secretoriis non exceptae sunt, e fibrino, ex albumine, quae fere in nullo secreto inveniuntur? Num nutritioni glandularum inserviunt, ut nonnulli viri docti affirmant? Non verisimile est propter immensam earum copiam. Itaque eam persuasum habeo esse vim in parietibus arteriarum in glandulis et aliis locis secernentibus, quod pro peculari horum locorum functione praecipue nervorum vi modificanda solis quibusdam sanguinis partibus transitum concedant, quae tum a parenchymate et ductibus secretoriis exceptae aut modificatae aut non mutatae excernantur.

Quae partes vero praecipue in hanc modificationem sub-

stantiarum e sanguine egressarum vim habeant, inprimis num sint in glandulis cellulae ductus secretorios obtegentes sic dictae epitheliales, id dijudicare hujus opusculi fines non concedunt.

4. De arteriarum contractilitate et expansibilitate, vi in sanguinis motum et in pulsum.

Arteriarum parietes elasticitate permagna sunt praediti.

Elucet haec elasticitas ex unoquoque experimento, quo elasticitatem corporis cujusdam inquirere solemus. Dubitari non potest, quin haec elasticitas etiam in corpore vivo vim habeat in sanguinis motum et arteriarum pulsum.

Praeter hanc elasticitatem num arteriae sint praeditae vitali quadam contractilitate et expansibilitate valde dissenserunt viri docti, et nostro quoque tempore dissentiunt. Virorum, qui in rebus physiologicis maxime excelluerunt, sententias breviter perlustrabo.

Negant quidem contractilitatem et expansibilitatem vitalem earumque vim in sanguinis circulationem sed cor et arteriarum elasticitatem causam unicam sanguinis motus esse dicunt: *Bichat*[1]), *Nysten*[2]), *Treviranus*[3]), *Rudolphi*[4]), *Carus*[5]), *Döllinger*[6]). *Richerand*[7]), *Oesterreicher*[8]) (qui motum quendam arteriarum quidem concedit, irritabilitatem vero plane negat,)

1) Anatomie générale, Paris 1818 Tom. I. pag. 261, 290, 298, 304, 305.
2) Recherches de physiologie et pathologie chimiques, Paris 1811 pag. 304, 314, 322.
3) Biologie, Bd. IV. pag. 262, 654.
4) Physiologie Th. I. pag. 88 und 248. Th. II. p. 328.
5) *Meckel's* Archiv Bd. IV. p. 419.
6) *Meckel's* Archiv Bd. II., pag. 256—258.
7) *Meckel's* Archiv Bd. IV. pag. 224.
8) Versuch einer Darstellung der Lehre vom Kreislauf. Nürnberg 1826. p. 70, 162, 170.

Magendie[1]), *Wedemeyer*[2]), *J. Müller*[3]), *Berzelius*[4]), *Hamernjk*[5]), *Mendelssohn*[6]) *et alii.*

Sententiam vero contrariam, multis experimentis affirmatam proposuerunt: *Haller*[7]), *Zimmermann*[8]), *Verschuir*[9]), *John Hunter*[10]), *Sennac*[11]), *Soemmering*[12]), *Osiander*[13]), *Everhard Home*[14]), *Cuvier*[15]), *Beclard*[16]), *J. F. Meckel*[17]), *Parry*[18]), *Thomson*[19]), *Kreisig*[20]), *Hastings*[21]), *Wilson Philip*[22]), *Bostock*[23]), *C. A. S. Schultze*[24]), *E. H. Weber*[25]), *Arnold*[26]), *Henle*[27]), *Grabau*[28]), *Valentin*[29]).

1) Physiologie, übersetzt von *Hofacker*. 1826. Bd. II. pag. 216.
2) Untersuchungen über den Kreislauf 1828. p. 95.
3) In den älteren Auflagen seiner Physiologie.
4) Lehrbuch der Chemie, Dresden 1840 pag. 112.
5) Physiologisch-pathologische Untersuchungen. Prag 1847. p. XI.
6) Der Mechanismus der Respiration und Circulation. Berlin 1845.
7) Elementa physiologiae 1757. I. pag. 70.
8) De irritabilitate. Goettingae 1751.
9) De vi venarum et arteriarum irritabili 1766.
10) Versuche über das Blut etc. 1797. I. p. 235.
11) Traité de la structure de coeur p. 227.
12) Gefässlehre 1762. p. 60.
13) Handbuch der Entbindungskunst. Tübingen 1819. Bd. I. pag. 588. Bd. II. p. 262.
14) *Meckel's* Archiv Bd. III. pag. 139.
15) Vergl. Anatomie, übers. v. *J. F. Meckel* IV. pag. 7 und 13.
16) Anatomie générale 1823 pag. 329.
17) Anatomie I. 1815 pag. 188.
18) Untersuchungen über den arteriellen Puls übersetzt v. *Embden* 1827.
19) Ueber Entzündung übersetzt v. *Krukenberg* 1820 p. 96.
20) Krankh. des Herzens III. p. 246.
21) De vi contractili vasorum Edinburgh 1818. Auszug v. D. Heusinger in Mekel's Archiv VI. p. 224.
22) On symptomatic fevers. London 1820 p. 20.
23) An elementary system of physiology. Vol. I. London 1824. pag. 402, 403.
24) Lehrb. der vergleichd. Anatomie I. 1828 pag. 116.
25) *Hildebrandt's* Anatomie 1831. III. p. 75.
26) Physiologie 1837. II. pag. 358.
27) Allgemeine Anatomie 1841. pag. 512. ff.
28) Die vitale Theorie des Kreislaufs 1841.
29) Physiologie. Bd. I. 1844. p. 455.

Inter viros in rebus physiologicis praeclaros recentiores nonnulli[1]) contenderunt, arteriarum fibras contractiles quidem esse irritationibus mechanicis et vitalibus allatis, verum in sanguinis motum et circulationem hanc contractilitatem nullam habere vim. Verba horum virorum ipsa referam.

R. Wagner dicit[2]): „Die Triebfedern des Kreislaufes sind in der Reihe der organischen Wesen verschieden. Es sind 1, Herzen oder pulsatorische Organe, beim Menschen, den Wirbelthieren und vielen Wirbellosen, welche an verschiedenen Stellen ins Gefässsystem eingeschoben sind ..... 2, Wimpern, welche an der inneren Gefässwand sitzen und den Saft weiterbewegen. Polypen, Medusen, Würmer. Alle früheren Annahmen, wonach der Kreislauf des Blutes bei den höheren Thieren und beim Menschen noch weiter vermittelt werden soll, als: durch Contractilität der Arterien, Anziehung der Organe u. s. w sind rein hypothetisch."

Contra loco alio dicit[3]): „1. Die Arterien besitzen eine natürliche und zwar beträchtliche Elasticität, welche unabhängig von ihren Lebenseigenschaften sich auch im Tode noch erhält. 2. Die Arterien besitzen eine lebendige Contractilität, welche vom Nervensystem abhängig ist."

Nescio, qua ratione appareat haec contractilitas, nisi in sanguinis motum vim habeat. Si arteria, vitalibus irritationibus allatis, contrahi potest, nonne irritatione sanguinis, arteriarum tunicas magna vi extendentis, contrahi necesse est?

Similem sententiam profert *J. Müller*, qui quum Henle fibras contractiles in arteriis accuratius descripsisset, dixit[4]): „Die Arterien und überhaupt die Blutgefässe besitzen ausser der Elasticität auch eine lebendige Zusammenziehungskraft. .... Von welchem Gewebe diese langsam wirkende, organische Contractilität herrühre, war bisher unbekannt. *Henle* hat in den

1) *R. Wagner* et *J. Müller* et alii.
2) Lehrbuch der Physiologie 3. Aufl. 1845. p. 163.
3) Loco citato pag. 450.
4) Physiologie 4. Aufl. 1844. Bd. I. pag. 162.

Wänden der Arterien eine eigenthümliche Schicht entdeckt, welche offenbar als der Sitz dieser Eigenschaft angesehen werden muss."

Quamquam, ut ex his locis apparet, *J. Müller* contractilitatem organicam in arteriis inesse affirmat, tamen non credit, hanc contractilitatem ullam habere vim in circulationem et propulsationem sanguinis. Dicit enim[1]):

„Aus allen diesen Thatsachen folgt, dass rhytmische Muskularcontractionen der Arterien durchaus nicht bei dem Kreislaufe wirken, und dass die Verminderung des Durchmessers der Arterien nach der Ausdehnung durch den Impuls des Blutes Folge ihrer Elasticität ist." Eodem modo:[2]) „Diese Bewegungen (des Blutes in abgeschnittenen Theilen) entstehen bloss durch das Ausfliessen des Blutes, während die Gefässe durch die Elasticität einen engeren Durchmesser annehmen, als sie vorher im Zustande gewaltsamer Ausdehnung hatten."

Nihilominus ad explicandam inanitatem arteriarum post mortem contractilitate utitur[3]): „Die vitale Contractilität scheint auch an dem sogenannten Leersein der Arterien Antheil zu haben. Die Arterien ziehen sich beim Sterbenden theils durch ihre Elasticität, theils durch ihre organische Contractilität bis auf das Minimum ihres Lumens zusammen."

Transeo nunc ad experimenta virorum nonnullorum doctorum, qui contractionem et expansionem arteriarum vitalem demonstraverunt et confirmaverunt.

John Hunter[4]) arteriam canis e partibus finitimis praeparavit, ita ut nuda jaceret, quo facto eam persecuit; paullatim arteria ita sese contraxit, ut cavum plane evanesceret. Quam contractionem non elasticitate effectam esse eo affirmavit, quod

1) Eodem loco pag. 170.
2) Loco citato pag. 177.
3) Loco citato pag. 171.
4) Versuche über das Blut etc. übersetzt v. *Uebenstreit* 1797. I. pag. 234.

post mortem canis arteriam expanderet, quo facto non in tantum contractionis gradum reversa est, sed aperta mansit.

Arterias canum, carotides, femorales, crurales nudas persecuit; sanguis magna vi prorupit, vero mox arteriarum lumina ita angusta facta sunt contractione, ut sanguis ex arteria modo exstillaret.

De tempore, per quod arteriae ex corpore excisae contractilitate vitali praeditae sint, *John Hunter* hoc fecit experimentum ingeniosum[1]): Funiculum umbilicalem infantis neonati tali modo persecuit inter duas ligaturas, ut sanguis in placenta remaneret, quo facto mox placenta sanguine impleta ex utero evenit. Post duodecim horas, ligatura secunda in funiculo umbilicali apposita, eum persecuit inter ambas ligaturas. Lumina arteriarum fuerunt valde aperta, verum post horas viginti quatuor ita erant contracta, ut lumen totum evanesceret. Eodem die denuo partem funiculi umbilicalis abscidit et viginti quatuor horis praeteritis eodem modo quo antea lumina contracta erant. Tum experimento denuo repetito nulla contractilitatis phaenomena apparuerunt. Per duos igitur dies in arteriis funiculi umbilicalis contractilitas vitalis remansit.

C. Hastings[2]) multis experimentis arteriarum contractilitatem ita demonstravit, ut, qui experimenta repeteret, nunquam contractilitatem vitalem negare posset. Nonnulla experimenta describam:

Arteriam femoralem felis nudam radendo cum scalpello irritavit, quo facto in loco irritato sese contraxit, tanquam filo constricta. Quum locis pluribus eam irritaret, omnibus in his locis contractione annulari affecta est, quae contractiones demum, quarta parte horae transacta, cesserunt.

Chemicis irritamentis allatis in vasa capillaria cutis natatoriae ranarum contractiones vidit magnas, quas ante eum jam

1) Loco citato pag. 236.
2) De vi contractili vasorum. Edinburgh 1818. Auszug v. *Hessinger* in *Meckels* Archiv. Bd. VI. 1820. pag. 224.

Zimmermann[1]) *Verschuir*[2]) *Thomson*[3]) descripserant. Quae experimenta minus gravia sunt ad contractiones vitales affirmandas, quia exempli gratia acidis fortibus arteriae prorsus mortuae contrahuntur.

Aliud vero experimentum ab *Hastings* descriptum hic commemorandum est, quia postea ab *Schwann*[4]) repetitum et proclamatum magnam commovit admirationem, id dico, quod frigoris irritamento vasa capillaria dilucide contrahuntur. Ranae pedem in aqua calida posuit, quo facto omnes arteriae valde dilatatae sunt. Glacie tum apposita in pedem, subito arteriae contractae sunt, quod microscopio accurate observavit. Alio in experimento glaciem attulit in ranae pedem et contractionem arteriarum observavit. Breve post tempus arteriae dilatatae sunt, sanguis haesit et coagulare coepit. Tum oleum Terebinthinae in hunc locum inflammatum fusit, quo facto fere omnia vasa denuo contracta sunt et sanguinis motus restitutus.

Nonne elucet ex hoc experimento, sanguinis circulationem per vasa capillaria ex eorum contractilitate pendere, neque ea remota fieri posse? Nam quum frigore vasorum capillarium tunicae primum ad nimiam contractionem irritatae, ideoque deinde paralysi affectae et dilatatae essent, motus sanguinis in iis haesit, et demum contractitate restituta rediit.

Experimenta de arteriarum irritabilitate a praeceptore Ill. *Schultze* jam ante plures et viginti annos facta descripta sunt a *Baumgaertner*[5]), ex quibus nonnulla referam: „Bei einem lebenden Schafe wurde die Carotis an zwei Stellen unterbunden und dann zwischen den unterbundenen Stellen angestochen, das Blut spritzte heraus, und die Arterie entleerte sich völlig, was aus der Elasticität der Arterien nicht erklärt werden kann. An demselben Thiere wurde die Carotis der andern Seite an zwei

1) De irritabilitate. Goettingae 1761.
2) De vi arteriarum et venarum irritabili. 1766.
3) Ueber Entzündung, übers. v. *Kruckenberg* 1820. pag. 96.
4) Berliner Encyclopädie, Artikel: Gefässe pag. 229.
5) Ueber die Natur und Behandlung der Fieber. Frankf. 1847 p. 71ff.

Stellen unterbunden, das unterbundene Stück wurde heraus geschnitten und um das Gerinnen des darin befindlichen Blutes zu verhindern, häufig bewegt. Nach einer Stunde wurde die Arterie angestochen, das Blut war flüssig, aber es wurde nur sehr wenig durch die Elasticität der Wände heraus getrieben.

Bei einem Schafe wurde die linke Carotis blos gelegt, sie zeigte gar keine Bewegung; der vorher durch die allgemeine Bedeckung und Muskeln hindurch fühlbare Puls, war jetzt weder durch den Finger noch das Auge zu erkennen, selbst wenn die Arterie durch einen untergeschobenen Messerstiel in die Höhe gehoben war, obgleich die davon abgehenden Zweige deutlich pulsirten. Die Ursache dieser Erscheinung war, wie es scheint, ein tonischer Krampf in der Arterie.

Die Carotis und der Nervus vagus eines eben geschlachteten Pferdes wurden blos gelegt, jene mit Silber und dieser, nachdem er durchschnitten war, an dem Ende oberhalb der Durchschneidung mit Zink bewaffnet; bei Schliessung der Kette zeigte sich mehre Male Contraction der Arterie. Dieses ist der merkwürdigste Versuch, der mir über diesen Gegenstand bekannt ist."

Similia experimenta in praelectionibus physiologicis Ill. *Schultze* vidi, quorum id mihi imprimis est in memoria, quod semper arteria ex eo momento, quo aër atmosphaericus eam tetigit, per quinque fere minutas rigida erat, neque digito, neque oculis pulsus percipi poterat, quasi esset in spasmo tonico. Eodem rigore affecta erat, quum in arteriam pulsantem aqua frigida guttatim instillaretur, quo facto subito pulsus cessavit.

Multa experimenta de hac re fecit *Parry*[1]), qui vidit carotidem ovis aëris irritatione sese contrahentem ex $^{225}/_{400}''$ usque ad $^{162}/_{400}''$.

1) Untersuchung über die Natur, Ursache und Verschiedenheit des arteriellen Pulses, übers. v. Embden. Hannover 1847. Exp. 15. 24. 26.

Kaltenbrunner[1]) in experimentis de inflammatione factis et *Schwann*[2]) observationibus microscopicis contractiones et dilatationes vitales arteriarum affirmant.

Experimenta novissima, quae mihi nota sunt de hac re, fecerunt *E. H. Weber* et *Ed. Weber*[3]). Vim galvanismi in contractiones saepe negatam experimentis simplicibus et facilibus stricte demonstraverunt.

Ope apparatus magneto-electrici effecerunt contractiones vasorum mesenterii ranae tales, ut vasa diametro $^1/_7 - ^1/_{17}'''$ usque ad dimidium diametri normalis contraherentur. Verum in vasis diametro majore nullis in animalibus contractiones sat perspicuas efficere potuerunt. Experimenta horum virorum repetii et omnia eodem modo successerunt, ut descripta sunt. Verum etiam in vasis majoribus contractiones evidentes effeci Galvanismo, id quod a permultis viris doctis negatur.[4]) Haec sunt experimenta, quae hac de re feci:

Apparatus, quo utebar est talis, qualem magneto-electricum per inductionem agentem (Magneto-electrischer Inductionsapparat) nominamus; et quo utimur ad paralyses aliosque morbos nervorum medendos.

In cane catulo carotidi communi sinistrae, nudatae, specillo interposito in lucem protractae, flumen electricum attuli per fila metallica in directione fibrarum circularium apposita. Nullas vidi contractiones carotidis. Ante applicationem electricitatis pulsaverat arteria in una minuta ducenties bis et quinquagies, verum postquam flumen electricum breve per tempus per carotidem et interdum etiam per corporis partem permeavit, pulsavit modo centies sexies ter. Ne magna vi sanguinis a corde

1) Exper. circum statum sanguinis et vasorum in inflammatione. Monachii 1826.
2) Berliner Encyclopädie. Artik.: Gefässe.
3) Ueber die Wirkungen, welche magneto-elektrischer Reiz der Blutgefässe bei lebenden Thieren hervorbringt. Müller's Archiv. 1847. pag. 232.
4) Confer Müller's Physiologie 4te Aufl. I. 1840. pag. 170.

propulsi contractiones per electricitatem forsan in arteria producendae impedirentur, carotidis partem pollicem unum et dimidium longam duabus ligaturis inclusi, ne sanguis affluereat. Tum electricitate iterum allata, ut in fibrarum circularium directionem perfluereat, illico arteria se contraxit loco affecto, quasi filo constricta. Omnibus in locis arteriae inter ligaturas sitae hae contractiones effici potuerunt.

Carotidi bovis hora sexta vespertina necati, dimidia hora post mortem praeterita, excisae et ab omnibus partibus finitimis liberatae flumen electricum ita allatum est, ut ambo fila metallica, ductores electricitatis, uni eidemque — ut ita dicam — fibrarum circularium fasciculo apponerentur, et quidem in oppositis carotidis lateribus. Hoc modo electricitate percurrente eandem habuit vim, ac filum constringens. Nam paucas post secundas carotis contractione annullari constricta erat. Unoquoque loco carotidis experimentum eodem eventu repetitum est.

Altera carotis inclusa in adipem et telam cellulosam multam reservata est. Post quatuor horas, post sex horas, et post quatuordecim horas eadem contractio, sed sensim minus arctior electricitate effici potuit, verum postea nullam contractionem animadvertere potui.

Contractionem fibrarum longitudinalium in carotidibus vel aortis animadvertere non potui, dilucide vero in vena jugulari, quae, quum fines ejus partis tres pollices longae, quam ad experimentum adhibui, metalli lamina circumdedissem, et flumen electricium in hos fines attulissem, satis perspicue fere tribus lineis brevior reddita est.

Contractiones arteriarum irritamento mechanico hoc modo effeci: Carotis bovis e corpore excisa dimidia hora post mortem volsella leviter premebatur. Volsella remota arteria loco affecto se contraxit sicut filo constricta. Experimentum saepe repetitum est in bovis, vituli et ovis carotidibus semper eodem eventu. Post quartam fere partem horae rursus dilatatae erant arteriae in locis antea contractis, et contractiones eodem modo repeti potuerunt per tres vel quatuor horas. Tum vero premente volsella non contractio in loco irritato, sed expansio effecta est. Eodem fere gradu, quo prius se contraxerant arteriae, nunc expanderunt, quae expansiones longo tempore praeterito non plane evanuerunt, sed arteria his expansionibus pluribus in locis exortis rugosa mansit.

Quas contractiones et expansiones nunquam in aorta vel trunco cleidocarotico neque in carotidum parte infima observare potui, sed in iis modo arteriis, quae pro maxima parte ex fibris contractilibus formatae sunt, quas supra descripsi.

Qua re illas arterias solas vitali contractilitate et expansibilitate praeditas esse censeo. A musculis vero hae arteriarum fibrae non solum constitutione chemica sed etiam contractionis modo ac ratione distinctae sunt. Musculi enim irritamento allato, imprimis electricitate subito et celeriter contrahuntur, fibrae vero arteriarum tardius electricitate et irritamentis chemicis et mechanicis allatis se contrahunt. Contractilitate vero et expansibilitate vitali esse praeditas, id satis affirmatum mihi videtur.

De pulsu arteriarum.

Pulsus arteriae est vicissitudo expansionis et contractionis, quae a sanguine efficitur propulso in arterias cordis systole. Quare eodem tempore fit, quo cor in systole sanguinem in arterias propellit. In iis modo arteriis, quae longe absunt a corde temporis puncto tardius pulsum observamus. In vasis capillaribus pulsus non adest, quia arteriarum parietum elasticitate et contractilitate sanguis etiam in cordis diastole ita premitur et propellitur, ut motus sanguinis in arteriis majoribus paullatim remittens in motum continuum transeat.

Nonnulli viri docti pulsum non in expansione et contractione sed in flexura arteriae et motu laterali posuerunt. Imprimis viri

Weitbrecht[1]) *Doellinger*[2]), *Parry*[3]), *Rudolphi*[4]), *Jäger*[5]), plane negaverunt expansionem et contractionem arteriarum in pulsu. Hoc experimento, quod jam *Hastings* fecit[6]), et quod vario modo commutari potest, illorum virorum sententia refutatur: Carotis seu alia arteria sat magna animalis cujusdam ita in lucem profertur, ut nulla alia parte obtegatur. Circa arteriam filum ceratum seu alio modo rigidum ponitur. Quod si in cordis systole arteria arcte a filo circumdatur, in cordis diastole tantum apparet spatium inter filum et arteriae parietem, ut facile specillum intermitti possit. Contractionem arteriae post expansionem non sola elasticitate fieri, sed vitali vi, ex eo apparet, quod, si arteria omnino est praedita vitali contractilitate, quae per irritamenta effici potest, etiam sanguinis impulsus tale irritamentum est. Irritationem per impulsum sanguinis, quo fortiter expanditur arteria, sequi debere contractionem vitalem, non possumus negare postquam contractilitas vitalis omnino demonstrata est.

Multa alia phaenomena demonstrant vim arteriarum in pulsum.

Omnes varietates pulsus, quae tantum habent momentum in morborum diagnosin, pulsum durum, mollem, magnum, parvum cet., num explicaremus e sola cordis systole et diastole? Non poterimus. In his pulsus varietatibus inest argumentum maxime eximium ad probandam contractilitatem vitalem arteriarum majorum, sicut varia sanguinis congestione ad organa vario modo irritata motus vitalis in arteriis minimis et vasis capillaribus probatur, de quibus phaenomenis alio loco accuratiorem disquisitionem, arteriarum nervos simul tractantem, mox eruditorum examini propositurum me esse spero.

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1) De circulatione sanguinis.
2) Vom Kreislauf des Bluts, in der Münchener Denkschrift VII. pag. 220.
3) Ueber den arteriellen Puls, übersetzt v. *Embden* 1817. p. 91
4) Grundriss der Physiologie. Berlin 1821, II. Abthl. p. 295.
5) De arteriar. pulsu. 1820. pag. 216.
6) *Meckel's* Archiv. Bd. 6. pag. 224.

Resultata totius operis.

1. Chemicis experimentis demonstratum est, fibras contractiles arteriarum e substantia proteïnica sic dicta constare, a fibris musculosis organicis vero aliorum organorum eo distinctas esse, quod aqua extrahi potest ex iis et hominis et animalium (bovis, vituli, ovis) magna copia substantiae cujusdam, quae omnes caseïni reactiones prae se fert. Praecipue in arteriis, quae prope e solis fibris contractilibus compositae sunt, magna hujus substantiae copia inest, exempli gratia in carotide bovis, in arteria femorali hominis (pag. 21 — 28).

2. Quum in nulla alia corporis parte praeter lac tanta caseïni copia inventa praesertim in nullo genere fibrarum caseïnum hucusque repertum sit, hoc signum praestantissimum fibrarum contractilium arteriarum statuendum est. Affinitatem quandam habent cum fibris lentis crystallinae, quae globulinum, substantiam caseïno valde similem, continent. Microscopica disquisitio quoad formam fibrarum contractilium in arteriis majorem cum musculis organicis similitudinem exhibet (pag. 27).

3. Fibrae elasticae arteriarum sicut omnes fibrae elasticae coctione gluten dant, quod *Mulder* et *Donders* negaverunt, sed solum in aqua temperaturam coctionis longe excedente plane solvuntur. Quae sit natura hujus glutinis non accuratius definire potui, quia ejus reactiones valde variabiles erant. Coctione longa, quae ad solvendum necessaria est, commutatio partialis seu totalis hujus quoque glutinis extare videtur. Verum multo magis glutini ex ossibus simile est, quam chondrino; forsan propria species (pag. 28—33).

4. Tunica excepto epithelio intima arteriarum vulgo dicta non ea est, quam *Henle* fenestratam nominat, sed formatur in arteriis maximis (aorta, arteriis iliacis, subclaviis, carotidibus) compluribus stratis e fibris tenerrimis, longitudinalibus, elasticis compositis, in arteriis minoribus ex uno strato tenerrimo, fibroso (pag. 12).

5. Tunicam hanc intimam circumdant fibrae longitudinales elasticae, latae, quae exceptis arteriis maximis laminas sic dictas fenestratas formant, et plerumque cum tunica intima fibrosa arcte conjunctae sunt (pag. 13.).

6. Omnes laminae fenestratae arteriarum e retibus fibrarum elasticarum exoriuntur, quare eas cum *Dondersio* laminas elasticas nomino (pag. 13. 15).

7. *Reicherti* sententia de transformatione epithelii in membranam fenestratam non probata inventa est (pag. 11.).

8. Tunica vulgo dicta media arteriarum constat e fibris et laminis elasticis commixtis cum fibris contractilibus, quae musculosis organicis similes sunt. Duo sunt genera fibrarum elasticarum in tunica media, latae et tenerae. Fibrae latae una cum laminis elasticis in solis arteriis maximis insunt cum paucis fibris contractilibus conjunctae, tenerae in arteriis minoribus cum multis fibris contractilibus (pag. 14).

9. Laminae elasticae in tunica media ita collocatae sunt, ut quasi tubulos forment, et paribus intervallis invicem remotae alterae ab alteris circumdentur. Quae intervalla fibris expleta sunt (pag. 15.).

10. Contractilitas et expansibilitas vitalis arteriarum majorum jam affirmata a nonnullis viris doctis sed a multis neglecta denuo per irritationes mechanicas et electricas demonstrata est (pag. 51.).

11. Electricitate in machina, quam apparatum inductionis magneto-electricae nominamus, effecta non solum arteriae minores, sicut nuper *E. H. Weber* descripsit, sed etiam majores (carotides canis et bovis) in tantum contractionis gradum redactae sunt, ut quasi filo constrictae videantur (pag. 52.)

12. In corpore vivo haec arteriae majoris contractio modo tum effecta est, quum electricitas applicaretur in arteriae partem duabus ligaturis a sanguinis impulsu munitam (pag. 51.).

13. In carotide e bove recens mactato excisa etiam quatuordecim horis post excisionem contractiones electricitate et irritatione mechanica effici potuerunt, si carotis cum partibus finitimis excisa et ab iis circumdata reservaretur (pag. 52).

15. Definitioni arteriarum usitatae haec praeferenda est: Omnia vasa circulationi sanguinis inservientia, in quibus sanguis a truncis in ramos et ramulos immittitur, arteriae sunt nominanda (pag. 3.).

Explicatio tabularum.

Tab. I. (In pagina 11 et sequent. de omnibus figuris huj. tab. uberius dictum est).

Fig. 1. Epithelium e carotide hominis adulti.
a. Cellulae ovales.
b. Cellulae squamiformes, rarissimae.

Fig. 2. Epithelium e carotide bovis. Cellulae in finibus productae sunt, et fibris appropinquantur.

Fig. 3. Epithelium ex eadem carotide bovis. Fibrae e cellulis exortae in membranam conjunctae sunt.

Fig. 4. Similis membrana e carotide infantis neonati.

Fig. 5. Membrana exorta e cellulis epithelii aortae infantis neonati. Nuclei in longitudinem valde producti sunt.

Fig. 6. Membrana ex eadem aorta. Nuclei fere evanuerunt. Striae tenerrimae exstant in membrana.

Fig. 7. Membrana ex eadem aorta in transformatione ad tunicam intimam. E fibris tenerrimis composita esse videtur Fibrae eo, quod in acido acetico non solvuntur, elasticis tunicae intimae appropinquantur.

Fig 8. Membrana epithelialis ex aorta hominis adulti. Nuclei partim corrugati, partim in fibras producti sunt.

Fig. 9. Tunicae intimae lamina ex aorta hominis adulti.

Fig. 10. Fibrae e tunica intima aortae vituli.

Fig. 11. Fibrae tunicae intimae ex eadem aorta in retis modum ramificatae.

Fig. 12. Fibrae tunicae intimae arteriae femoralis hominis adnatae ad laminam fenestratam.

Tab. II.

Fig. 1. Fibrae elasticae longitudinales ex aorta hominis tunicam intimam sequentes ita ramificatae, ut paene laminam forment (pag. 13.).

Fig. 2. Lamina elastica, cujus e superficie fibrae elasticae exoriuntur, e tunica media aortae abdominalis bovis (pag. 16).

Fig. 3. Lamina elastica in altero latere a fibris elasticis obtecta e tunica media aortae hominis adulti. Dilucide apparet, fibras laminam constituentes eadem in directione currere ut fibrae circulares laminam obtegentes (pag. 16).

Fig. 4. Lamina elastica sine ullo vestigio fibrarum constituentium. Nonnullae breves fibrae tenerrimae cum lamina arcte conjunctae sunt (pag. 16).

Fig. 5. Lamina elastica e tunica fibrarum longitudinalium arteriae iliacae communis hominis. Fibrae elasticae latae laminam constituentes in directione a litera *b* ad *b* sitae sunt, rete e fibris tenerrimis elasticis in directione contraria cum lamina conjunctum est (pag. 13).

Fig. 6. Lamina elastica e tunica media aortae hominis. Fibrarum constituentium lineae evanuerunt, rete fibrarum tenerarum laminae adnatum est (pag. 16).

Tab. III.

In pag 14 et 15 uberius de omnibus his figuris dictum est.

Fig. 1. Fibrae contractiles commixtae cum fibris elasticis teneris ex arteria femorali hominis.

Fig. 2. Partes fibrarum contractilium, in quibus nonnullis acido acetico allato nucleus distincte elucet.

Fig. 3. Fibrae contractiles commixtae cum elasticis teneris e carotide bovis.

Fig. 4.

a. Nuclei e fibris contractilibus singuli natantes in aqua, in qua fibrae contritae sunt.

b. c. Fibrae novellae, cellulaeformes cum nucleis distinctis.

d. Partes fibrarum longiorum terendo deletarum.

Fig. 5. Partes fibrarum contractilium et fibrae contractiles breves commixtae cum fibris elasticis latis e tunica media aortae hominis.

Tab. I.

Fig. 1.

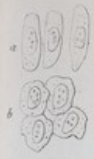


Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.

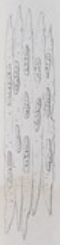


Fig. 6.



Fig. 7.



Fig. 8.



Fig. 9.



Fig. 10.



Fig. 12.



Fig. 11.



Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.



Tab. III

Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Zur

Entwicklungsgeschichte

der

Oxalurie.

Ein dritter Beitrag

zur

physiologischen Heilkunde.

Von

Dr. Friedrich Wilhelm Beneke.

Göttingen,
bei Vandenhoeck und Ruprecht.
1852.

Herrn

Hofrath Dr. Carl Friedr. Theod. Krause, M. D.

Professor der Anatomie und Physiologie,

Mitglied der königl. ärztlichen Prüfungsbehörde und Landphysikus zu Hannover,

Mitglied der kaiserl. Leop. Carol. Akademie der Naturforscher und der naturforschenden und ärztlichen Gesellschaften zu Erlangen, Göttingen, Halle, Hannover, Wien u. a. m.

hochachtungsvoll gewidmet

vom

Verfasser.

Sucht man die einzelnen Erscheinungen eines gegebenen Krankheitszustandes durch ihre verschiedenen Entwicklungsstufen hindurch zu verfolgen und auf ihre letzten Ursachen zurückzuführen, so geht man damit über die Anforderungen der Aetiologie hinaus. Die Lehre von den näheren und entfernteren Ursachen wird zu einer Lehre von der Entwicklung, zu einer Entwicklungsgeschichte des Krankheitszustandes erweitert. Macht aber ein jeder Krankheitszustand, bevor er uns zu Gesichte kommt, seine bald längere, bald kürzere Reihe von Entwicklungsstufen durch, reiht sich nur langsam Symptom an Symptom, bis endlich einmal das Krankheitsbild als ein mehr oder weniger vollendetes dasteht, so wird die Darstellung einer Entwicklungsgeschichte für jeden einzelnen Fall eine nur um so dringendere Aufgabe, als damit allein richtige Anschauungen über das Wesen und die Bedeutung der einzelnen Erscheinungen selbst, als damit allein Anhaltspunkte für eine rationelle Therapie gewonnen werden können. — Ich habe es in dem vorliegenden Schriftchen versucht, die einzelnen und verschiedenartigen Entwicklungsstufen einer einzelnen, oftmaligen Krankheitserscheinung darzustellen, und aus dem obigen Grunde demselben nicht den Titel eines Beitrages „zur Aetiologie", sondern „zur Entwicklungsgeschichte" der Oralurie gegeben.

Im Verlaufe auch dieser Abhandlung werden die Verhältnisse der anorganischen zu den organischen Blutbestandtheilen als besonders wichtig hervortreten. Die Oxalurie kann auf Mißverhältnissen der stickstoffhaltigen, auf Mißverhältnissen der stickstofffreien, aber eben sowohl auch auf Mißverhältnissen der anorganischen Blutbestandtheile beruhen. Möchte ich in dem Bestreben die hohe Wichtigkeit der letztern gebührend hervorzuheben den treffenden Worten Liebig's einen Beweis der Anerkennung gegeben haben, daß „die Entwicklung des Gesetzes der Abhängigkeit der Funktionen und Beschaffenheit des Blutes von der Natur und Quantität der unverbrennlichen Bestandtheile desselben, den untersten Stein der Grundlage der Medicin und Physiologie bildet, und daß es vollkommen thöricht ist, vor der Legung dieses Steins an eine rationelle Heilwissenschaft nur zu denken."

Es steht die vorliegende kleine Arbeit in engster Beziehung zu meinem ersten und zweiten Beitrage zur physiologischen Heilkunde. Ich übergebe sie der Oeffentlichkeit in der Hoffnung auf eine gleich nachsichtige Beurtheilung, wie sie die letztern beiden gefunden haben. — Die Thatsachen, welche ich mittheilte, sind zuverlässig und wahr; ob ich ihnen die richtige Deutung gab, ob ich richtige Schlüsse für die Therapie daraus zog, das überlasse ich dem reifern Urtheile Anderer.

Hannover d. 2. März 1852.

Mit dem Ausdruck „Oxalurie" wird passend eine Erscheinung bezeichnet, welche die verschiedenartigsten Krankheitsprocesse begleitet. Das Wort selbst bedarf keiner weitern Erklärung. Enthält der Urin Oxalsäure (oxalsauren Kalk), so nimmt eben die Oxalurie einen Platz in der Symptomatik des Krankheitszustandes ein. — Wenn es nun aber wahr ist, daß der phosphorsaure Kalk ein nothwendiges Requisit für die Bildung der thierischen Zelle abgiebt, wenn es ferner wahr ist, daß die Oxalsäure als das Lösungsmittel für den phosphorsauren Kalk betrachtet werden muß und eine retardirte Metamorphose derselben nicht nur Oxalurie, sondern zugleich eine abnorme Ausscheidung des Kalkes und damit eine Abnahme des Zellenbildungsprocesses bedingt, so ist die Frage nach den Ursachen dieser Retardation der Metamorphose und also der Oxalurie selbstverständlich für Pathologie und Therapie eine überaus wichtige. — Eine genügende Reihe von Beobachtungen läßt mich an der Richtigkeit der beiden obigen Behauptungen nicht zweifeln *); um so mehr war es daher Aufgabe, der bezeichneten Frage die vollste Aufmerksamkeit zuzuwenden. Die Darlegung der Resultate, zu welchen ich dabei gelangte, ist der Zweck der folgenden Blätter.

Schon in meinem zweiten Beitr. z. physiol. Heilk.: „Zur Physiol. und Pathol. des phosphors. und oxals. Kalkes" wurde die Aetiologie der Oxalurie in Kürze besprochen. Es wurden vier Punkte als Hauptursachen derselben hervorgehoben, und zwar:

1. Mißbrauch zucker- und mehlhaltiger Nahrungsmittel;
2. Mangel an reiner, frischer Luft;
3. eine pathologische Metamorphose der stickstoffhaltigen Blutbestandtheile; und
4. ein vermehrter Alkali-Gehalt des Blutes.

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*) S. meinen ersten und zweiten Beitrag zur physiolog. Heilkunde. Göttingen. 1850.

Diese Angaben haben in weitern Beobachtungen nur eine Bestätigung gefunden. Wenn ich jedoch damals hinzufügte, daß die Frage, obgleich in einzelnen Punkten gelöst, durchaus noch nicht als erledigt anzusehen sei, so haben jene weitern Beobachtungen in der That nicht nur zu Erweiterungen der frühern Ansichten, sondern namentlich auch zu Berichtigungen der theoretischen Erklärung einiger Facta geführt. Es wurde damit eine neue Bearbeitung des Gegenstandes erforderlich.

Was zunächst die Oxalurie im Allgemeinen anbetrifft, so hat sie, um es zu wiederholen, in einer Störung, und zwar in einer Retardation der Stoffmetamorphose ihren Grund *). Der normale Urin enthält keine Oxalsäure. Wenn man mehrfach behauptet hat, sie komme auch im Urin gesunder Personen vor, so hat diese Behauptung nur in dem unbegränzten Begriff der „Gesundheit" ihren Grund. Aus ihrem tausendfältigen Vorkommen im Urin leicht und schwer erkrankter Individuen muß dagegen mit Bestimmtheit geschlossen werden, daß die Oxalsäure

*) Ich lasse hier die in Folge des Genusses gewisser Vegetabilien entstehende Oxalurie unberücksichtigt. Die Thatsache des Erscheinens oxalsaurer Kalkcrystalle nach dem Genusse von Rheum, Oxalis-Arten u. s. w. ist bekannt, wenn auch noch nicht hinreichend erklärt. Die reichlichen Quantitäten oxalsauren Kalkes im Pferdeharn schreibt G. Bird auf Rechnung des stets mit dem Heu gemischten Klees. — Ueber das Erscheinen der Crystalle im Urin nach dem Genuß von Allium porrum, portugiesischen Zwiebeln, Möhren, rothen Rüben, Blumenkohl, Spargel, Aepfeln, Apfelsinen und Trauben stellte C. B. Rose (London Med. Gaz. 1850. August 23) Untersuchungen an; nach Genuß der erstern beiden scheint sich stets Oxalurie einzustellen; nach dem der letztern waren die Resultate ungewiß. — Nach Rhabarber habe ich selbst oft große Mengen oxalsauren Kalkes in meinem eigenen Urin beobachtet, allein zu sehr verschiedenen Zeiten nach dem Genuß; bald früher, bald später. Er darf deshalb bei derartigen Untersuchungen nicht unterlassen werden, den Urin mehrmals in den dem Genusse folgenden 24 Stunden zu prüfen. — Nicht zu übersehen ist die Bemerkung Lehmann's (Physiol. Chem. I. pag. 50. 1850), daß die Quelle des oxalsauren Kalkes im Urin in diesen Fällen wohl nicht lediglich in den präformirten oxalsauren Salzen, sondern in dem Gehalt der fraglichen Nahrungsmittel an pflanzensauren Alkalien, „welche für sich auch eine Vermehrung des Kalkoxalats bedingen," zu suchen sein dürfte.

ein stetiges Produkt der Stoffmetamorphose im menschlichen Organismus ist, und daß sie sofort im Urin erscheint, wenn ihre normale Oxydation zu Kohlensäure in irgend welcher Weise beeinträchtigt wurde *). Ich muß zugeben, daß ihr directer Nachweis im Organismus, in specie im Blute, fehlt. Die einzelne Beobachtung Garrod's ist noch nicht entscheidend, wenn Golding Bird derselben auch eine schlagende Gültigkeit vindicirt **). Die Vermuthung Prout's, daß die Oxalsäure im Blute in Verbindung mit Harnstoff oder Ammoniak vorkomme und in dieser Form in den Nieren ausgeschieden werde, ist eben nichts, als eine Vermuthung ***). Allein ich glaube schwerlich, daß es leicht und oft gelingen wird, die verhältnißmäßig sehr geringe Quantität Oxalsäure im Blute nachzuweisen, wenn es kaum einmal gelingt, die bei Weitem beträchtlichern Mengen Harnstoffs aufzufinden. Die Unzugänglichkeit der Mittel- und Endglieder der einzelnen Reihen der Stoffmetamorphose, so lange sie sich im Organismus selbst bewegen, wird leider dem rationellen Schluß noch lange, wenn nicht immer, einen Platz offen lassen müssen. Findet man aber Oxalsäure bei Individuen jeden Alters, jeder Constitution und wird ihr Erscheinen bei den verschiedenartigsten Krankheitsprocessen beobachtet, so kann sie ihre Produktion unmöglich einzelnen bestimmten pathologischen Zuständen verdanken, sie muß vielmehr immer im Organismus producirt werden und nur ihr Erscheinen im Urin ist abhängig von diesen oder jenen Krankheitszuständen.

In welcher Weise kommt nun jene Störung oder Retardation der Stoffmetamorphose zu Stande? — Wie eine jede, so muß auch sie ihren Grund entweder auf Seiten des zu metamorphosirenden Materiales oder auf Seiten der das Material in Angriff nehmenden Agentien, der atmosphärischen Luft und dem belebten Organ, oder auf Seiten beider gleichzeitig haben. — Wir wenden uns zunächst der Betrachtung des erstern zu und sprechen also

*) 1 At. Oxalsäure = $C_2 O_3$;
1 At. Oxalsäure + 1 At. $O = C_2 O_4 = 2$ At. Kohlensäure.

**) Siehe „Urinary deposits" 1851. pag. 223.

***) Siehe „Stomach and renal diseases" 1848. pag. 564.

I. Von den Störungen des Nahrungs- oder Bildungsmaterials des Organismus als letzten Ursachen der Oxalurie.

Unsere Nahrungsmittel bestehen aus stickstoffhaltigen, stickstofffreien und anorganischen Verbindungen. In Mißverhältnissen eines jeden dieser drei Componenten kann die Ursache der Oxalurie liegen. — Was

1. die stickstoffhaltigen Verbindungen

anbetrifft, so ist die Möglichkeit des Hervorgehens der Oxalsäure aus ihnen schon lange bekannt. — Liebig und Wöhler wiesen die rasche Umwandlung der Harnsäure zu Oxalsäure, Harnstoff und Allantoin in Folge einer Sauerstoffaufnahme aus sauerstoffreichen Verbindungen nach *); durch Aufnahme von 4 At. Wasser und 3 At. Oxygen verwandelt sich ein Atom Harnsäure in 2 At. Harnstoff und 3 At. Oxalsäure **); die Thatsache, daß der in Amerika verladene Guano oft arm an Harnsäure, aber reich an Oxalsäure in England anlangt, spricht entschieden dafür, daß die Harnsäure unter Sauerstoff- und Wasseraufnahme außerhalb des Organismus in Oxalsäure und Harnstoff zerfällt; — die Möglichkeit eines gleichen Vorganges im Organismus ist einleuchtend. — Allein es fehlte noch immer der Beweis, daß ein solches Zerfallen innerhalb des Organismus auch wirklich Statt habe, bis derselbe vor Kurzem von Wöhler und Frerichs beigebracht ist. W. u. F. fütterten sowohl Hunde mit Harnsäure, als auch injicirten sie ihnen dieselbe in die Venen. In beiden Fällen wurde im Urin eine Vermehrung des Harnstoffs und Oxalsäure gefunden ***). Es konnte diese Oxalurie in

*) 1 At. Harnsäure = $C_{10}N_4H_4O_6$ 1 At. Oxygen = O_2 3 At. Wasser = H_3O_3 } = { $C_2N_2H_4O_2$ = 1 At. Harnstoff. $C_4 \quad O_6$ = 2 At. Oxalsäure. $C_4N_2H_3O_3$ = 1 At. Allantoin.

$C_{10}N_4H_7O_{11} = C_{10}N_4H_7O_{11}$

**) 1 At. Harnsäure = $C_{10}N_4H_4O_6$ 4 At. Wasser = H_4O_4 3 At. Oxygen = O_3 } = { $C_4N_4H_8O_4$ = 2 At. Harnstoff. $C_6 \quad O_9$ = 3 At. Oxalsäure.

$C_{10}N_4H_8O_{13} = C_{10}N_4H_8O_{13}$

***) Annalen d. Chem. u. Pharmac. Bd. 65. S. 338—342.

nichts anderm, als dem Genuß oder der Injection der Harnsäure ihren Grund haben, und wir müssen schließen, daß die letztere innerhalb des Organismus durch Aufnahme von Wasser und Sauerstoff in ähnlicher Weise in Harnstoff und Oxalsäure zerfällt, wie es beim Guano außerhalb desselben der Fall ist. — Ist nun aber die Harnsäure ein stetiges Produkt der Metamorphose unserer stickstoffhaltigen Blutbestandtheile und schließen wir aus dem tausendfältigen Vorkommen der Oxalsäure im Urin mit Recht, daß sie ebenfalls ein stetiges, aber nur unter pathologischen Verhältnissen als solches im Urin erscheinendes Produkt der Stoffmetamorphose ist, so dürfen wir nicht anstehen auch hier auf die Harnsäure zu recurriren und in den stickstoffhaltigen Blutbestandtheilen die Quelle der Oxalsäure zu suchen. Wir müssen annehmen, daß überall, wo in Folge irgend welcher Verhältnisse die Quantität der Harnsäure im Organismus vermehrt, wo die Metamorphose der stickstoffhaltigen Blutbestandtheile retardirt ist, sich ein gleiches Resultat herausstellt, wie in den Experimenten von Wöhler und Frerichs, daß, mit andern Worten,

in der Vermehrung oder der retardirten Metamorphose der stickstoffhaltigen Blutbestandtheile die Hauptursache der Oxalurie liegt.

Gegen die Richtigkeit dieser Annahme a priori ist nichts einzuwenden. Aber es fehlt auch nicht an hinreichenden Beweisen für dieselbe, und zwar entnehme ich diese:

1. aus der Beobachtung, daß die Oxalurie fast immer von Erscheinungen begleitet ist, welche mit Bestimmtheit eine Vermehrung oder eine Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile darthun;
2. aus der Erfahrung, daß die Oxalurie entschieden nach der Verringerung des stickstoffhaltigen Nahrungsmateriales abnimmt; und
3. aus der Beobachtung, daß die beschleunigte Metamorphose der stickstoffhaltigen Blutbestandtheile zur Abnahme oder zum Schwunde der Oxalurie führt.

Bei diesen Sätzen müssen wir einen Augenblick verweilen.

ad 1.

In allen Beobachtungen, welche ich über die Verhältnisse des oxalsauren Kalkes im Urin anstellte, habe ich der Quantität

des Urins, seinem Säuregrade und seinem specifischen Gewichte immer, dem Harnsäuregehalt meistens, dem Schwefelsäure- und Harnstoffgehalt mehrfach und unter den unorganischen Bestandtheilen, den Erdphosphaten stets besondere Aufmerksamkeit zugewandt. In manchen Fällen reihten sich diesen Beobachtungen oberflächliche Blutuntersuchungen an. Fasse ich die Ergebnisse kurz zusammen, so stellte sich überall, wo die Oxalurie eine nur einigermaßen beträchtliche Höhe erreicht hatte, auch eine gleichzeitige Vermehrung stickstoffhaltiger Harn- oder Blutbestandtheile heraus. — Die der letztern wurde direct nachgewiesen bei chronischen Gelenkrheumatismen und bei s. g. Hämorrhoidalleiden, welche mit Hyperämieen der Leber, Stuhlverstopfungen u. s. w. einhergingen. — Der Fibringehalt des Blutes war hier fast durchgehends vermehrt, eine Beobachtung, mit der alle frühern Blutanalysen von Andral, Becquerel und Rodier u. s. w. übereinstimmen. Das Blut tuberkulöser Individuen, welche an Oxalurie litten, habe ich nie untersucht; andre Beobachter wollen in demselben aber ziemlich constant eine Vermehrung der Albuminate gefunden haben. — Die Untersuchungen des Urins andrerseits ergaben fast immer eine Vermehrung des Harnsäure- und oft auch des Harnstoffgehalts; es dürfen daraus Rückschlüsse auf das Verhalten der stickstoffhaltigen Blutbestandtheile gemacht werden. — Ich muß hier die Bemerkung Golding Bird's wiederholen, daß der Harnstoffgehalt des oxalsauren Kalk haltenden Urins oft selbst bedeutend größer ist, als man aus dem specifischen Gewichte schließen sollte, ja daß er in 85 Fällen von Oxalurie 24mal so groß gefunden wurde, daß bei einfachem Zusatz von Acid. nitric. eine Crystallisation des salpetersauren Harnstoffs erfolgte. — Diese Beobachtungen habe ich einige Male bestätigt gefunden. Bei chronischen Rheumatismen, bei Leberhyperämieen und chronischen Dyspepsieen, bei scrophulösen Kindern und Erwachsenen habe ich in vielen Fällen ein sehr hohes specifisches Gewicht des Urins (1025 — 1030) ohne erhebliche Quantitätsabnahme, mehrmals vermehrten Harnstoffgehalt, fast immer oxalsauren Kalk, und nicht selten Harngries oder Sedimente von harnsauren Salzen (letztere namentlich bei scrophulösen Kindern) gefunden, so daß ich fast behaupten möchte, der oxalsaure Kalk findet sich nicht ohne gleichzeitige Vermehrung irgend eines stickstoffhaltigen Bestandtheils des Urins in demselben

vor*). Ein Jeder wird sich leicht und bald von der Richtigkeit dieser Behauptung überzeugen können und sind die stickstoffhaltigen Blutbestandtheile die Quelle der Oxalsäure, so muß und kann es auch nicht anders sein **). Es scheinen dieselben in der Norm ihre Metamorphose in der Weise zu durchlaufen, daß ein Theil in Harnsäure, ein Theil dieser Harnsäure aber wieder in Harnstoff und Oxalsäure umgewandelt wird; daß die Oxalsäure, zu Kohlensäure oxydirt, den Organismus verläßt, der Harnstoff aber (zum Theil vielleicht auch schon weiter zerfallen) nebst dem nicht metamorphosirten Theile der Harnsäure im Urin entleert wird. — Wird nun die Metamorphose der stickstoffhaltigen Blutbestandtheile nicht in normaler Weise zu Ende geführt (retardirt), so wird nicht nur die Oxalsäure zum Theil nicht zu Kohlensäure oxydirt, sondern es wird auch ein größerer Theil der Harnsäure einem weitern Zerfallen in Oxalsäure und Harnstoff entzogen, — man findet Harngries und oxalsauren Kalk im Urin; oder es wird der aus der Harnsäure hervorgehende Harnstoff, ohne weiterer Umwandlung zu unterliegen, sofort nebst der Oxalsäure entleert, — man findet vermehrten Harnstoffgehalt und oxalsauren Kalk neben normaler Quantität Harnsäure; oder es ist endlich die Metamorphose in der Weise retardirt, daß ein Theil der Harnsäure zwar in Harnstoff und Oxalsäure zerfällt, diese aber sofort entleert werden; ein ander Theil der Harnsäure dagegen, und zwar ein größerer als im Normalzustande, unzerlegt bleibt und neben Harnstoff und oxalsaurem Kalk als Harngries im Urin erscheint. — In

*) Willis in seinen „Urinary diseases" sagt: „The circumstances, „that may be supposed likely to lead to the state of urine, in which „oxalic acid is thrown down in combination with an earthy base, and „that consisting of lime, are in all probability intimately connected with „those, which lead to the deposition of lithic acid."

**) Ungern vermißt man bei Golding Bird's Bemerkung eine Angabe über die Quantitäten des entleerten Urins. Es darf dieselbe nie fehlen. Es findet sich eben bei an Oxalurie leidenden Individuen die Urinquantität sehr häufig verringert, man beobachtet bei Zusatz von Acid. nitric. die Bildung von Harnstoffcrystallen, aber die absolute Quantität des in 24 Stunden entleerten Harnstoffs ist dennoch nicht vermehrt, wie denn überhaupt eine solche Vermehrung seltner vorzukommen scheint, als man oft annimmt. G. Bird's Bemerkungen dürfen deshalb nur mit größter Vorsicht aufgenommen werden.

Fällen, wo durch die mannigfachsten Erscheinungen eine Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile erwiesen werden konnte, habe ich nicht selten gleichzeitig mit Vermehrung der Harnsäure neben dem oxalsauren Kalk auch Eiweiß (durch Ac. nitric. und Kochen erwiesen) im Urin gefunden, eine Beobachtung, welche mit unsern vorherigen Angaben in vollstem Einklange steht. Diese temporäre oder momentane Albuminurie findet sich durchaus nicht selten bei Individuen, die weder an Herzleiden, noch an Morb. Bright., höchstens an leichtern Hindernissen im venösen Kreislauf, als Leberhyperämieen u. s. w. leiden, deren Blut aber durch einen abnormen Gehalt an Albuminaten ausgezeichnet ist. Sie wird meistens übersehen; dies aber nur deshalb, weil man nur die 24stündigen Gesammtquantitäten prüft und in diesen die Quantitäten des Eiweißes zu gering sind, als daß sie durch unsere bisherigen Methoden mit Sicherheit nachgewiesen werden könnten. Sobald man die einzelnen zu verschiedenen Tageszeiten gelassenen Urine besonders untersucht, wird man oftmals der bezeichneten Albuminurie begegnen. So fand ich u. A. bei einem an chronischer Dyspepsie und Stuhlverstopfung, chronischen Rheumatismen und hypochondrer Gemüthsstimmung leidenden Hospitalpatienten, der am 15. Sept. um 7½ Uhr Morgens sein Frühstück (Thee und Butterbrod), um 12½ Uhr sein Mittagsessen (Rindfleisch und Blumenkohl) und um 7½ Uhr sein Abendessen (Thee und Butterbrod) bekam, folgende Verhältnisse:

Zeit.	Quantität	äuß. physik. Beschaffenheit.	Spec. Gew.	Säuregrad	Oxals. Kalk
7 U. 30 M.	12 Unzen	mittel-goldgelb.	1019	+ 26	0
9 „ 10 „	3½ „	dº.	1023	+ 34	zieml. viel.
11 „ 45 „	2½ „	dº. (enthält Eiweiß.)	1021	+ 45	sehr viel.
3 „ —	3 „	goldgelb; sehr leichtes Sediment von harnsauren Salzen.	1026	+ 64	0
7 „ —	4½ „	goldgelb; starkes Sediment von harnsauren Salzen.	1032	+ 54	0
11 „ 30 „	7½ „	goldgelb, klar.	1028	+ 38	mäßig viel.

An einem andern Tage, dem 23. Sept., wurden bei demselben

Patienten, während er Morgens 7 Uhr sein Frühstück (Thee und Butterbrod), Mittags 2 Uhr gekochtes Obst und Brodpudding und Abends 7 Uhr ein reichliches Abendessen mit Fleisch und Wein genoß, folgende Verhältnisse beobachtet:

Zeit.	Quantität	äuß. physik. Beschaffenheit. u. s. w.	Spec. Gew.	Säuregrad	Oxals. Kalk
6 U. 30 M.	8 Unzen	tiefgoldgelb, klar. (Spur von Zucker.)	1022	+ 27	sehr viel.
8 „ 45 „	2¾ „	dº. (schwache Spur von Zucker.)	1027	+ 41	wenig.
11 „ 45 „	3½ „	dº. (kein Zucker.)	1021	+ 30	viel.
5 „ —	5 „	dº. (kein Zucker, aber Albumen.)	1021,5	+ 46	sehr viel.
12 „ —	8 „	dº. (kein Zucker.)	1027	+ 32	sehr viel.

Ich zweifle nicht, daß sich derartige Verhältnisse, die mit Sicherheit auf eine Vermehrung oder Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile hinweisen, stets da nachweisen lassen, wo Oxalurie Statt hat. Die quantitativen Verhältnisse der Harnsäure und des Harnstoffs sind in dieser Beziehung besonders auffällig und interessant, und das zeitweilige Erscheinen von Eiweiß im Harn wird nicht lange mehr als Seltenheit betrachtet werden. — Wenn wir aber weiterhin wissen, daß bei an Oxalurie leidenden Individuen die oftmalige Bildung von Forunkeln, Ecthyma u. s. w. eine der gewöhnlichsten Erscheinungen ist, sollten wir nicht auch diese als einen Beleg für die retardirte Metamorphose der Albuminate des Blutes betrachten dürfen? Liefern diese Produkte, die nothwendiger Weise in einer Bluterkrankung ihren Grund haben müssen, nicht einen Beweis für das Vorhandensein überschüssiger Albuminate, für die Retardation der Metamorphose derselben? Berechtigen nicht jene massenhaften Ablagerungen in den Drüsen, jene Impetigines u. s. w. scrophulöser Kinder zu einer gleichen Annahme? Daß sich jene Produkte nicht organisiren, daß sie in Eiter zerfallen, hängt nicht von einem Mangel an eiweißartigen Verbindungen ab; es scheint diese Organisationsunfähigkeit vielmehr von dem mit der Oxalurie verbundenen Verlust an Erdphosphaten, von einem Mangel an dem für den Zellenbildungsproceß nothwendig erforderlichen phosphorsauren Kalk bedingt zu werden, und von ihr erleidet die obige Annahme somit keinen Einwurf.

ad 2.

Es bedarf kaum der Bemerkung, daß bei der Beurtheilung der von mir aufgefundenen Verhältnisse der Oxalsäure im Urin die jedesmalige Diät, so wie die Medicamente der Patienten in Anschlag gebracht wurden. Die Resultate, welche sich dabei herausstellten, lassen sich in Kürze dahin zusammenfassen, daß sich die Oxalurie in manchen Fällen beim Gebrauch von Mineralsäuren, in manchen beim Gebrauch des Eisens, in manchen durch Aufenthalt an der Seeküste u. s. w. besserte; der entschiedenste und fast allgemein gültige Einfluß wurde jedoch durch eine geeignete Veränderung der Diät, insonderheit durch Verminderung der stickstoffhaltigen Bestandtheile des Nahrungsmateriales ausgeübt. — Zunächst, statt vieler, eine einzelne Beobachtung.

Ein 30jähriger Mann, Finkenstein, von robustem Körperbau, frischem Colorit, aber melancholischem Temperamente, ein Glaser, litt seit langer Zeit an chronischem, intensivem Gelenkrheumatismus. Erscheinungen bedeutender Leberhyperämie waren zugegen. Er war während der Monate September, October und November im Hospital. Im September und October (bis zum 17ten) genoß er täglich 12 Unzen Brod, 6 Unzen Fleisch, 12 Unzen Gemüse, 1 Quartier Thee (aus etwa 1½ Drachmen) und ½ Quartier Milch. Allein Rheumatismus und Magenatarrh besserten sich während dieser Zeit trotz aller angewandten Heilmittel auch nicht im Geringsten. Nitrum, Colchicum, pflanzensaure Salze, Calomel, schwefelsaure Salze, Chinin u. s. w. Alles wurde vergeblich versucht. Sein Urin enthielt täglich eine beträchtliche Quantität oxalsauren Kalkes, eine entsprechende Quantität von Erdphosphaten, nicht selten Sedimente von Harnsäure und harnsauren Salzen und hatte einen hohen Grad von Acidität. Vom 17. Octob. an wurde das Fleisch ausgesetzt und statt der bisherigen 12 Unzen 16—24 Unzen vegetabilische Nahrungsmittel gegeben. Die übrige Kost blieb dieselbe. Am 24. Octob. bemerkte ich zum ersten Male eine Abnahme der Oxalate und, von dem Tage an, verringerte sich ihre Quantität mehr und mehr, ohne daß irgend weitere Veränderungen vorgenommen wurden. Am 14. November fehlten die Oxalate zuerst ganz. Der Rheumatismus besserte sich während dieser Zeit entschieden, und am 15. Nov. wurde Patient in bedeutend gebessertem Zustande entlassen. Gegen Ende November aber kehrte er zurück.

Er hatte einen neuen Anfall von Gelenkrheumatismus ohne acute Erscheinungen. Sein Urin enthielt wieder eine geringe Quantität oxalsauren Kalkes. Bei einer fast ausschließlich vegetabilischen Mittagsmahlzeit, dem täglichen Genuß von 16 Unzen Brod und 1½ Quartier Thee, besserten sich jedoch die Erscheinungen auch diesmal und kehrten nicht wieder bis zu Anfang Januar, wo der Patient als genesen entlassen wurde. Während dieser ganzen Zeit fanden sich nur höchst selten vereinzelte oxalsaure Kalk-Crystalle, und die Quantität der im Urin enthaltenen Erdphosphate überstieg die normale nicht. Arzneimittel wurden nicht angewandt, außer vielleicht einmal eine Pil. Rhei c. aloë, oder einige Eßlöffel Bittersalzlösung. Die möglichste Beschränkung stickstoffreichen Nahrungsmaterials führte zum Schwunde einer ausgeprägten Oxalurie *).

Nach dieser und ähnlichen ersten Beobachtungen richtete ich insonderheit auf die Verminderung des Stickstoffgehaltes des Nahrungsmaterials bei den betreffenden Patienten meine Aufmerksamkeit. Entweder wurden ihnen nur eine geringe Quantität Fleisch und daneben Gemüse und wenig mehl- und zuckerhaltige Nahrungsmittel erlaubt, oder es wurde — je nach dem vorliegenden Falle — das Fleisch durchaus gemieden und der Patient auf eine fast rein vegetabilische Diät gesetzt. Die Resultate waren erfreulich. In manchen Fällen und zwar namentlich in solchen, wo keine beträchtliche Störungen des Nervensystems und keine bedeutende anatomische Läsionen einzelner Organe vorlagen, wo die Constitution im Allgemeinen nicht geradezu schwächlich war, wurde oft ein gänzlicher Schwund der Oxalurie beobachtet, fast in allen aber bei consequent fortgesetzter Behandlung eine Abnahme derselben erreicht. — Es blieb mir zuletzt kein Zweifel

*) Die Untersuchung auf oxalsauren Kalk wurde stets der Art vorgenommen, daß ein Theil des Urins eines Tages in einem Probirgläschen hingestellt und nach 24 Stunden ein Tropfen seines Sedimentes, nach vorsichtigem Abgießen der darüber stehenden Flüssigkeit bis auf etwa 6 zurückbleibende Tropfen, mit einem Deckblättchen bedeckt, unter das Mikroskop gebracht wurde. In Bezug auf die sanduhrförmigen Crystalle muß ich eines, im Laufe des 2ten Halbjahrs 1850 in der London med. Gazette erschienenen Aufsatzes von Golding Bird erwähnen. Es wird in demselben mit großer Wahrscheinlichkeit dargethan, daß diese Crystalle aus Harnstoff in Verbindung mit oxalsaurem Kalk bestehen. G. Bird nennt sie: „oxalurates of lime."

übrig, daß die Verringerung des stickstoffreichen Nahrungsmaterials von dem wesentlichsten Einfluß auf die fraglichen Zustände war, und daß dieselbe in der Therapie der Oralurie den ersten Platz behaupte.

Es lag nach diesen Erfahrungen nur zu nahe, zu fragen, ob sich denn nach Mißbrauch stickstoffhaltiger Nahrungsmittel die Oralurie auch vermehre? Ich selbst habe die Frage leider bis dahin nicht genügend lösen können. — Lehmann stellt die Behauptung Bird's, daß stickstoffreiche Nahrungsmittel Niederschläge oder auch nur eine Vermehrung von Kalkoxalat bedingten, in Abrede *). Allein es bedarf zur Entscheidung dieses wichtigen Gegenstandes weit genauerer Untersuchungen, als sie bis dahin angestellt sind. Die einfache Negation eines Factums genügt nicht, ohne die gleichzeitige Angabe des Weges, auf welchem man zu ihr gelangte. — Es ist sehr wohl möglich, daß nach einem ein- oder mehrmaligen Genuß stickstoffreicher Substanzen keine Vermehrung oder kein Auftreten von Oxalsäure im Urin beobachtet wird. Allein der Schluß, daß solcher Genuß keinen Anlaß geben könne zur Oralurie, ist dennoch nicht gerechtfertigt. — Um ein sicheres Resultat zu gewinnen, muß, wie oben angedeutet, genau bezeichnet werden, was der Patient oder das betreffende Individuum neben den stickstoffhaltigen Substanzen genoß. — Verringert man neben dem reichlichen Genusse dieser die Quantität des stickstoffarmen Nahrungsmaterials, so kann die Metamorphose jener vollkommen zu Ende geführt werden; es wird allerdings mehr Oxalsäure im Organismus gebildet werden, aber die sämmtliche Quantität wird dennoch oxydirt, es erscheint keine Oxalsäure im Urin. Verringert man dagegen neben dem Genusse großer Quantitäten stickstoffreichen Nahrungsmaterials die des stickstoffärmern nicht, so muß, wenn die frühern Gesammtquantitäten den Anforderungen des Organismus genügten und unter übrigens gleichbleibenden Lebensverhältnissen, auf alle Fälle eine Störung der Stoffmetamorphose resultiren, und ich glaube a priori annehmen zu können, daß Oralurie ein Symptom dieser Störung sein wird. — Man müßte also Versuche der Art anstellen, daß man einem nicht oder nur in geringem Grade an Oralurie leidenden Individuum neben ganz unveränderter Diät und unter ganz unveränderten Lebensverhältnissen in weitester Bedeutung des Wortes, längere Zeit hindurch eine erhöhte Quantität stickstoffreichen Nahrungsmateriales darreichte und nun prüfte, ob oxalsaurer Kalk im Urin auftritt oder ob sich der etwa schon vorhandene vermehrt. — Keine Frage über die Wirkung oder Metamorphose dieser oder jener Nahrungsmittel ist zu entscheiden ohne die genaueste Berücksichtigung der die einzelnen Reihen der Stoffmetamorphose beherrschenden Abhängigkeitsgesetze und ohne Veranschlagung der sämmtlichen die Metamorphose bedingenden Agentien. — Der inductive, vollgültige Beweis für die obige Frage fehlt also bis dahin. — Dagegen kann ich mit Bestimmtheit behaupten, daß ein sorgfältiges Examen der an Oralurie leidenden Patienten fast immer Anhaltspunkte für den Nachweis einer directen Vermehrung oder einer retardirten Metamorphose der stickstoffhaltigen Blutbestandtheile auffinden läßt. Läßt man sich von den fraglichen Patienten die genauesten Angaben über ihre bisher befolgten diätetischen Maaßnahmen machen, so erfährt man in vielen Fällen, daß die Quantität des genossenen stickstoffreichen Nahrungsmaterials (Milch, Fleisch, Eier) eine sehr beträchtliche war, und in den meisten Fällen, daß neben einer solchen eine ebenfalls beträchtliche Quantität süßer oder fetter Mehlspeisen, viel Brod und Kartoffeln, viel Butter oder Bier, oft auch viel Kochsalz genossen wurde. Es wurde damit entweder direct eine Vermehrung der stickstoffhaltigen Blutbestandtheile herbeigeführt, oder es wurde durch einen Mißbrauch der stickstoffärmern Nahrungsmittel die Metamorphose der stickstoffreichern retardirt (s. u.). Je länger aber die ätiologischen Momente eines Krankheitszustandes vorhanden sind, desto intensiver sind seine Erscheinungen, desto intensiver wird bei den fraglichen Krankheitszuständen die Oralurie sein. Eine vorübergehende Störung der diätetischen Verhältnisse führt jenachdem nur zu vorübergehenden, oft selbst schwer nachweisbaren Störungen der Stoffmetamorphosen, ein Punkt, auf den in Betreff der oben proponirten Versuche über die künstliche Herbeiführung der Oralurie durch Vermehrung der Fleischkost besondere Rücksicht genommen werden muß. — Es wird leider in den bisherigen Anamnesen oder Aetiologieen auf die scheinbar unbedeutenden Verhältnisse der Diät wenig Rücksicht genommen, und doch liegen ohne Frage eben in der täglichen Wiederholung klein-

*) Physiol. Chem. 1850. Bd. I. pag. 49.

ster Mißgriffe die ursächlichen Momente der bedeutendsten Krankheitsprocesse, jener chronischen Zustände, die oft allem Heilverfahren Trotz bieten. Ist es anders, als mit Berücksichtigung der sämmtlichen, auch noch so unbedeutend scheinenden diätetischen Verhältnisse möglich dahin zu gelangen, wohin die Aetiologie des Krankheitsprocesses gelangen muß, zur Darstellung vollständiger Entwickelungsgeschichten einzelner Krankheitsfälle oder zusammengehöriger Gruppen von Krankheitszuständen, insonderheit der Blutdyskrasieen? Ist es anders möglich, zu einer rationellen Therapie den Grund zu legen? —

ad 3.

Den dritten, vollgültigen Beweis für die Richtigkeit unserer obigen Annahme entnehme ich aus dem Untersuchungsresultate, daß die Oralurie einer beschleunigten Metamorphose der stickstoffhaltigen Blutbestandtheile ihre Entstehung **nicht** verdankt. — In einer Reihe von acuten Krankheitsfällen habe ich bei der täglichen quantitativen Bestimmung des Schwefelsäuregehalts des Urins die Beobachtung gemacht, daß dieser Gehalt proportional den Fieberbewegungen zu- und abnimmt, vorausgesetzt, daß keine schwefel- oder schwefelsäurehaltigen Arzneimittel genossen sind. — Es kann nun aber die Schwefelsäure im Urin in keinen andern Stoffen ihre Quelle haben, als in den stickstoffhaltigen Blutbestandtheilen, und ihre Zu- und Abnahme muß uns einen Maaßstab für die größere oder geringere Intensität der Metamorphose derselben an die Hand geben. Ich weiß den vermehrten Schwefelsäuregehalt des Urins in fieberhaften Zuständen daher auf keine andere Weise, als durch eine beschleunigte Metamorphose jener Bestandtheile zu erklären. Nun aber wurde schon in meinem zweiten Beitrag zur physiol. Heilkunde erwähnt, daß mit der Oralsäure gerade ein umgekehrtes Verhältniß Statt hat; — sie nimmt in fieberhaften Zuständen eben ab. Leidet Jemand an Oralurie und wird er von einem acuten Krankheitsvorgange ergriffen, so findet man stets entweder einen Schwund oder wenigstens eine Abnahme der Oralsäure im Urin; eine Beobachtung, zu der namentlich chirurgische Krankheitsfälle, in denen man den Kranken vor einer etwaigen Operation kennen lernte und nun in einem durch die Operation hervorgerufenen fieberhaften Zustande beobachten kann, die beste Gelegenheit darbieten. —

Die beschleunigte Metamorphose der stickstoffhaltigen Blutbestandtheile führt also zur Abnahme des Oralsäuregehalts des Urins, und es folgt daraus, daß die Oralurie einer solchen ihren Ursprung nicht verdanken kann.

Somit stimmen Theorie, Experiment und Erfahrung überein; die Richtigkeit des obigen Satzes, daß eine Vermehrung oder eine Retardation der Metamorphose stickstoffhaltiger Blutbestandtheile Anlaß giebt zur Oralurie, scheint mir danach keinem weitern Zweifel zu unterliegen.

Wir kommen

2. zu den stickstofffreien Bestandtheilen des Nahrungsmaterials.

Die Möglichkeit, daß die Oralsäure auch in ihnen eine Quelle hat, ist allerdings vorhanden. — Auf die feste Beobachtung gestützt, daß Mißbrauch zucker- und mehlhaltiger Nahrungsmittel Anlaß geben zur Oralurie, verleitet ferner durch die große Plausibilität der chemischen Formeln glaubte ich früher in der That annehmen zu müssen, daß die stickstofffreien Bestandtheile des Nahrungsmateriales ebensowohl, als die stickstoffhaltigen Quelle der Oralsäure seien. Allein, so fest auch jene Beobachtung steht, weitere Untersuchungen haben mich eines andern belehrt. Auf Seite 88. meines erwähnten Schriftchens wurde gesagt: „Ich habe Patienten, welche wenig oder gar keinen oralsauren „Kalk im Urin entleerten, längere Zeit hindurch täglich 1/4 Pfund „Rohrzucker nehmen lassen, ohne in ihrer gewöhnlichen Kost eine „Veränderung zu machen; es stellte sich während des Gebrauches „Dyspepsie, meistens mit saurem Aufstoßen und Appetitlosigkeit „verbunden, ein. Die Urinuntersuchungen aber ergaben, daß „sich während des Gebrauches selbst weniger, nach „demselben aber viel oralsaurer Kalk und dem entsprechend viel „Erdphosphate im Urin vorfanden." Der daraus gezogene Schluß, daß der Mehrgenuß von Zucker die Ursache zur Erscheinung der Oralsäure im Urin abgab, ist und bleibt richtig. Dann aber hieß es weiter: „Andrerseits mögen auch die die „Umwandlung des Zuckers in Oralsäure und Wasser darstellenden Formeln einen Beweis für die Leichtigkeit abgeben, mit „welcher diese Umwandlung (d. i. des Zuckers in Oralsäure) „erfolgen kann und höchst wahrscheinlich erfolgt"; es wurde also

die im Urin aufgefundene Oxalsäure direct aus dem genossenen Zucker hergeleitet. Die Formeln selbst lauteten:

Crystallis. Traubenzucker = $C_{12}H_{24}O_{14}$
18 At. O. = O_{18}
= $C_{12}H_{24}O_{32}$ = 6 At. Oxals. + 14 At. Wasser.

Crystallis. Rohrzucker = $C_{12}H_{22}O_{11}$
18 At. O. = O_{18}
= $C_{12}H_{22}O_{29}$ = 6 At. Oxals. + 11 At. Wasser.

Milchzucker = $C_{10}H_{20}O_{10}$
15 At. O. = O_{15}
= $C_{10}H_{20}O_{25}$ = 5 At. Oxals. + 10 At. Wasser.

In der That, die Plausibilität dieser Formeln ist so groß, daß sie nur zu leicht zu der obigen Annahme verleiten konnte. Dennoch, die Formeln scheinen nichts weniger zu sein, als der Ausdruck für das factische Geschehen. Es muß hier vor Allem auf den früher geringer geachteten Umstand aufmerksam gemacht werden, daß sich während des Gebrauches des Zuckers selbst weniger, wohl aber nach demselben viel oxalsaurer Kalk im Urin fand. Gäbe der Zucker die directe Quelle der Oxalsäure ab, so müßte sich eben während des Zuckergenusses mehr Oxalsäure im Urin finden, als nach demselben. Ueber diesen Punkt mußte jedoch erst vollkommne Klarheit herrschen, und es war deshalb nochmals zu untersuchen, in welchem temporären Verhältnisse das Erscheinen der Oxalsäure im Urin zu dem Mißbrauche zucker- oder mehlhaltiger Nahrungsmittel stehe?

Es fehlt bis dahin durchaus an Untersuchungen, aus denen eine bestimmte Antwort auf diese Frage entnommen werden kann; daher die große Unsicherheit früherer Angaben. Prout, Bence Jones u. Golding Bird haben sich namentlich mit dem Gegenstande beschäftigt. Ihre Resultate sind ebenso different, als unsicher. Prout sagt: „the too free use of sugar gives occasion to the oxalic acid form of dyspepsia." Dies „gives occasion" ist ein zweideutiger Ausdruck; aus Allem aber was Prout in seinem trefflichen Werke weiter bemerkt, muß ich schließen, daß er die Oxalsäure aus dem Zucker direct herleitete. Er läßt sich selbst nicht weiter darüber aus; nirgends aber ist der Erklärung, die ich weiter unten als die richtige bezeichnen zu

können glaube, nur gedacht. Bence Jones sagt: „whether sugar gives rise to oxalic acid is far from being determined." Dies „gives rise" bezeichnet die directe Entstehung. B. Jones stellt jedoch nur einen Zweifel an der frühern Prout'schen Lehre auf, bringt aber selbst die Frage zu keiner weitern Entscheidung. Golding Bird endlich sagt in seiner neuen, dritten Ausgabe der „Urinary deposits" pag. 220: „I am convinced myself, that the „connection between oxalate of lime and sugar is by no means „a necessary one." Allein, wo es sich um bestimmte Beobachtungen und Thatsachen handelt, können Ueberzeugungen (convinced) nichts helfen, und der Ausdruck „a necessary connection" ist nebenbei so unsicher, daß mit dem ganzen Satze nichts gewonnen ist. — Nach diesen Vorlagen habe ich die Frage einer weitern Untersuchung unterzogen, und es läßt sich aus derselben mit hoher Wahrscheinlichkeit folgern, daß die nach dem Mehrgenuß mehl- und zuckerhaltiger Nahrungsmittel im Urin aufzufindende Oxalsäure nicht direct aus dem Zucker und Amylon hervorgeht; daß sie vielmehr ihre Quelle auch hier in den stickstoffhaltigen Substanzen hat und daß die durch einen Mißbrauch mehl- und zuckerhaltiger Nahrungsmittel retardirte Metamorphose dieser letztern in diesem Falle die Oxalurie veranlaßt. — Ich schließe dies:

1. aus dem Beobachtungsresultate, daß die Oxalsäure selten während, meistens aber nach dem längern Mißbrauch zucker- oder mehlhaltiger Substanzen im Urin auftritt oder in ihrer frühern Quantität zunimmt; daß sie bei kürzerm Mißbrauch jener Substanzen dagegen mitunter weder während noch nach dem Genuß derselben erscheint;
2. aus der unzweifelhaften Thatsache, daß die Oxalsäure ein Glied in der Metamorphosenreihe der stickstoffhaltigen Blutbestandtheile bildet und eine Vermehrung oder retardirte Metamorphose dieser Bestandtheile zur Oxalurie führt; und
3. aus dem directen Untersuchungsresultate Böcker's, daß der Zuckergenuß in der That die Metamorphose der stickstoffhaltigen Substanzen retardirt.

ad 1.

Was zunächst die Beobachtungen über das Auftreten der Oxalsäure im Urin bei Mehrgenuß von Zucker betrifft, so mögen die folgenden kurzen Mittheilungen genügen.

1. Adam Müller, 34 Jahr alt, litt seit längerer Zeit an einer sehr hartnäckigen Acne mentagra. Er war schon seit nahe 10 Wochen im Hospital. Bei der fast täglichen Untersuchung seines Urins wurden im September fast immer einzelne, im October sehr selten vereinzelte Oxalate gefunden. — Robuste Constitution, energischer Charakter, heitere Gemüthsstimmung, ein leicht anaemisches Colorit zeichneten ihn aus. Von weitern pathologischen Erscheinungen bedarf nur eine anhaltende leichte Dyspepsie der Erwähnung. Das Frühstück des Mannes bestand seit längerer Zeit aus 1/2 Quartier Milch und 6 Unzen Brod; sein Mittagsessen aus 3/4 Pfund Kartoffeln, 1 Pfund grünen Gemüses und 4 Unzen Brod; sein Nachtessen aus 1/2 Quartier Milch und 6 Unzen Brod. Diese Diät wurde nach wie vor beibehalten. — Am 4. November wurden vereinzelte Oxalate gefunden; dann vom 6. — 17. November täglich 1/4 Pfund Rohrzucker gereicht. Allein obgleich am 8. und 9. November, gleichwie am 4., einige wenige Oxalate erschienen, vom 10. an fand ich sie weder bis zum 17., noch nachher bis zum 22. — Der tägliche Genuß eines viertel Pfundes Zuckers neben der unveränderten frühen, zur Sättigung des Mannes hinreichenden Quantität von Nahrungsmitteln führte also nicht zum Auftreten von Oxalsäure im Urin, weder während, noch nach dem Zuckergenuß. Gegen Ende des letztern steigerten sich die Erscheinungen der Dyspepsie beträchtlich; sie schwanden wieder nach dem Aussetzen des Zuckers.

2. Ein anderer Patient, Johann Schmidt, 36 Jahr alt, litt seit langer Zeit an chronischer Dyspepsie und chronischem Gelenkrheumatismus. Allgemeine Schwäche und Abmagerung, schlaffe Muskulatur, ein kleiner weicher Puls, deprimirte Gemüthsstimmung, hohe Irritabilität des Nervensystems bildeten Hauptzüge des Krankheitsbildes. Auch er hatte schon längere Zeit im Hospital unter meiner Aufsicht gestanden. Sein Urin, der täglich untersucht wurde, enthielt stets beträchtliche Quantitäten oxalsauren Kalkes und von Erdphosphaten; letztere oft zu dem Betrage von 40 — 60 Gran in 24 Stunden. Es lag hier

ein exquisites Beispiel jenes Zustandes vor, den Prout als „oxalic acid diathesis“ bezeichnete. Die Diät des Patienten war folgende: zum Frühstück: 1/4 Quartier Milch und 6 Unzen Brod; zu Mittag: 4 Unzen Fleisch, 8 Unzen Kartoffeln und 4 Unzen Brod; zu Nacht: 1 Quartier Graupen- oder Gerstensuppe und 6 Unzen Brod. Täglich 1 Unze Butter. Arzneimittel wurden, außer täglich 3 Dessertlöffel voll Ol. jecor. Asell., nicht genommen. Versuchs halber erhielt nun Patient vom 7. — 19. April täglich 1/4 Pfund Zucker neben der angegebenen Quantität Nahrungsmittel. Die Erscheinungen der Dyspepsie steigerten sich während dieser Zeit; der Appetit nahm ab; starke Säurebildung im Magen stellte sich ein, und der Allgemeinzustand war weit entfernt von irgend welcher Besserung. Während des Zuckergenusses fand jedoch vom 7. — 12. April eine entschiedene Abnahme der Oxalate im Urin gegen früher Statt; am 13., 14. und 15. fanden sich mehr, als in den ersten Tagen, aber auch noch entschieden weniger, als früher; am 16., 17., 18. u. 19. stand dagegen die Quantität der vor dem Zuckergenuß beobachteten gleich und zwei Tage lang nach dem Aussetzen des Zuckers nahm sie noch beträchtlich zu. Bei der mikroskopischen Untersuchung fand sich in den letzten 4 Tagen das ganze Gesichtsfeld mit Crystallen übersäet, und während sich früher nur selten die s. g. sanduhrförmigen Crystalle fanden, waren dieselben jetzt in reichlicher Menge mit den Octaedern vermischt. Die quantitativen Verhältnisse der Erdphosphate liefen durchaus parallel mit denen des oxalsauren Kalkes. — Bemerkt mag auch werden, daß sich am 21. April ein Sediment von Harnsäure und harnsauren Salzen vorfand, ohne daß in der Quantität oder Acidität des Urins eine Veränderung eingetreten wäre. — In einem Individuum also, das längere Zeit hindurch beträchtliche Quantitäten oxalsauren Kalkes im Urin entleerte, nahm die Quantität desselben während des täglichen Genusses von 1/4 Pfund Zucker neben unveränderter früherer Diät anfangs beträchtlich ab, dann etwas zu, erreichte die frühere Höhe und überstieg diese endlich sowohl während der letzten Tage des Zuckergenusses, als auch in den ersten Tagen nach demselben.

3. Johann Huskamp, 24 Jahr alt, Zuckerbäcker, litt an Lungentuberkulose. Er hatte vor kurzer Zeit eine acute Tuberkel-Ablagerung in der obern linken Lungenspitze überstanden. Die

acuten Erscheinungen waren geschwunden; die der chronischen Lungentuberkulose lagen vor. Der Urin des Patienten enthielt am 17., 18., 19. u. 20. April sehr viel Oralate, eine entsprechende Quantität von Erdphosphaten, hatte bei der Quantität von 50 — 60 Unzen ein specif. Gewicht von 1026 — 1025 und einen sehr hohen Säuregrad (+ 40 — + 48). Seine Diät bestand aus täglich 6 Unzen Fleisch, 16 Unzen Brod, 1 Unze Butter, 16 Unzen Gemüse und 1 Quartier Milch. Daneben erhielt er täglich 1½ Unzen Leberthran und dreimal eine Messerspitze voll phosphorsauren Kalk. Vom 23. April bis 9. Mai wurde neben der bezeichneten Diät täglich ¼ Pfund Zucker gegeben. — Der Urin zeigte während dieser Zeit folgende Veränderungen:

vom 24. — 28. April fiel die Quantität von 50 bis auf durchschnittlich 40—45 Unzen. Das spec. Gew. sank auf 1021, am 28. sogar auf 1014. Am 24. — 25. fanden sich noch Sedimente von harnsauren Salzen; am 26., 27. u. 28. dagegen fehlten dieselben, der Urin war klar, behielt aber seine sehr dunkle, tiefrothe Färbung. Der Säuregrad verminderte sich von + 48 auf + 24; die Oralate vermehrten sich nicht, verminderten sich dagegen an demselben Tage, an welchem die harnsauren Sedimente nicht erschienen;

vom 24. April bis 10. Mai verminderte sich die durchschnittliche 24stündige Quantität bis auf 30 - 35 Unzen. Das specif. Gew. schwankte zwischen 1016 u. 1022. Besonders bemerkenswerth waren die in dieser Periode erscheinenden Sedimente reiner Harnsäure. Die tiefe, dunkel-goldgelbe Färbung blieb; der Säuregrad schwankte zwischen + 15 und + 36; er lief parallel mit den Schwankungen des specifischen Gewichtes. Die Oralate fehlten nie; ihre Quantität war aber an einigen Tagen absolut geringer, und niemals größer als vor der Zeit des Zuckergenusses; die Erdphosphate zeigten dem entsprechende Schwankungen;

vom 10. — 16. Mai endlich, also nach der Zeit des Zuckergenusses blieb die Quantität des gelassenen Urins meistens unverändert, nur am 16. erreichte sie die Höhe von 50 — 60 Unzen. Das specif. Gew. aber nahm wieder zu; es stieg auf 1020—1024—1026; die Sedimente von reiner Harnsäure fehlten; nur leichte Sedimente von harnsauren Salzen erschienen. Die Farbe blieb tiefrothgelb. Der Säuregrad stieg wieder auf + 36 — + 48.

Die Quantität der Oralate war constant eine bedeutende. — Das Allgemeinbefinden des Patienten erlitt in dieser Zeit wenig Veränderung. Ein mäßiger Husten ohne Auswurf war zugegen. Der Digestions-Apparat war bis auf den 5. und 6. Mai, an denen ein Magencatarrh auftrat, frei von Krankheitserscheinungen; im Circulationsapparat wurden keine Störungen wahrgenommen. Nur bildete sich in dieser Periode mehr und mehr ein chlorotischer Zustand hervor und Patient magerte dabei ab.

Die Bestimmung der Quantität des oxalsauren Kalkes durch's Mikroskop kann einer exacten Forschung nicht genügen. Dennoch läßt sie bei tagtäglicher Vornahme, bei der Beobachtung durchweg gleicher Maaßregeln, bei gehöriger Sorgfalt und längerer Uebung allgemeine Schlüsse zu, welche zuverlässig sind. Wer sich nur die Mühe geben will, tagtäglich die Sedimente einer größern Anzahl von Urinen (24stündige Gesammtquantitäten) in der angegebenen Weise zu untersuchen, wird sich alsbald von der großen Differenz der Quantitäten oxalsauren Kalkes überzeugen, und das Auge reicht hin, um approximativ zu bestimmen, was durch die Waage **kaum** mit größter Mühe und unendlichem Zeitaufwand ermittelt werden kann. Für die Praxis ist es aber irrelevant, ob man weiß, wie viel 1/10 oder 1/100 Gran oxalsauren Kalkes im Urin enthalten sind; es genügt zu wissen, ob sehr viel, viel, wenig oder gar keine Oralate im Urin entleert wurden. — Ein Uebelstand bei diesen Untersuchungen ist der, daß man nicht ermitteln kann, wann sich im Urin aller oxalsaure Kalk ausgeschieden hat. Ob er, wie Lehmann meint, in löslicher Form im Urin vorkommt, oder ob die Oxalsäure, wie Prout vermuthet, in Verbindung mit Harnstoff oder Ammoniak ausgeschieden wird, wage ich nicht zu entscheiden; soviel nur steht fest, daß man 2 — 3 Stunden nachdem der Urin gelassen ist, oft viel weniger Crystalle von oxalsaurem Kalk findet, als nach Verlauf von etwa 24 Stunden. Indeß ich glaube annehmen zu können, daß durch die regelmäßige Prüfung des Urins 24 Stunden nach der Entleerung jenem Uebelstande größtentheils abgeholfen wird. Genauere Untersuchungen werden über die Ausscheidungsverhältnisse noch entscheiden müssen.

Bei allen Beobachtungen, die ich in Betreff der Oxalurie angestellt habe, wurde der Diät, und zwar ihren quantitativen Verhältnissen sowohl, als den qualitativen, die mögliche Auf-

merksamkeit geschenkt. Ohne dieser Anforderung zu entsprechen, ist es unmöglich, zu irgend welchen brauchbaren Schlüssen zu gelangen. Es würde thöricht sein, einem Patienten eine beliebige Quantität Zucker zu geben, und, jenachdem nun der Urin Oxalsäure enthält oder nicht, schließen zu wollen, der Zucker veranlasse Oxalurie oder keine. Wenn nicht bekannt ist, was und wie viel der Patient neben dem Zucker genossen, welche Bewegung, welchen Aufenthaltsort, welche Constitution er hatte, welcher Art ferner sein Gesammtkrankheitszustand war, so ist die Beobachtung nutzlos. Diesen Anforderungen ist bis dahin nicht entsprochen und daher die Unsicherheit früherer Angaben.

Aus den obigen Beobachtungen, deren Zahl ich noch um einige vermehren könnte, folgt nun, daß während des täglichen Genusses von 1/4 Pfund Zucker neben der den Anforderungen des Organismus genügenden Quantität Nahrungsmittel keine Oxalsäure im Urin erschien, wo sie sich in den vorhergehenden Tagen doch wenigstens spurweise gezeigt hatte; daß bei Individuen, die schon an Oxalurie litten, die Quantität der Oxalate im Beginne des Zuckergenusses abnahm, gegen Ende desselben wuchs, nach dem Aussetzen des Zuckers aber erst diejenige und selbst eine höhere Quantität erreichte, welche sie vor dem Zuckergenuß hatte. Wir schließen daraus für den Augenblick nur so viel, daß der Zucker selbst nicht die Quelle der im Urin erscheinenden Oxalsäure sein kann.

ad 2.

Dieser Satz wurde oben bereits hinreichend besprochen und bedarf hier deshalb keiner weitern Auseinandersetzung.

ad 3.

Böcker's Untersuchungsresultate in Betreff der Einwirkung des Zuckergenusses auf die Stoffmetamorphose, finden wir im ersten Bande seiner „Beiträge zur Heilkunde" Crefeld 1849, mitgetheilt. Es wird darin nachgewiesen, daß der Zuckergenuß die „Mauser" der Organe und insonderheit der stickstoffhaltigen Blutbestandtheile hemmt. Es war während desselben die Quantität des Urins geringer, als im Normalzustande; sein Harnstoffgehalt nahm absolut ab; der Harnsäuregehalt ebenfalls und nicht minder die sämmtlichen unorganischen Bestandtheile, insonderheit die

Erdphosphate. — Wenn aber weiterhin B. das Resultat aufstellte, daß die Erdphosphate auch nach dem Zuckergenuß in verminderter Quantität entleert werden, so muß ich auch hier wieder darauf aufmerksam machen, daß diese Angabe Folge eines einfachen, in meinem zweiten Beitr. z. physiol. Heilk. nachgewiesenen Rechnungsfehlers war. Die erste Angabe, daß die Erdphosphate während des Zuckergenusses abnahmen, ist richtig; die zweite aber muß dahin corrigirt werden, daß nach dem Zuckergenuß die Quantität der im Urin entleerten Erdphosphate steigt. — Der Wichtigkeit der Frage gemäß lasse ich die von Böcker gefundenen Mittelzahlen folgen.

	Mittel aus 3 Untersuchungen vor dem Zuckergenuß.	Mittel aus 9 Untersuchungen während des Zuckergenusses.	Mittel aus 11 Untersuchungen nach dem Zuckergenuß.
24stündige Quantität	2873,000 Grmm.	2592,466 Grmm.	2446,727 Grmm.
Spec. Gewicht	1014,5 "	1014,1 "	1017,7 "
Feste Bestandtheile	69,609 "	55,671 "	69,846 "
Harnstoff	28,658 "	17,432 "	23,336 "
Harnsäure	0,268 "	0,222 "	0,384 "
Feuerbeständige Salze	23,727 "	19,572 "	25,178 "
In Wasser lösliche Salze	20,364 "	17,338 "	22,611 "
" " unlösliche "	3,363 "	2,234 "	2,567 "
Phosphors. Kalk	1,498 "	0,630 "	1,925 "
" Magnesia	0,570 "	0,301 "	0,390 "
Schwefelsäure	7,831 "	—— "	1,753 "
Feuerflüchtige Salze und Extractivstoffe	16,956 "	18,445 "	20,948 "

Es stehen diese Untersuchungen noch einzig in ihrer Art da. Diejenigen, welche ich in ähnlicher Weise in Bezug auf einzelne Urinbestandtheile angestellt habe, führten mich freilich zu ganz gleichen oder ähnlichen Resultaten. Allein sie haben durchaus keinen gleichen Anspruch auf Genauigkeit, da ich die Quantität der Erdphosphate nur approximativ bestimmte. Für eine rationelle Diätetik sind Böcker's Untersuchungen von hohem Werthe. — Was nun aber vom Zucker gilt, gilt selbstverständlich auch vom Amylon und andern ihm verwandten oder ähnlichen Stoffen. Sie alle durchlaufen den Untersuchungen zufolge in ihrer Metamorphose im thierischen Organismus die Stufe des Zuckers und dienen in gleicher Weise, wie dieser, zur Retardation der

Metamorphose der stickstoffhaltigen Verbindungen. — Weitere Anhaltspunkte für eine Retardation der Stoffmetamorphose im Allgemeinen während eines übermäßigen Zuckergenusses finden wir in dem sich in der Regel einstellenden Appetitverlust, in den gastrisch-catarrhalischen Erscheinungen u. s. w., und wenn wir die Oxalurie als eine auf einer solchen Retardation beruhende Erscheinung betrachten, so dürften die Angaben von Scherer, Lehmann und Marchand, daß neben der Gegenwart einer erheblichen Menge oxalsauren Kalks im Harn immer auch Milchsäure gefunden wird für die Richtigkeit dieser Betrachtung in Anschlag zu bringen sein *). Aus welchen Stoffen die im Urin aufzufindende Milchsäure auch herstammt, ob aus den stickstoffhaltigen oder stickstofffreien, im normalen Urin findet sie sich nicht, ihr Erscheinen muß in einer irgendwie veranlaßten Retardation der Metamorphose seinen Grund haben.

Hiernach kommen wir auf die obige Hauptfrage und das dort mitgetheilte Factum zurück. Die schon in Kürze gegebene Antwort wird kaum eines weitern Beweises ihrer Richtigkeit bedürfen. — Der Zucker oder das Amylon scheinen nicht die directe Quelle der im thierischen Organismus producirten Oxalsäure zu sein; die große Plausibilität der chemischen Formeln, welche die Metamorphose des Zuckers in Oxalsäure und Wasser in Folge einer Aufnahme von Sauerstoff darstellen, verleitet leicht, aber fälschlich zu einer solchen Annahme. Dagegen ist einmal durch Theorie, Experiment und Beobachtung erwiesen, daß die Oxalsäure unbezweifelt ihre Quelle in den stickstoffhaltigen Blutbestandtheilen haben kann und hat, und andrerseits durch das Experiment dargethan, daß die Metamorphose dieser letztern in Folge eines Mißbrauchs von Zucker retardirt, d. h. nicht zu Ende geführt wird, und es folgt daraus, daß wenn Mißbrauch von Zucker und mehlhaltigen Nahrungsmitteln Anlaß giebt zur Oxalurie, dieser Erfolg aus der die Metamorphose der stickstoffhaltigen Organ- und Blutbestandtheile retardirenden Einwirkung des Zuckers erklärt werden muß.

Noch eines Umstandes muß ich erwähnen, der dennoch Zweifel an dieser Behauptung aufkommen lassen könnte. Sowohl aus den sorgfältigen Untersuchungen Prout's, als aus

*) Cf. Lehmann physiol. Chem. Bd I, p. 105.

denen Golding Bird's geht hervor, daß zuckerhaltiger Urin und namentlich der der Diabetiker selten Oxalsäure enthält. Bei zwei Diabetikern habe auch ich keinen oxalsauren Kalk im Urin gefunden. Allein es wurde schon in meinem mehrerwähnten Schriftchen S. 89. bemerkt, daß ich eigenen, namentlich bei scrophulösen Kindern gemachten Beobachtungen zufolge durchaus den weitern Bemerkungen Prout's beistimmen muß. Prout sagt nämlich: „Urine containing oxalic acid seldom contains „sugar; yet I have seen a few instances, in which the two „unnatural ingredients have not only co-existed, but appeared „to pass into each other, that is, sugar has gradually given „place to the oxalate of lime and v. v. oxalate of lime to „sugar." Diese Beobachtungen weisen zu offenbar auf den nahen Zusammenhang des Harnzuckers und der Oxalsäure im Urin hin, als daß man an dem Hervorgehen der letztern aus dem erstern zweifeln könnte *). Nun ist es aber eine noch fast allge-

*) Es ist in Beziehung auf diesen Punkt von hohem Interesse, die Verhältnisse des Zuckers, der Oxalsäure und der Erdphosphate im Urin scrophulöser Kinder längere Zeit hindurch genau zu verfolgen. — Es scheint mir, daß sich folgende Erscheinungen bei fortgesetzter Beobachtung als ziemlich constant nachweisen lassen werden. — Bei jenen Kindern, die den s. g. »lymphatischen« Habitus mit klar ausgesprochenem scrophulösen Typus an sich tragen, findet man meistens eine geringe Quantität Zucker im Urin, dabei aber keine oder nur wenig Oxalsäure und einen nur wenig erhöhten Gehalt an Erdphosphaten. Die Kinder magern in diesem Zeitraume nicht ab, haben oft selbst ein leidlich gutes Colorit; bieten aber meistens gastrisch-catarrhalische Erscheinungen dar, sind mit Hautausschlägen, Blepharadenitiden u. s. w. behaftet. — Eine geeignete Behandlung führt die Zustände zur Besserung. — Die Beschaffenheit des Urins ändert sich damit bedeutend. Es findet sich kein Zucker mehr, aber viel Oxalsäure, und eventualiter viel Erdphosphate; sicher in Folge des Verlustes der letztern tritt zugleich eine geringe oder größere Abmagerung hervor; der »lymphatische, gedunsene« Habitus verliert sich. — Dies »Uebergangsstadium« zur Besserung, wie ich es in vorliegendem Falle nennen möchte, trägt also noch hinreichende Merkmale einer tiefgewurzelten Blutkrankung; dennoch ist es ein Schritt zur Besserung und die größte Aufmerksamkeit ist jetzt auf die Verminderung des Oxalsäuregehalts im Urin zu richten. Wird sie erreicht, schwindet selbst die Oxalurie ganz, so sind meistens auch die Gesammterscheinungen des scrophulösen Leidens gebessert; die Kinder können, unter Beibehaltung der für sie geeignet befundenen Diät, aus der Behandlung entlassen werden. Die sich anfangs in dem Zucker- und später in dem Oxalsäuregehalt des Urins aussprechende Retardation der Metamorphose der stickstoff-

mein gängige Ansicht, daß der Harnzucker der Diabetiker seine Quelle in den mehl- und zuckerhaltigen Bestandtheilen des Nahrungsmaterials habe. Man sah mitunter den Diabetes schwinden oder abnehmen, wenn man die Patienten auf eine fast exclusive Fleischdiät setzte, will wenigstens allgemein von dem Vermeiden zucker- und mehlhaltiger Nahrungsmittel Vortheil gehabt haben, und nichts war natürlicher, als zu schließen, der Zucker im Urin gehe aus diesen Nahrungsmitteln hervor. Die Erfahrung, daß man in dem diabetischen Urin keine oder nur höchst selten Oxalsäure fand, stimmte mit dieser Annahme vortrefflich; der Zucker der Nahrungsmittel erreichte in seiner Metamorphose nicht die Stufe der Oxalsäure, er blieb eben auf der des Zuckers stehen. Wenn aber ferner ein unmittelbares Hervorgehen der Oxalsäure aus dem im Urin enthaltenen Zucker vielfachen Beobachtungen zufolge überaus wahrscheinlich war, so war es weiterhin nur zu natürlich, zu schließen, daß wie der Harnzucker aus den mehl- und zuckerhaltigen Nahrungsmitteln, so auch die Oxalsäure aus diesen hervorgehe. Allein so klar diese Verhältnisse zu sein schienen, so wenig ich selbst daran zweifelte, als ich die Oxalsäure noch direct aus den mehl- und zuckerhaltigen Nahrungsmitteln herleiten zu müssen glaubte, es fehlt uns bis dahin jeder Beweis für die Annahme, daß der Harnzucker des Diabetikers wirklich aus den mehl- und zuckerhaltigen Nahrungsmitteln hervorgehe; es häufen sich im Gegentheil mehr und mehr Thatsachen, die zu der Ueberzeugung führen müssen, daß die stickstoffhaltigen Blutbestandtheile die directe Quelle des Harnzuckers bilden. Daß zunächst der Harn- und Milchzucker aus den stickstoffhaltigen Bestandtheilen des Blutes hervorgehen können, geht aus den Untersuchungen von Bensch über den Zuckergehalt der Milch ausschließlich mit Fleisch gefütterter Thiere hervor, und die Thatsache, daß Lebern von ausschließlich mit Fleisch gefütterten Thieren, mit Wasser gekocht, unzweifelhaft Spuren von Zucker entdecken lassen, bietet ebenfalls einen Beweis dafür dar. — Daß aber der Harnzucker der Diabetiker in vielen Fällen auch nothwendiger Weise aus den stickstoffhaltigen Blutbestandtheilen hervorgehen muß, auch dafür fehlt es nicht an Belegen. Ginge er nur aus den mehl- und zuckerhaltigen Nahrungsmitteln hervor, so müßte eine absolute Ausschließung derselben unbedingt immer zur Heilung führen. Man kann aber erfahrungsgemäß Diabetiker ausschließlich auf Fleischdiät setzen, und dennoch bleibt ihr Urin eben so zuckerhaltig als zuvor. Es steht diese Erfahrung so fest, daß man schon mehrfach beim Diabetes eine ganz gewöhnliche Kost erlaubt und nur die Quantität in summa beschränkt. Und hat man in andern Fällen bei Ausschließung mehl- und zuckerhaltiger Nahrungsmittel auch entschieden eine Besserung des Diabetes eintreten sehen, so stehen solche Beobachtungen durchaus nicht im Widerspruch mit der obigen Annahme. Was wir oben von der Oxalsäure gesagt, gilt ebenso vom Zucker. Geht er aus den stickstoffhaltigen Blutbestandtheilen hervor und erscheint er im Urin, so giebt uns der Zuckerharn einen ebenso offenbaren Beweis einer gehemmten, und zugleich qualitativ in irgend welcher Weise veränderten, Metamorphose jener Bestandtheile, als die Oxalurie es that. Zu solcher Hemmung giebt nun aber vor Allem Mißbrauch zucker- und mehlhaltiger Nahrungsmittel Anlaß, und schafft man diesen fort, so wird sicher auch meistens eine Besserung des Diabetes eintreten, d. h. die Metamorphose der stickstoffhaltigen Bestandtheile wird weniger gehemmt. — Sind demnach auch unzweifelhafte Beweise für das Hervorgehen der Oxalsäure aus Harnzucker, wenigstens von dem so oft zu beobachtenden Alterniren beider vorhanden, so ist ihr Zusammenhang nicht hinwegzuläugnen; aber es scheinen beide, der Zucker sowohl, wie weiterhin die Oxalsäure aus den stickstoffhaltigen Bestandtheilen hervorzugehen, und es ist mit all den Beobachtungen über Diabetes jene Behauptung, daß die Oxalurie direct durch einen Mißbrauch zucker- und mehlhaltiger Substanzen nicht veranlaßt werde, nichts weniger als widerlegt.

Es ist bis dahin nicht die Rede davon gewesen, ob die Oxalsäure auch aus andern als mehl- und zuckerhaltigen, stickstofffreien Bestandtheilen des Nahrungsmaterials herstammen könne,

haltigen Blutbestandtheile ist nicht mehr vorhanden. — Daß sich aber, wenn die Erscheinungen des s. g. Uebergangsstadiums lange anhalten, jene Formen des serophulösen Leidens herausstellen, welche durch beträchtliche Abmagerung, durch Geschwürsbildungen, durch Osteomalacie u. s. w. ausgezeichnet sind, bedarf nach dem, was ich früher über den Effekt großen Verlustes an phosphorsaurem Kalk mitgetheilt habe, kaum noch einer Erwähnung, und es kann demnach nicht sorgfältig genug auf die Entfernung der Erscheinungen dieses Stadiums, auf die rasche Beendigung desselben hingearbeitet werden.

ob namentlich nicht aus stickstofffreien Säuren, wie sie sich aus Fetten u. s. w. hervorbilden. Der Gedanke daran liegt so fern nicht. — Jene Säuren, welche Lehmann unter dem Namen der Buttersäuregruppe zusammengefaßt hat, wie die Ameisen-, Essig-, Metaceton-, Buttersäure u. s. w., wurden von Kolbe zuerst als gepaarte Oxalsäuren betrachtet. Seine interessanten Untersuchungen führten ihn zu der Annahme, daß die Ameisensäure eine Wasserstoffoxalsäure, die Essigsäure eine Methyloxalsäure, die Metacetonsäure eine Aethyloxalsäure, die Buttersäure eine Metaethyloxalsäure u. s. w. sei *). — Es ist sehr möglich, daß ein Zerfallen der Essig- und Buttersäure im Organismus in der Art stattfindet, daß sich die Oxalsäure von ihrem Radical trennt; allein es fehlt an allen Beweisen für die Wirklichkeit einer solchen Trennung; sie ist sogar unwahrscheinlich, wenn man sich erinnert, daß Jacubowitsch, Frerichs u. A. den Uebergang der Amylaceen in Rohrzucker und Milchsäure, und Frerichs weiterhin den Uebergang der Milchsäure in Buttersäure (unter Abgabe von Kohlensäure und Wasserstoff) nachwiesen, wir aber von mehl- und zuckerhaltigen Nahrungsmitteln sahen, daß sie nicht die directe Quelle der Oxalsäure sind. — Ueber den Einfluß eines reichlichen Genusses von Butter auf die Quantitäten der Oxalsäure im Urin weiß man nichts Bestimmtes. So weit meine geringe Erfahrung reicht, kann ich nur angeben, daß die an ausgeprägter Oxalurie leidenden Individuen sich besser ohne Butter, als bei dem Genusse derselben befinden. Von andern Fetten weiß man noch weniger. Nur von dem Leberthran kann ich behaupten, daß er den betreffenden Patienten meistens gut bekommt, und daß ich bei seinem Genuß die Oxalurie oft entschieden habe abnehmen sehen. Aber über seine Metamorphosen im Organismus ist nichts bekannt, und die aufgestellte Frage muß daher für jetzt in suspenso bleiben.

Wir gelangen

3. zu den unorganischen Bestandtheilen des Nahrungs- oder Bildungsmateriales des Organismus.

Auch in Mißverhältnissen ihrerseits können Ursachen der Oxalurie liegen. — Es sind insonderheit zwei der fraglichen

*) S. Lehmann, Physiol. Chem. Bd. I. 1850.

Bestandtheile, welche in Betracht kommen; das Eisen und die alkalischen Basen, in specie das Natron. In dem zu geringen Gehalt des Blutes an ersterm, und in dem zu großen Gehalt an letztern müssen wir ätiologische Momente der Oxalurie anerkennen. Was

a. den verminderten Eisengehalt, die Verminderung gefärbter Blutkörperchen,

anbetrifft, so sind wir über die spontane Entstehung der Chlorose noch keineswegs im Klaren. Fest dagegen steht, daß sie künstlich durch große Blutverluste herbeigeführt werden kann. Veranlaßt also der Mangel gefärbter Blutkörperchen, der chlorotische Zustand, Oxalurie, so muß eine solche nach großen Blutverlusten, vorausgesetzt, daß keine anderweitige Veränderungen in der Diät, dem Aufenthaltsorte u. s. w. des Patienten Statt hatten, auftreten oder, wenn sie vorher schon vorhanden war, zunehmen. Zum Belege, daß dies in der That der Fall ist, theile ich das Resultat einer genauen Beobachtung mit.

Wilhelm Jacobs, ein kräftiger Bursche von 16 Jahren, von frischem Colorit, dunkelm Haar, heiterm Temperamente, kam am 5. October 1850 in's deutsche Hospital in London. Er klagte über eine, seit 3 Jahren in der Entwicklung begriffene Geschwulst am rechten Oberschenkel, die ihn des zunehmenden Umfangs halber incommodirte. Die Eltern waren gesund. Er erinnert sich nicht jemals krank gewesen zu sein. Sein Allgemeinbefinden war ungestört. Eine bestimmte Ursache für die Entwicklung der Geschwulst war nicht aufzufinden. Die Diagnose wurde auf eine Exostosis femoris festgestellt. Patient entschloß sich gern zur Operation. Die Beobachtung einige Tage vor derselben ließ durchaus keine weitern Krankheitserscheinungen auffinden. Die Untersuchung des in je 24 Stunden entleerten Urins ergab ein specif. Gew. von 1016 — 1018, eine normale Farbe, einen normalen Säuregrad (+ 15 — + 18), einen normalen Gehalt an Erdphosphaten; es fand sich kein oxalsaurer Kalk, kein Albumen, kein Zucker. Es wurde während dieser Zeit eine quantitativ genau festgestellte „half diet“ genossen. Am 8. October Nachmittags fand die Resection der beträchtlichen Exostose Statt. Ich schätze den dabei erlittenen Blutverlust auf 24 Unzen. Darnach traten folgende Erscheinungen hervor:

1. Vom 8. bis 13. October. Es entwickeln sich ein bedeutendes Fieber, stark ausgeprägte Erscheinungen eines acuten Magencatarrhs mit bitterm Geschmack, und s. g. rheumatische Schmerzen im Kreuze. Leichte Stuhlretardation wird durch Ol. Ricini beseitigt. Diese Symptome halten bis zum 11. Oct. an; schwinden, und sind geschwunden am 13. October. Die Wunde heilt fast per primam intentionem. Am 12. zeigt sich eine geringe Quantität guten Eiters. Während der bezeichneten Zeit wird nichts genossen als Wassersuppe. Die täglich vorgenommene Urinuntersuchung ergiebt Folgendes: die Quantität ist nicht, außer an 2 Tagen verringert; sie beläuft sich auf 50, 70, 50, 35 und 45 Unzen. Die Farbe wird hochroth. Das specif. Gew. steigt auf 1025, 1020, 1025, 1023, 1022. Der Säuregrad steigt auf + 35, + 26, + 35, + 25 und macht am 13. einer ammoniakalischen Reaction Platz. Der Gehalt an Erdphosphaten übersteigt nicht das Normalmaaß (in einer Unze 0,15 — 0,25 Gran); oxalsaurer Kalk, Eiweiß und Zucker sind nicht vorhanden. — Ich habe früher nachgewiesen, daß alle fieberhaften Zustände das Auftreten der Oxalsäure im Urin ausschließen oder vorher vorhandene Quantitäten sich in ihnen verringern.

2. Vom 13. October bis 8. November. Die Wunde schließt sich bis zum 1. Nov. vollkommen. Am 2. Nov. kann Patient das Bein wieder gebrauchen. Das Allgemeinbefinden ist während der ganzen Zeit ungestört. Vom 18. Oct. an treten jedoch Erscheinungen der Chlorose, ein blasses Colorit, ein leichter Magencatarrh, zeitweilige Kopfschmerzen u. s. w. hervor. Es halten dieselben bis zum 6. — 8. November an. Saurer Geschmack findet sich nie. — Die Diät bestand vom 13. — 16. aus Milch und Brod Morgens und Abends und einer Fleischbrühe Mittags. Vom 16. Oct. bis 8. Nov. aus der frühern „half diet", zu dem täglichen Betrage von 1 Quart. Milch, 15 Unzen Brod, 4 Unzen Fleisch (zubereitet), 1 – 1½ Pfund Gemüse. An Arzneimitteln erhielt Patient vom 19. — 23. October Acida mineralia (Acid. hydrochl. dil. Acid. nitric. dil. aa ℥ β), vom 23. Oct. — 3. Nov. Tinct. ferr. sesquichlorid. 3ml tägl. 10 Tr.; vom 3. — 7. Nov. (eines Versuches halber) Acid. tartaric. 3ml täglich 10 Gran. — Der Speichel, welcher vom 8. — 13. Oct. Morgens 10 Uhr stets stark sauer reagirte, verlor in der zweiten Periode an Säure, wurde sehr schwach sauer oder leicht alkalisch. Der Urin bot folgende Erscheinungen dar: die Quantität war stets eine sehr beträchtliche; sie belief sich in der Regel auf 80 — 100 Unzen. (Die Quantität des genossenen Getränkes ist leider nicht notirt). Die Farbe war stets hell- oder mittelgoldgelb. Das specif. Gew. schwankte stets zwischen 1014 — 1020. Die Reaction war am 13., 14. u. 15. ammoniakalisch; vom 16. — 21. kaum sauer; vom 22. — 28. fand sich ein Säuregrad von + 10 — + 12; vom 28. Oct. — 2. Nov. trat wieder eine sehr schwach saure, am 2., 3. u. 4. eine alkalische, dann wieder saure Reaction ein, die sich bis zum 8. auf einen Säuregrad von + 20 hob. — Die Quantität der Erdphosphate vermehrte sich vom 13. — 15. Oct. von 0,15 — 0,2 Gran auf 0,45 — 0,5 Gran in einer Unze Urins; blieb mit geringen Schwankungen bis zum 1. Nov. auf dieser Höhe stehen und sank von da an gradatim bis zum 8. Nov. auf die normale Quantität herab. Gleichzeitig und ganz proportional diesen Schwankungen fand sich oxalsaurer Kalk vom 14. Octob. — 8. November; er schwand am 7. Nov. ganz.

3. Vom 8. bis 19. November. Ungestörtes Wohlbefinden. Die chlorotischen Erscheinungen sind geschwunden. Es wird Tinct. Bestuscheff zu 15 Tropfen 3ml tägl. gegeben. Der Urin bietet dieselben Erscheinungen dar, wie vor der Operation. Oxalsaurer Kalk wird nicht wieder beobachtet.

Es gehen also in dem vorstehenden Falle Auftreten, Zunahme und Schwund chlorotischer Elscheinungen ganz parallel mit dem Auftreten, der Zunahme und dem Schwunde der Oxalsäure im Urin einher. Die Diät, der Aufenthaltsort, alle übrigen Verhältnisse konnten nicht die Ursache zur Oxalurie abgeben. Es mußte dieselbe in dem erlittenen Blutverlust und dem dadurch herbeigeführten chlorotischen Zustande gesucht werden. — Ich bin der Ueberzeugung, daß es sich aus einer sorgfältigen Durchführung ähnlicher Beobachtungen als Thatsache herausstellen wird, daß der Mangel gefärbter Blutkörperchen und damit des Eisens eine entschiedene Ursache der Oxalurie bildet, und der Therapie für Verwundete, Operirte u. s. w., der Kritik der Lehre vom Aderlaß, wird damit ein neuer Anhaltspunkt gegeben. An jener Abnahme des Körpergewichtes, an jenem verringerten Nutritionsproceß, wie sie nach großen Blutverlusten Statt haben,

trägt sicher die durch die letztern veranlaßte Retardation der Metamorphose der Oxalsäure einen Theil der Schuld; sie führt zur Hinwegnahme einer für einen normalen Zellenbildungsproceß durchaus erforderlichen Quantität phosphorsauren Kalkes und beeinträchtigt damit diesen Proceß; sie kann nicht eher schwinden, als bis durch geeignete Diät und den etwaigen Gebrauch von Eisen der Verlust an gefärbten Blutkörperchen ersetzt ist, und die Oxydationsprocesse wieder ihre frühere Intensität erreichen. In der mangelhaften Durchführung dieser hat die Oxalurie, haben eine Menge anderer Erscheinungen ihren Grund, welche die Chlorose begleiten.

Es wurde bis dahin meines Wissens kein directer Beweis für die Entstehung von Oxalurie in Folge eines Mangels gefärbter Blutkörperchen beigebracht. Es fehlt dagegen nicht an Erfahrungen über den Schwund der Oxalurie in Folge einer Beseitigung jenes Mangels, und diese, in welcher Weise sie auch erfolgen mag, wird in vielen Fällen offenbar durch den Gebrauch des Eisens herbeigeführt. — Prout, G. Bird u. A. reden dem Eisen in seiner Anwendung gegen Oxalurie entschieden das Wort; ich selbst glaube seinem Gebrauche einige günstige Resultate zuschreiben zu müssen; auf seinem Mangel scheint der Mangel gefärbter Blutkörperchen, so wie die Verringerung der Intensität der Oxydationsprocesse zu beruhen; eine solche aber führt unter allen Umständen zur Oxalurie. — Ich darf es nicht unerwähnt lassen, daß je ausgeprägter die chlorotischen Erscheinungen sind, desto ausgeprägter in der Regel auch die Oxalurie ist. In dem Urine scrophulöser Kinder mit einem blassen, erdfahlen Colorit findet sich meistens eine bei Weitem beträchtlichere Quantität Oxalsäure, als in dem der Kinder mit frischem, blühendem Colorit; und bei jenen dyspeptischen Zuständen Erwachsener, die so häufig von Oxalurie begleitet sind, findet ein durchaus gleiches Verhältniß Statt. — Ich glaube daher auch auf Grund bisheriger therapeutischer Erfahrungen den Mangel gefärbter Blutkörperchen, und damit des Eisens als ein ätiologisches Moment der Oxalurie bezeichnen zu müssen. — Was

b. die Vermehrung der alkalischen Basen, in specie des Natron

anbetrifft, so wurde auch darauf in meinem zweiten Beitr. zur

physiol. Heilk. schon hingewiesen. Es scheint diese Vermehrung oft mit der Intensität der Oxalurie in proportionalem Verhältnisse zu stehen, und wenn wir auch noch den Mangel eines directen Nachweises derselben beklagen, eine Menge von Thatsachen liegt vor, die kaum einen Zweifel an ihrem Vorhandensein übrig lassen.

Was wir zunächst von dem Einflusse des Gebrauchs alkalischer Basen auf die Quantität der Oxalsäure im Urin wissen, so bemerkt Prout *): „I have reason to believe, that the free use of the liquor potassae occasions the development of oxalic acid in some individuals.“ — Lehmann giebt an **), daß die pflanzensauren Alkalien an und für sich eine Vermehrung des Kalkoxalats bedingen können; es verwandeln sich dieselben im Organismus bekanntlich in kohlensaure. — Ich selbst glaube ferner nach dem Gebrauch von kohlensaurem Natron eine gleiche Vermehrung der Oxalurie, wie Lehmann nach pflanzensauren Alkalien, beobachtet zu haben, und muß hier an die schon in meinem ersten Beitrage zur phys. Heilk. S. 69. nur angedeutete Beobachtung erinnern, daß man mitunter durch die längere Darreichung von kohlens. Natron Urinsedimente von phosphorsaurem Kalk hervorzubringen vermag. Es erschienen diese Sedimente zu Anfang des Natron-Gebrauchs (in Folge der Alkalescenz des Urins) in nur geringer Quantität, d. h. der phosphorsaure Kalk im Urin war zu Anfang nicht oder nur wenig vermehrt. Nach dem Gebrauch des Natron erlitten sie aber eine nicht unbeträchtliche Zunahme, d. h. der phosphorsaure Kalk wurde in vermehrter Quantität ausgeschieden. — Wir wissen jetzt, daß die Oxalsäure das Lösungsmittel für den phosphorsauren Kalk ist; in der durch den Natron-Gebrauch vermehrten Produktion derselben hätten wir demnach wahrscheinlich die Erklärung für die Zunahme der Kalk-Elimination zu suchen.

Zu zweit sprechen die Untersuchungen des Urins auf seinen Gehalt an basischem Alkali für die obige Annahme. Der Urin der an Oxalurie leidenden Individuen pflegt in der That oft um so reicher an basischem Alkali zu sein, je ausgeprägter die Oxalurie selbst ist. Meine bisher in Betreff dieses Punktes angestellten

*) l. c. pag. 564.

**) Physiol. Chem. I. pag. 50.

Beobachtungen erhalten durch die von Bence Jones schen früher gemachten eine Stütze. B. Jones sagt in den Philos. transact. Part II. 1845. pag. 344.: „It not unfrequently happens, „that alkalescence (of the urine) is caused by fixed alkaline „salts in those, who, though not ill, yet suffer from indige„stion, whilst leading sedentary lives. I have more especially „observed it, where the octahedral crystals, usually supposed „to be oxalate of lime, have been present." — Ein Ueberschuß des Urins an alkalischen Basen kann aber nur entweder durch eine vermehrte Einnahme derselben oder durch eine verminderte Ausscheidung auf einem andern dazu bestimmten Wege erfolgen; es ist offenbar, daß in beiden Fällen ein vorübergehender oder anhaltender Ueberschuß derselben Basen im Blute vorhanden sein muß. Verfolgen wir aber Aetiologie und Diagnose genauer, so ergiebt sich auch in der Regel, daß die betreffenden Patienten entweder sehr viel Kochsalz oder sodahaltige Wässer, neben Fleischkost reichliche Quantitäten der an alkalischen Basen reichern Vegetabilien genossen, oder daß sie an einer Hyperämie der Leber leiden, die bald in Folge diätetischer Ausschweifungen, bald in Folge des „sedentary life", bald in Folge andauernder Gemüthsbewegungen entstehen kann. Wir dürfen vielleicht annehmen, daß in diesen Zuständen die Secretion der Galle eine beschränkte ist, daß die Ausfuhr einer bestimmten Quantität von Natron auf diesem Wege abgeschnitten wird, und erhalten damit in beiden Fällen Anhaltspunkte für die Erklärung jenes Ueberschusses des Blutes an alkalischen Basen, der in der höhern Alkalescenz des Urins seinen Ausdruck findet.

An dritter Stelle suche ich endlich einen Beleg für die Vermehrung dieser Basen in einer Erscheinung, die bei keiner Classe von Patienten so oft vorkommt, als denen, die an Oralurie leiden. Es ist dies eine intensive Säurebildung in „den ersten Wegen." — Es tritt dieselbe insonderheit bei chlorotischen Individuen oft und stark hervor. Sofort nach dem Genusse von Mahlzeiten, namentlich solcher, die an Amylon oder Zucker reich waren, stellt sich ein stark saurer Geschmack ein. Weitere Erscheinungen der Dyspepsie, belegte Zunge, Flatulenz, Druck in der Magengegend fehlen dabei selten. — Gegen diese Zustände hat man vielfach, ja immer, die Antacida empfohlen; ich selbst habe namentlich das Natr. carbonic. gebraucht; es ist wahr

und leicht erklärlich, für den Augenblick nehmen sie die Säure fort. Allein alsbald tritt sie nur immer wieder stärker hervor, und bei Patienten, die früher nicht an Säurebildung litten, sah ich dieselbe mitunter auftreten, sobald sie längere Zeit hindurch Soda gebrauchten. Ich glaubte daraus schließen zu müssen, daß ein pathologisches Plus von Natron die Säurebildung nur vermehre, und der Erfolg, den die Anwendung von Mineralsäuren in diesen Fällen hatte, diente dazu, eine solche Ansicht zu bestätigen. Die Mineralsäuren, deren Gebrauch doch nothwendig zur Hinwegnahme oder Neutralisation einer Quantität basischen Alkali's führen muß *), führen entschieden zur Tilgung jener Säurebildung, — eine Erfahrung, welche ein Jeder leicht bestätigt finden kann; — wir müssen also auch in dem pathologischen Plus an Natron die Ursache der vermehrten Säurebildung suchen. — Nur zu wichtig ist die Bestätigung, welche dieser der Erfahrung am Krankenbette entnommene Schluß, durch die Mittheilungen und Untersuchungen von Frerichs erhält. F. sagt in seinem ausgezeichneten Artikel: „Verdauung" in Wagner's Handwörterb. d. Physiol. pag. 757.: „Von besonderm Interesse werden „Alkali und Säure durch den Einfluß, welchen sie auf die Fer„mentkörper üben. Der Magensaft äußert nur bei Gegenwart „freier Säure (Milchsäure) seine eigenthümliche Kraft; das Alkali „wirkt dadurch bestimmend auf die Umsetzungsprodukte, als „es **) die Metamorphose der Kohlehydrate zu „Milchsäure u. s. w. beschleunigt." — Die weitere Beobachtung (s. l. c. pag. 772.), daß die durch Vermischung eines alkalischen Speichels mit Amylon bedingte Metamorphose des letzteren zu Zucker weit langsamer erfolgt, wenn der Speichel durch Salz-, Essig- oder Schwefelsäure sauer gemacht wird, führt zu gleicher Annahme; wir dürfen somit nicht anstehen, in der Vermehrung der alkalischen Basen im Organismus eine Ursache beschleunigter Säurebildung im Magen zu suchen. — Kommt nun aber diese Erscheinung insonderheit bei an Oralurie leidenden Individuen vor, bessert sich nicht allein sie, sondern

*) S. Archiv f. physiol. Heilk. X. p. 479. Miquel: über den Gehalt des Urins an alkalischen Basen nach Einnahme von Schwefelsäure.

**) Im Originale steht statt »es«: »sie«. Es ist dies nach brieflicher Mittheilung des Hr. Verf. ein Druckfehler. »Es« (das Alkali) ist richtig.

die Oralurie selbst auch nach vielfachen Erfahrungen (Prout, Golding Bird u. A.) bei dem Gebrauche von Mineralsäuren, so können wir auch nicht umhin, jener Vermehrung eine wichtige Rolle in der Aetiologie der Oralurie zuzuschreiben; — ein Satz, der für die Therapie offenbar von hoher Bedeutung ist. —

Die Thatsache läßt kaum einen Zweifel zu. In welcher Weise kann aber die Vermehrung der alkalischen Basen und die damit herbeigeführte Beschleunigung der Säurebildung im Magen Anlaß geben zur Oralurie? — Die Frage ist schwer zu beantworten. Als ich die Oralsäure noch direct aus den stickstofffreien Bestandtheilen des Nahrungsmateriales herleitete, glaubte ich auch in der durch dasselbe veränderten Metamorphose diese eine Erklärung suchen zu müssen. Diese Ansicht kann jetzt nicht mehr in Frage kommen. — Dagegen wage ich eine andre Vermuthung auszusprechen, nach welcher das Natron nicht direct, sondern indirect auf die Oralurie von Einfluß ist. — Wenn jene Erscheinungen der Säurebildung u. s. w. auf einem Ueberschuß an alkalischen Basen hinweisen, so muß ich nochmals besonders hervorheben, daß diese Erscheinungen namentlich bei Individuen hervortreten, die ein chlorotisches, erdfahles Colorit tragen. Sollte nun eine vermehrte Säurebildung im Magen nicht vielleicht irgendwie die weiterhin erfolgende Bildung gefärbter Blutkörperchen, durch Herstellung einer abnormen Verbindung des Eisens, beeinträchtigen? Sollte ferner nicht ein erhöhter Alkaligehalt des Magensaftes innerhalb des Magens eine ähnliche Zersetzung der Labzellen und damit eine Beeinträchtigung der Metamorphose der stickstoffhaltigen Nahrungsbestandtheile herbeiführen, wie es außerhalb desselben der Fall ist? Sollten sich nicht in dieser Weise, in letztem Gliede also durch das Plus an alkalischen Basen, die chlorotischen Zustände spontan heranbilden? In Betreff der ersten Frage, so bemerkt Frerichs (l. c. pag. 801.), „die Auflösung der Erden und Metalle im „Magen erfolgt nach reinen Affinitätsgesetzen und findet ihre „Gränze in der beschränkten Menge der Säure." Eine pathologische Vermehrung der Säure muß zur Herstellung abnormer Verbindungen führen, so wenig uns auch noch thatsächlich über die unorganischen Bestandtheile der Nahrungsmittel in dieser Beziehung bekannt ist. — Und in Bezug auf die zweite Frage, muß nicht im Magen dasselbe eintreten, was außerhalb desselben

Statt hat, d. i. eine putride Zersetzung der in Wasser vertheilten, und mit kohlensaurem oder kaustischem Alkali versetzten Labzellen? Frerichs sagt davon (l. c. pag. 778.): „die Masse „bekommt nach und nach einen penetranten Fäulnißgeruch, es „entwickelt sich Schwefelwasserstoff-Ammoniak, die Umsetzung hat „eine ganz andre Richtung genommen." Die interessante spätere Bemerkung von Frerichs (l. c. pag. 804.), daß in den erbrochenen Massen chlorotischer Mädchen wiederholt eine enorme Quantität von Hefenpilzen, so wie die Entwicklung von Kohlensäure beobachtet wurde, daß in solchen Fällen eine wahre Gährung stattfinde, welche mit der Bildung von Essigsäure ihr Ziel erreiche, steht damit im Einklang. — Es muß endlich in jener Vermehrung alkalischer Basen seinen Grund haben, daß man bei Chlorohen oft Monate lang vergeblich Eisen anwendet; man findet das Eisen nicht selten im Urin wieder; daß aber in eben diesen Fällen das Eisen wieder zu erfreulichen Resultaten führt, wenn man seinem Gebrauche den von Mineralsäuren vorausschickt. Ich kann mich demnach der Vermuthung nicht entschlagen, daß in dem überschüssigen Alkali die letzte Ursache zu den beregten chlorotischen Zuständen liegt; und daß endlich, wenn jenes auf die Herbeiführung der Oralurie von Einfluß ist, dieser Einfluß in dieser Weise, d. h. durch Beeinträchtigung der Bildung gefärbter Blutkörperchen und damit der normalen Oxydationsprocesse ausgeübt wird.

Damit schließen wir die Betrachtung des Nahrungsmaterials in seiner Beziehung zur Oralurie ab. — Mißbrauch stickstoffhaltiger sowohl, als stickstofffreier Substanzen, Mangel an Eisen und Vermehrung alkalischer Basen lernten wir als Ursachen der letztern kennen. Was sich daraus für die Therapie ergiebt, ist zu klar, als daß es einer besondern Bemerkung bedürfte. — Wurde jedoch schon oben auf den wohlthätigen Einfluß der Verminderung stickstoffhaltiger Bestandtheile der Nahrungsmittel hingewiesen, wurde für gewisse Fälle von Oralurie eine vorwaltend vegetabilische Diät empfohlen, so darf nicht vergessen werden, daß wir stickstoffhaltige sowohl, als stickstofffreie Substanzen fast nur in Verbindung mit gewissen anorganischen Stoffen genießen und von Seiten dieser bei der fraglichen Modification der Diät stets besondere Vorsichtsmaaßregeln erforderlich gemacht werden. — Wird einem Individuum statt einer aus Fleisch und

Vegetabilien gemischten, eine fast rein vegetabilische Kost gereicht, so wird damit allerdings die Quantität stickstoffhaltigen Nahrungsmaterials verringert; allein man entzieht gleichzeitig dem Organismus eine Quantität phosphorsaurer Salze, die er zu seiner normalen Existenz bedarf, und führt ihm mit der Pflanzenkost andrerseits eine größere Quantität alkalischer Basen, oder pflanzensaurer und kohlensaurer Salze zu, als er erfordert. Letztere lernten wir aber oben geradezu als Vermittler oder Befördern der Oralurie kennen, und wir würden also mit der vegetabilischen Kost auf der einen Seite verlieren, was wir auf der andern gewinnen. — Dennoch ist die Verringerung der stickstoffhaltigen Blutbestandtheile nicht anders als mit dieser vegetabilischen Kost zu erreichen; wir müssen auf sie greifen; nur muß gleichzeitig das damit herbeigeführte Mißverhältniß in den unorganischen Bestandtheilen künstlich ausgeglichen werden. Ich habe je nach den individuellen Verhältnissen der vegetabilischen Diät bald etwas Phosphor- oder Salz- und Salpetersäure, bald etwas Eisen und Kalk (kohlensauren oder phosphorsauren) zugesetzt; ich suchte damit den Ueberschuß an alkalischen Basen zu neutralisiren und einen etwaigen Mangel an phosphorsauren Salzen zu ersetzen; und ich glaube durch eine in dieser Weise künstlich modificirte Diät meinen Zweck, die Abnahme der Oralurie, erreicht zu haben. — In dieser Weise behandelt, nimmt die Diät immer den obersten Platz in der Therapie ein. Oder wird die Qualität des Blutes nicht in oberster Reihe von der Qualität und Quantität der einzelnen Nahrungsbestandtheile bedingt?*) — Ein zweiter Punkt, welcher bei der angegebenen Veränderung der Diät zu berücksichtigen ist, ist die Verschiedenheit des Einflusses, den eine animalische und eine vegetabilische

*) Es darf hier nicht unerwähnt bleiben, daß, wenn die alkalischen Basen einerseits, und wahrscheinlich in Folge der beschleunigten Säurebildung, die Oralurie befördern, sie andrerseits als solche die Metamorphose der stickstoffhaltigen Blutbestandtheile beschleunigen und damit der Oralurie hindernd in den Weg treten. Auf solcher Wirkung beruht sicher zum Theil der wohlthätige Einfluß mancher Mineralwässer, wie z. B. derer von Ems u. s. w. — Die Beurtheilung dieser Verhältnisse ist schwierig. Aeußerer Habitus, Colorit, Urinuntersuchungen u. s. w. müssen zur Indication benutzt werden. — Wo sich Erscheinungen von Säurebildung vorfinden, erfordert der Gebrauch der Alkalien die größte Vorsicht.

Kost einmal auf die Verdauungsorgane selbst und andrerseits auf das Nervensystem ausübt. Man nennt die animalische Diät oft und nicht mit Unrecht eine „stimulirende". In vielen Fällen hört man von an Oralurie leidenden Individuen die Aeußerung: „ich muß Fleisch essen, um mir Kraft zu geben." — Die Patienten fühlen sich oft den ganzen Morgen hindurch matt und schwach und leben erst nach einem guten Mittagsmahle wieder auf. — Die Qualität der Mahlzeit ist ohne Frage von bedeutendem Einfluß auf das Nervensystem, und welche Bedeutung dieses wieder für den Ablauf der Stoffmetamorphose hat, werden wir unten sehen. Allein die weniger stimulirende Eigenschaft einer vegetabilischen Mahlzeit ist deshalb keine Contraindication für ihre Anwendung. Auch hier ist eine künstliche Hülfe möglich und ein Glas leichten Weines, einige Tropfen der Valeriana-Tinctur u. s. w., eine geistige Anregung, scheinen in bester Weise zu ersetzen, was die vegetabilische Diät in dieser Beziehung zu wünschen übrig ließ. Die schwierige Aufgabe, die Verringerung stickstoffhaltiger Blutbestandtheile wird aber erfüllt und damit dem Fortbestande der Oralurie die Hauptquelle entzogen. — Kräftiger schon ist der Einwand, der von Seiten der Schwerverdaulichkeit der Vegetabilien im Allgemeinen gegen eine längere Fortsetzung der vegetabilischen Diät erhoben wird. Das Pflanzenfaser- und Zellgewebe ist den lösenden Verdauungsflüssigkeiten unbezwingbar und geht unverdaut mit den Excretis wieder fort. Stellen sich daher Erscheinungen von hoher Reizbarkeit der Verdauungsorgane, von Flatulenz u. s. w. heraus, so muß allerdings die vegetabilische Diät ausgesetzt und mit einer leichten animalischen Kost vertauscht werden. — Jeder einzelne Fall wird in dieser Beziehung jedoch besondere Maaßregeln erforderlich machen; genüge es deshalb nur im Allgemeinen auf die vorstehenden Punkte aufmerksam gemacht zu haben.

Wenn nun nach meinen frühern Mittheilungen die Oralurie und der damit verbundene Verlust von Erdphosphaten in der Entwicklungsgeschichte keines Leidens eine so hervorstechende Rolle spielt, als in der des tuberkulösen und scrophulösen, und wenn wir dieselbe nach sämmtlichen vorstehenden Bemerkungen als auf einer retardirten Metamorphose der stickstoffhaltigen Blutbestandtheile beruhend betrachten müssen, so muß damit auch eine solche Retardation als dem Wesen des tuberkulösen oder scrophulösen

Krankheitsprocesses eigenthümlich bezeichnet werden. — Ich muß damit die auf pag. 102. meines mehrerwähnten Schriftchens ausgesprochene Ansicht, daß diese Krankheitsprocesse eben durch eine beschleunigte Metamorphose der stickstoffhaltigen Blutbestandtheile ausgezeichnet seien, für falsch erklären; durch die irrige Anschauung von dem directen Hervorgehen der Oxalsäure aus stickstofffreien Verbindungen war sie hervorgerufen; die Vermehrung der Harnsäure und die zeitweilige Vermehrung des Harnstoffs glaubte ich, wenn auch in unbekannter, doch in ähnlicher Weise erklären zu müssen, wie sie durch starke Bewegung u. s. w. herbeigeführt wird. — Die jetzige These wird, wie ich glaube, den Thatsachen Stand halten; sie wird uns weiterhin richtige Fingerzeige für eine erfolgreiche Behandlung der Leiden an die Hand geben. — Ein genauer Verfolg ihrer Entwicklung weist in der That stets auf Verhältnisse hin, welche Aussprüche für eine retardirte Metamorphose der stickstoffhaltigen Blutbestandtheile sind; einerlei ob das Leiden ererbt oder ob es spontan entstanden ist. Dort litten die Eltern selbst an Tuberkulose, oder sie litten an jenen Erkrankungen des Blutes, die sich durch s. g. Hämorrhoidalleiden, durch Rheumatismen, durch gichtische Erscheinungen kund geben, die aber sämmtlich mit absoluter Vermehrung stickstoffhaltiger Blutbestandtheile einhergehen; hier begegnen wir in der langen Stufenreihe von Erscheinungen, welche bis zur vollkommnen Ausbildung der Dyskrasie durchlaufen wird, bald reichlichen Ablagerungen von Albuminaten in den Drüsen, bald einer anhaltenden Bildung von Ekthyma, Furunkeln und Abscessen; bald wieder einer oftmaligen Albuminurie und Vermehrung des Harnsäure- und Harnstoffgehalts des Urins; bald und fast immer endlich einer stark ausgeprägten Oxalurie. — Und wenn eine alte Erfahrung den Mißbrauch zucker- und mehlhaltiger Nahrungsmittel insonderheit scrophulösen Kindern verbietet, wurde damit nicht unbewußt einer Retardation der Metamorphose stickstoffhaltiger Blutbestandtheile, und damit wieder der Oxalurie, so wie dem Verluste an Erdphosphaten entgegengearbeitet? — Es ist keine Frage, man kann und darf scrophulöse Kinder auf animalische Kost setzen; der Mißbrauch von Amylaceen ist ihnen entschieden schädlich. Allein es darf dabei nie vergessen werden, daß die stickstoffhaltigen Blutbestandtheile, also die animalische Diät, die directe Quelle der Oxalsäure bilden; — und ich möchte

deshalb auch hier wieder auf jene künstliche Modification einer vegetabilischen Diät verweisen, welche ich oben erwähnte. — Ich habe scrophulöse Kinder auf fast ausschließliche animalische Kost gesetzt, und bei der sorgfältigsten Regulirung der quantitativen Verhältnisse, je nach dem Gehalte des Urins an stickstoffhaltigen Substanzen und Oxalsäure, allerdings oft gute Erfolge derselben gesehen. In einigen Fällen erreichte ich damit aber dennoch den Schwund der Oxalurie nicht, und in diesen suchte ich eben eine Kost zu reichen, die mit den organischen Verbindungen der Vegetabilien die unorganischen der animalischen Nahrungsmittel verband. Ich gab den Kindern wenig Brod, Mittags frische Gemüse, Kartoffeln nicht ausgenommen, wenig Milch, ein leicht verdauliches Fett, am liebsten Leberthran, und reichte ihnen dabei etwas Phosphor- oder andre Mineralsäuren, etwas Eisen und meistens kohlens. oder phosphorsauren Kalk. — Mögen weitere Untersuchungen entscheiden, ob ich Recht habe, wenn ich glaube, daß sich die Patienten in Folge dieser Behandlung besserten; die Aufgaben wenigstens, welche uns eine rationelle Therapie stellt, scheinen damit erfüllt zu werden; der Gehalt des Blutes an stickstoffhaltigen Verbindungen muß abnehmen, ohne daß in dem Normalbestande der unorganischen Bestandtheile eine erhebliche Veränderung eintritt *).

Damit kehren wir zu dem an die Spitze gestellten Satze zurück, daß die Oxalurie, falls sie nicht etwa auf Zufälligkeiten beruht, in einer Störung der Metamorphose der Blutbestandtheile ihren Grund hat, daß diese Störungen aber immer nur von zwei Seiten, entweder von Seiten des zu metamorphosirenden Materiales, oder von Seiten der das Material in Angriff nehmenden Angentien, der atmosphärischen Luft und dem belebten Organe, bedingt werden können. Die Störungen auf

*) Sollte nicht auch der Erfolg, den man bei tuberkulosen Individuen von dem Gebrauche der süßen Molken gesehen haben will und steht, zum größten Theil von der Mischung abhängen, in welcher hier organische und unorganische Bestandtheile gereicht werden? Die Molken sind eine Milch ohne Caseïn; Milchzucker, ein Theil des Fettes, und die unorganischen Bestandtheile der Milch bleiben übrig; sie bilden eine Flüssigkeit, die unsern obigen Auseinandersetzungen gemäß in den fraglichen Leiden die Stelle eines rationellen Heilmittels einnimmt, vorausgesetzt, daß die übrige Diät des Patienten auch entsprechende Modificationen erfährt.

Seiten des erstern haben wir in Betracht genommen; wir sprechen

II. Von den Störungen der das Nahrungs- oder Bildungsmaterial des Organismus angreifenden Agentien, als letzten Ursachen der Oxalurie.

Was zunächst die atmosphärische Luft anbetrifft, so ist es klar, daß wenn die Oxalurie auf einer Retardation der Metamorphose stickstoffhaltiger Blutbestandtheile beruht, und diese Metamorphose durch die Aufnahme einer bestimmten Quantität von Sauerstoff bedingt wird, Alles, was die letztere beeinträchtigt, zur Oxalurie Anlaß geben muß. — Von einer absoluten Abnahme des Sauerstoffgehaltes kann nur unter außergewöhnlichen Verhältnissen, in enggeschlossenen Räumen u. s. w. die Rede sein; der Gehalt der freien atmosphärischen Luft an Sauerstoff bleibt sich stets mehr oder weniger durchaus gleich. — Dagegen verursachen Schwankungen der Temperatur in bekannter Weise Schwankungen des inspirirten Sauerstoffquantums, und in dieser Weise können die Oxydationsprocesse im Organismus möglicherweise eine Störung erfahren. — Es erhellt dies am deutlichsten aus den Versuchen von Vierordt, nach welchen der Verbrauch von Kohlenstoff mit der Zu- und Abnahme der Lufttemperatur fällt und steigt; es wird darnach in einer höhern Temperatur unter übrigens gleichbleibenden Verhältnissen die Oxydation vorhandener Oxalsäure langsamer und in geringerm Grade erfolgen müssen, als in einer niedrigern. — Man findet demgemäß auch oft, daß sich an Oxalurie leidende Patienten an klaren, heitern Wintertagen am besten befinden, daß sie im Sommer dagegen leicht über entsetzliche Mattigkeit und Abgeschlagenheit klagen; Erscheinungen, welche sehr häufig und andrerseits so leicht verständlich sind, daß sie keiner weitern Erklärung bedürfen.

Ein anderes ätiologisches Moment der Oxalurie kann in einem zu großen Wassergehalt der Luft und einer unzureichenden Ventilation derselben liegen. Insonderheit durch erstern wird die Wasser- und Kohlensäure-Ausgabe durch Haut und Lunge beeinträchtigt, die Oxydationsprocesse im Organismus erleiden damit wieder eine Störung, und Oxalurie ist eine unausbleibliche Folge. — In dem schätzenswerthen Werke Prout's „On stomach

„and renal diseases" lesen wir: „Among exciting causes of the „oxalic acid diathesis, one of the most striking I am acquainted „with, is a residence in a damp and malarious district." Immerhin mögen Armuth, unzureichende Nahrung u. s. w. bei den fraglichen Individuen einen Theil der Schuld tragen; ich glaube jedoch mitunter den unmittelbaren und nachtheiligen Einfluß der feuchten Luftbeschaffenheit beobachtet und nachgewiesen zu haben. Die betreffenden Patienten befinden sich an feuchten und namentlich naß-kalten Tagen am schlechtesten, und ich habe an solchen Tagen wohl größere Quantitäten oxalsauren Kalkes in ihrem Urin gefunden, als an andern. — Andrerseits ist es bekannt, daß ein zeitweiliger Ortswechsel, ein Aufenthalt auf Bergen oder an der Seeküste die Oxalurie schwinden macht oder wenigstens bessert, ohne daß in der Bewegung oder Diät wesentliche Veränderungen vorgenommen würden. Es werden diese Beobachtungen nicht selten bei Herrn gemacht, die Tag für Tag in der meistens trüben, feuchten Atmosphäre London's zu leben genöthigt sind und einen Landaufenthalt oder einen Aufenthalt an der Seeküste zur Erholung benutzen. Die Ausspannung aus Geschäften, die Entfernung aller aufregenden Gemüthseindrücke ist hier in Anschlag zu bringen; allein auch bei Kindern, bei denen von solchen Dingen nicht wohl die Rede sein kann, wird dieselbe Beobachtung gemacht. Die Quantität des im Urin entleerten oxalsauren Kalkes fand ich bedeutend geringer bei scrophulösen Kindern, die sich an der Seeküste in Margate (Kent) aufhielten, als bei solchen, die beständig in London lebten, wiewohl die Quantität ihrer Nahrungsmittel dort eine größere war, als hier. — Die Beobachtungen über das Verhältniß der Oxalsäure im Urin zu der Beschaffenheit und den Veränderungen der Atmosphäre, in verschiedenen Klimaten u. s. w. sind nur noch sehr mangelhaft; ein genauerer Verfolg derselben wird zu manchen interessanten Aufschlüssen, gewiß aber auch zu Bestätigungen der wenigen, vorliegenden Erfahrungen führen.

Als ein hervorragendes, ätiologisches Moment der Oxalurie wird Mangel an Bewegung in freier Luft bezeichnet. Bei Beurtheilung solcher Angaben hat man jedoch meistens Quantität und Qualität der genossenen Nahrungsmittel, so wie weitere Lebensverhältnisse nicht in Anschlag gebracht, und sie dürfen deshalb nur mit Vorsicht aufgenommen werden. Im Allgemeinen

läßt sich nur so viel mit Bestimmtheit behaupten, daß bei geringer Anzahl von Respirationen, geringer Muskelaction, langsamer Blutbewegung eine geringere Quantität von Nahrungsmaterial erforderlich ist, als da, wo tägliche Bewegung und Anstrengung in freier Luft stattfindet. Wird daher unter den ersteren Verhältnissen die Quantität des Nahrungsmaterials dem Minderverbrauch entsprechend verringert, so wird sich schwerlich Oxalurie einstellen; findet eine solche Verringerung nicht Statt, so wird sich sicher alsbald oxalsaurer Kalk, als Zeichen einer retardirten Metamorphose im Urin nachweisen lassen. Es kann keinem Zweifel unterliegen, daß die Therapie in diesem Falle ebensowohl durch Verringerung des Nahrungsmaterials, als durch Vermehrung der Respirationsakte, Beschleunigung der Circulation und gesteigerte Muskelthätigkeit, wie sie die Bewegung in freier Luft bedingt, erfreuliche Resultate erzielt. Ein gesteigerter Oxydationsproceß, eine beschleunigte Metamorphose ist das Resultat jener einzelnen Actionen (Vgl. Lehmann's Artikel: „Harn" in Wagner's Handwörterb. d. Physiol. Bd. I.). — Allein will man die körperliche Bewegung und Anstrengung in dieser Weise benutzen, so ist dabei eine Doppelwirkung nie zu übersehen. Der Muskelanstrengung folgt die Ermattung und sie eben vermag den beschleunigenden Einfluß der Bewegung auf die Metamorphose zu paralysiren, ja unter Umständen eine Retardation herbeizuführen, die die Beschleunigung an Größe der Wirkung übertrifft. — Ich habe früher selbst an Oxalurie leidenden Individuen angestrengte Märsche anempfohlen und ihnen gerathen, das sich dabei einstellende Gefühl der Ermattung zu überwinden. Allein ich fand mehrfach an den solchen Anstrengungen folgenden Tagen nur eine Vermehrung der Oxalsäure im Urin; die Patienten fühlten sich matter als zuvor; und wenn auch nach mehrfacher Wiederholung jener Anstrengungen die Oxalurie mitunter abnahm, wenn ihre augenblickliche Zunahme vielleicht in einer in Folge der Bewegung vermehrten Produktion von Harnsäure und in deren Zerfallen ihre Erklärung fand, es sollte dennoch niemals jene Doppelwirkung körperlicher Anstrengungen übersehen, dieselben wenigstens niemals bis zur Ermattung fortgesetzt werden.

In zweiter Reihe werden die Metamorphosen im Organismus von Seiten des belebten Organs bedingt. — In

pathologischen Zuständen desselben finden wir eine Menge von ätiologischen Momenten für die Oxalurie. — Anknüpfend an die obigen Betrachtungen müssen wir voranstellen, daß eine Mehreinnahme, ein Ueberschuß an stickstofffreiem Nahrungsmaterial, die Metamorphose des stickstoffhaltigen retardirt, daß ferner bei einer Mehreinnahme, einem Ueberschuß an stickstoffhaltigem Material die Metamorphose desselben ebenfalls nicht zu Ende geführt wird. In beiden Fällen kann Oxalurie als Symptom der Retardation der Stoffmetamorphose auftreten. Ein durchaus gleiches Resultat muß sich aber herausstellen, wenn die Metamorphose des stickstofffreien oder des stickstoffhaltigen Materials in Folge einer materiellen oder functionellen Störung eines Organs retardirt wird, in der Quantität und Qualität der Einnahmen die Ursache der Oxalurie also nicht liegt. Der Betrachtung dieser Störungen wenden wir uns jetzt zu.

Von drei Seiten wird das dem Organismus dargebotene Nahrungsmaterial namentlich in Angriff genommen; von Seiten der Verdauungsorgane, von Seiten der Circulations- und Respirationsorgane und indirect von Seiten des Nervensystems.

Was zunächst die Verdauungsorgane anbetrifft, so steht es erfahrungsmäßig fest, daß fast jeder von Oxalurie begleitete Krankheitsvorgang auch von mehr oder weniger intensiven Catarrhen der Magenschleimhaut begleitet ist. Prout sagt l. c. pag. 69: „There is a positive derangement of the „converting function of the stomach"; „the non-assimilation of „oxalic acid taken as food and the mal-assimilation of saccha„rine aliments, and in extreme cases perhaps, of albuminous „and oleaginous aliments are proximate causes of the presence „of oxalic acid in the system." — Golding Bird sagt l. c. pag. 233: „Among chronic diseases especially in certain forms „of chronic dyspepsia, attended by gastralgia, oxalate of lime „often abounds in the urine." — Unter meinen eigenen Beobachtungen findet sich kaum ein Fall, in welchem nicht neben der Oxalurie Erscheinungen der Dyspepsie vorhanden gewesen wären. — Belegte Zunge, insonderheit constanter Beleg an der Wurzel, von fast untilgbarem Hunger bis zur Abscheu vor Speisen wechselnder Appetit, leichte und intensive Säurebildung nach dem Genuß von Speisen, Gasentwicklung im Magen, Druck im Epigastrium nach der Mahlzeit, diese sämmtlichen Erscheinun-

gen deuten oft hinreichend auf gestörte Secretion und Innervation des Magens und auf eine in Folge davon gestörte Metamorphose der Nahrungsmittel hin. — Die einzelnen Glieder dieser Störung sind noch weniger bekannt; wir vermuthen nur mit einiger Sicherheit eine Alteration der Fermentkörper und eine in Folge davon veränderte Metamorphose der Albuminate*). Mit Bestimmtheit läßt sich dagegen behaupten, daß vermehrte Milch- und Buttersäurebildung im Magen ein fast constanter Begleiter dieser Zustände ist, mag sie nun in Folge einer durch die catarrhalische Beschaffenheit der Magenwände verlangsamten Resorption der Nahrungsmittel, mag sie in dem Vorgange wahrer Gährungsprocesse (Pilzbildung, Entwicklung von Kohlensäure und Essigsäure) oder mag sie in einer beschleunigten Metamorphose der Amylaceen im Magen selbst (erhöhter Alkaligehalt der Verdauungssäfte) ihren Grund haben. — Von dieser Säurebildung aber sahen wir oben, daß sie zur Oralurie Anlaß zu geben vermag, und es würde demnach die Reihenfolge der hier in Frage stehenden Vorgänge etwa folgende sein:

Catarrh der Magenschleimhaut — Vermehrte Milch- und Buttersäurebildung; abnorme Fermentationsprocesse im Magen — Beeinträchtigte Bildung gefärbter Blutkörperchen (erfahrungsmäßig, aber noch nicht experimentell bewiesen) — verminderter Oxydationsproceß und verringerte Innervation (typische Kraft) des Gesammt-Organismus — direct und indirect retardirte Metamorphose der stickstofffhaltigen Blutbestandtheile — Oralurie.

Wo aber liegt die Quelle des ersten Gliedes? Der Catarrh der Magenschleimhaut kann sicher zur Oralurie führen, oder eine vorhandene Oralurie steigern, allein als letzte Ursache derselben darf er, wie mir scheint, nicht betrachtet werden. Er selbst ist nur Symptom eines localen Magenleidens oder einer primären oder secundären Bluterkrankung. In diesen ist die Ursache zum Catarrh, ist die letzte Ursache zur Oralurie zu suchen. — Die Hyperämie der Magenschleimhaut kann zunächst eingeleitet werden durch den Genuß zu großer Quantitäten von Nahrungsmitteln, durch eine zu reizende Qualität derselben, vielleicht auch durch

*) Vgl. Frerichs „Verdauung" l. c.

zu oftmaligen Genuß; das Blut wird in Folge des zu vielen und zu oftmaligen Essens überladen mit Nahrungsbestandtheilen, wenn die Gesetze der En- und Exosmoose einer solchen Ueberladung auch eine bestimmte Gränze setzen; die Metamorphose der Blutbestandtheile wird aber dennoch nicht zu Ende geführt und ihre Retardation giebt sich kund in Abnormitäten der Se- u. Excretionsproducte (Oralurie, vermehrter Harnsäuregehalt des Urins, Albuminurie u. s. w.), sowie in Hyperämie der Se- u. Excretions-Organe, der Muskulatur u. s. w. — Es entsteht je nach der Qualität der Bluterkrankung oder der Prädisposition bestimmter Organe Rheumatismus, Catarrh der Lunge u. s. w., es entsteht insonderheit Catarrh des Magens, als eines Organs, welches unter den angegebenen Verhältnissen als locus minoris resistentiae bezeichnet werden darf. — Die Hyperämie der Magenschleimhaut und eventueller Catarrh derselben sind ferner Symptome irgendwie entstandener Bluedyskrasieen oder Nervenleiden; sie zeigen sich beim Carcinom, bei Tuberkulosen, beim Typhus, bei Bright'scher Krankheit, bei primären oder secundären Affectionen des Vagus (S. Frerichs „Verdauung" l. c. pag. 823 u. 824: „die Magenschleimhaut wird nach Durchschneidung des Vagus „hyperämisch, mit Ecchymosen bedeckt, der Magensaft nimmt „eine alkalische Beschaffenheit an und verliert seine digestiven Eigenschaften" u. s. w.) und chronischen Leiden des Nervensystems überhaupt; in der Bluedyskrasie, in der veränderten Innervation wird in den meisten Fällen die letzte Ursache des Magencatarrhs und damit der Oralurie, falls sie vorhanden ist, zu suchen sein. — Die Hyperämie der Magenschleimhaut entsteht endlich bei allen bedeutendern Hindernissen in der Blutcirculation, bei Stenose der Herzostien, Lungenemphysem, pleuritischen Exsudaten und Lungencompression, bei Leberdegenerationen u. s. w.; es entsteht Catarrh, und Oralurie kann wieder durch denselben herbeigeführt oder gefördert werden. Aber es ist bei all den fraglichen Zuständen nicht zu übersehen, daß sie selbst in einer Bluterkrankung begründet und die Ursachen zu neuen Alterationen in der Zusammensetzung des Blutes sind; in diesen Dyskrasieen des Blutes kann ebensowohl die Ursache zur Oralurie liegen, als in dem Magencatarrh; beide sind wieder nur Symptome ein und desselben Grundleidens.

Einen nächsten Platz unter den zur Oralurie führenden Lei-

den der Verdauungsorgane nehmen Krankheiten der Leber ein. Hyperämie derselben und alle daraus hervorgehenden Degenerationen: Muskatnußleber, granulirte Leber u. s. w., sind nicht selten von Oralurie begleitet. — Es kommen diese sämmtlichen Zustände entweder auf mechanische Weise durch Leiden andrer Organe zu Stande, sie sind bedingt, oder sie sind durch örtliche Reizungen, sitzende oder unmäßige Lebensweise u. s. w. primär herbeigeführt, sie sind bedingend für andre Leiden, für Circulationsstörungen, Blutdyskrasieen u. s. w. — Wir vermögen nicht zu veranschlagen, welchen der einzelnen Coefficienten des Gesammtkrankheitszustandes die Oralurie, ein Symptom des letztern, ihre Entstehung hauptsächlich verdankt. Mit Sicherheit läßt sich aber annehmen, daß das Bestehen des Leberleidens der Oralurie wenigstens förderlich ist. — Frerichs sagt mit Recht: „die blutreinigende Function der Leber bedarf einer schärfern Bearbeitung, als ihr bisher zu Theil wurde." Allein auch ohne die einzelnen Quellen der Gallenbestandtheile zu kennen, ohne die Function der Leber klar zu übersehen, man darf annehmen, daß bei einer beförderten oder unterdrückten Aussonderung der stickstoffhaltigen Chol- und Choleinsäure, der stickstofffreien Fette: Margarin, Elain und Cholestearin, so wie endlich der anorganischen, in der Galle vorkommenden Bestandtheile, Störungen der Stoffmetamorphose resultiren müssen, welche sich im erstern Falle durch eine direct oder indirect retardirte Metamorphose der stickstoffhaltigen Blutbestandtheile, im letztern dagegen mit der Zeit vielleicht durch eine pathologische Vermehrung alkalischer Basen, namentlich des Natron (??), eventuell beschleunigte Bildung von Säure im Magen u. s. w. kund geben. — Die letztere Hypothese hat nur die schwache Stütze, daß sich bei den meisten hierhergehörigen Individuen am häufigsten und intensiver, als bei andern, Säurebildung in den ersten Wegen vorfindet. Es muß dahin gestellt bleiben, welchen Theil der Schuld dabei die behinderte Leberfunction, welchen der meistens gleichzeitig vorhandene Magencatarrh, welchen ein etwaiger Ueberschuß alkalischer Basen trägt. — Ob aber Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile, ob vermehrte Säurebildung im Magen resultirt, in beiden haben wir ätiologische Momente der Oralurie kennen gelernt.

Die weitern bei der Verdauung betheiligten Organe, das

Pancreas und die Darmschleimhaut mit ihren verschiedenen Secretionsorganen, tragen zur Metamorphose der im Magen noch nicht resorbirten Nahrungsbestandtheile bei. — Die feine Vertheilung des Fettes wird nach Lenz's*), Frerichs' u. A. Untersuchungen wahrscheinlich durch eine gemeinschaftliche Einwirkung des Pancreas- und Lebersecrets befördert; ein Mangel derselben wird eine mangelhafte Resorption des Fettes, eine Anhäufung desselben im Darmkanal, pathologische Buttersäurebildung und Gasentwicklung in demselben veranlassen. Die Pancreasflüssigkeit hat nach Frerichs weiterhin den entschiedensten Einfluß auf die Metamorphose der im Magen nicht metamorphosirten Amylaceen; ein Mangel derselben wird wieder zur retardirten Metamorphose dieser, zur pathologischen Anhäufung von Milch- und Buttersäure im Darmkanal führen müssen. Dem Succus entericus liegt es ferner nach Zander's**) Untersuchungen ob, die Metamorphose der im Magen nicht metamorphosirten Albuminate zu vermitteln; Störungen seiner Secretion geben zur Retardation jener Metamorphose, zu Gasentwicklungen, zu Störungen der Nutrition u. s. w. Veranlassung. — Die Beweise für das Auftreten dieser einzelnen Erscheinungen werden in den bezeichneten Arbeiten geliefert. — Zu ganz ähnlichen Resultaten führen aber jene Zustände, in denen ein verminderter Motus peristalticus des Darmkanals ein abnorm langes Verharren der Excretionsstoffe in demselben veranlaßt. Zersetzungsprocesse, die erst außerhalb des Organismus erfolgen sollten, gehen schon im Darmkanal vor sich; Gasentwicklung, Milch- und Buttersäurebildung finden sich in der ganzen Länge des Darmtractus. Von Seiten des Nervensystems scheint insonderheit jene Verminderung der Darmbewegung bedingt zu werden. Eine Anästhesie oder Parese der vasomotorischen und die Darmmuskelcontraction vermittelnden Fasern des Sympathicus verändert die Qualität und Quantität der Secrete selbst, vermindert andrerseits die Darmbewegungen. Ob direct oder indirect entstanden, ob von den Centralorganen oder der Peripherie ausgehend, die Parese ist vorhanden und giebt sich in der bezeichneten Weise kund. — So weit stehen die Beobachtungen fest. —

*) Lenz: De adipis concoctione et absorptione. Dorpat. 1850.
**) Zander: De succo enterico. Dorpat. 1850.

Wenn die Erfahrung nun aber lehrt, daß die sämmtlichen Erscheinungen der gestörten Darmverdauung, die sich fast immer gleichzeitig mit denen der Dyspepsie vorfinden, auch fast immer von Oralurie begleitet sind, so läßt sich zur Zeit durchaus nicht bestimmen, ob die vermehrte Säurebildung im Darmkanal, ob das längere Verharren der Fäcalstoffe in demselben und eventuelle Resorption von Zersetzungsproducten, ob eine Beeinträchtigung der Bildung gefärbter Blutkörperchen (in Folge der Retardation oder qualitativen Alteration der Metamorphose der stickstoffhaltigen Nahrungsbestandtheile, der Säurebildung u. s. w.) den Hauptanlaß zur Oralurie giebt; es läßt sich, wie bei dem Magencatarrh, nicht entscheiden, welchen Antheil an der Entstehung der Oralurie das locale Darmleiden, welchen jene Allgemeinleiden des Nervensystems oder Blutes haben, deren Symptom das locale Leiden nur ist. Das Einzige, was wir mit Bestimmtheit wissen, ist das, daß die beregten Störungen der Darmverdauung, daß insondertheit träge Darmentleerung eine ebenso durchgehends vorkommende Klage der an Oralurie leidenden Individuen ist, als es oben von der Dyspepsie des Magens bemerkt wurde; darin stimmen sämmtliche Beobachter überein — Leichte drastische Aperientia, zeitweilige Gaben von Calomel u. s. w. thun solchen Patienten wohl. Eine rationelle, gegen letzte Ursachen ankämpfende Therapie wird sich aber gegen jene Allgemeinleiden des Blutes oder Nervensystems zu richten haben und durch eine sorgfältige Ueberwachung der Quantität und Qualität der Diät, durch Regulirung der äußern Lebensverhältnisse und möglichste Beseitigung psychischer Alterationen nicht nur die localen Leiden der Verdauungsorgane, sondern gleichzeitig die Oralurie, die ja beide nur Symptome des Gesammtkrankheitszustandes sind, zu heben im Stande sein.

Zu zweit kommen Krankheiten derjenigen Organe in Betracht, welche der Respiration und Circulation vorstehen. — Es bedarf keiner Auseinandersetzung, in wie fern beide auf den Ablauf der Metamorphose der Blut- und Organbestandtheile influiren. Wissen wir aber, daß der normale Zustand derselben unerläßliche Bedingung für den normalen Ablauf der Oxydationsprocesse im Organismus ist, und ist es gewiß, daß eine Behinderung dieser Processe unmittelbar zur Oralurie führen muß, so folgt, daß ein jeder Zustand der fraglichen Organe, der eine

solche Behinderung bedingt, auch als ätiologisches Moment für die Oralurie betrachtet werden muß. Demgemäß findet man denn auch Oralurie bei Emphysem der Lungen *), bei Lungenhepatisationen, bei Stenosen der Herzostien oder Insufficienzen der Klappen u. s. w. u. s. w. Allein die Coefficienten der Producte der Stoffmetamorphose liegen auf zwei Seiten; einmal auf Seiten des zu metamorphosirenden Materials, andrerseits auf Seiten der die Metamorphose bedingenden Agentien. Sind diese zu schwach für eine bestimmte Größe des Materials, so wird und muß eine Hemmung der Metamorphose resultiren; sie resultirt deshalb beim Emphysem, bei Herzfehlern u. s. w., sobald das zu metamorphosirende Material eine bestimmte, die Kraft der Agentien übersteigende Größe besitzt. Wird dagegen die Menge des Materials der verringerten Aufnahme von Sauerstoff entsprechend vermindert, so wird keine Oralurie beobachtet werden, und in dieser Weise nur ist es zu erklären, weshalb in manchen Fällen von Emphysem u. s. w. große Mengen, in manchen gar kein oxalsaurer Kalk im Urin angetroffen wird. — Ich habe bei einem 74jährigen Patienten, der an einem bedeutenden Emphysem beider Lungen litt, 4 Wochen lang vergeblich nach oxalsaurem Kalk gesucht; derselbe genoß durchschnittlich täglich nicht mehr als 6 Unzen Brod, zwei Unzen Fleisch, 8 Unzen Gemüse, 10 Unzen Bier und 20 Unzen Thee. Bei andern Emphysematikern beobachtete ich dagegen vermehrten Harnsäuregehalt des Urins und oxalsauren Kalk in Menge, und ein Blick auf die Diät-Tabelle ergab, daß sie in der genossenen Quantität nicht weit hinter gesunden Individuen zurückstanden. — Es ist deshalb immer nur relativ richtig, wenn man behauptet, Oralurie sei ein Symptom der die Blut-Circulation und Respiration beeinträchtigenden organischen Leiden. Der Therapie wird aber eben durch diese Beobachtungen ein wichtiger Anhaltspunkt dargeboten.

Mangel gefärbter Blutkörperchen, vermehrte Alkalescenz des Blutes, wurden oben als ätiologische Momente der Oralurie

*) Lehmann, Physiol. Chemie. Bd. I. pag. 49: Am constantesten kommen reichliche Abscheidungen von oxalsaurem Kalk bei entweder schon ausgebildetem Emphysem der Lungen oder der nach öfter wiederholten Catarrhen eingetretenen Verminderung der Elasticität des Lungengewebes vor.«

bezeichnet. Es darf nicht unbemerkt bleiben, daß Emphysem der Lunge und Herzleiden nach einiger Zeit stets zu jenem Mangel, Herzleiden aber auch zur Vermehrung der alkalischen Basen im Blute führen. Man hat hier mit der interessanten Beobachtung zu thun, daß sich in Folge organischer Störungen die Proportionen unorganischer Blutbestandtheile krankhaft verändern, und es ist einleuchtend, daß bei der Beurtheilung der den fraglichen Störungen eigenthümlichen, pathologisch-chemischen Erscheinungen auch diese Momente in die Waagschaale gelegt werden müssen. Die Frage, wodurch der Mangel gefärbter Blutkörperchen im concreten Fall bedingt sei, würde uns hier zu weit führen; das Factum ist hinreichend bekannt. Wenn es aber durch C. Schmidt's Untersuchungen bewiesen wurde, daß bei Hydrops und Albuminurie im Gefolge von Herzleiden u. s. w. für das ausgeschiedene Albumen ein Diffusionsäquivalent Salz in der Blutflüssigkeit zurückgehalten werde, so wollen wir es nicht unerwähnt lassen, daß sich vielleicht bei den in Frage stehenden Leiden in dieser Weise jener vermehrte Gehalt an alkalischen Basen entwickelt, welchen wir oben unter die ätiologischen Momente der Oralurie sowohl, als unter die der spontan entstehenden, chlorotischen Zustände einzureihen versuchten. Die hier nur palliative Therapie wendet sich unter diesen Verhältnissen vielleicht nicht ohne Erfolg dem Gebrauch von Mineralsäuren und später von Eisen zu. — Im Allgemeinen scheint aber dennoch bei schon länger bestehenden Herzleiden Oralurie eine seltene Erscheinung zu sein; ich habe sie in etwa 15 Fällen mehrfach gar nicht, einige Male in nur sehr unbedeutendem und nur zweimal in ausgeprägtem Grade gefunden; letzteres bei jungen Individuen, die vor kurzer Zeit einen acuten Rheumatismus überstanden und eine Stenosis ost. venos. sin. mit Insufficienz der Klappe davon getragen hatten. Unzweifelhaft liegen in der Circulationshemmung, in der Beeinträchtigung der Oxydationsprocesse, in der Verminderung des Gehaltes an gefärbten Blutkörperchen, in der sich wahrscheinlich meistens vorfindenden erhöhten Alkalescenz des Blutes förderliche Momente für die Oralurie; um jedoch Thatsachen und Theorie in Einklang zu bringen, muß man a priori vermuthen, daß den fördernden Momenten bei Herzleiden ein entgegengesetztes zur Seite steht; in der Albuminurie und in den hydropischen Ergüssen, mit denen dem Blute stickstoffhaltiges Material entzogen

wird und die Hauptquelle der Oralurie versiecht, ist vielleicht ein solches zu suchen.

Wir gelangen endlich zur Besprechung jenes Coefficienten der Stoffmetamorphosen, der bei Betrachtung pathologisch-chemischer Vorgänge nur zu oft außer Rechnung gelassen wurde und dennoch einen durchaus gleichen Einfluß auf jene ausübt, als die Aufnahme von Sauerstoff, als die materielle Beschaffenheit der Respirations- und Circulationsorgane selbst. Es ist das Nervensystem, welches mit der ihm immanenten Kraft die Functionsqualität eines jeden Organs, und in dritter Instanz den Ablauf der Stoffmetamorphosen unter allen Umständen influencirt. — Wir kennen nicht die Verbindung des Seelenlebens mit dem des Körpers; aber wir wissen, daß dasselbe, an die Gegenwart des Gehirns gebunden, primäre Depressionen und Excitationen zu erleiden vermag, und daß sich diese in Depression oder Excitation organischer Thätigkeiten kund geben. Die Depression oder Excitation theilt sich scheinbar dem Substrate des Seelenlebens mit, und ob in der Sphäre der Sensibilität oder der Motilität, ob in den Erscheinungen der Irradiation oder der Reflexbewegung, irgendwo giebt sich die Depression oder Excitation kund, irgendwo führt sie zur Retardation oder Beschleunigung des Stoffwechsels. Die freudige Bewegung, sei sie das Resultat eines rein geistigen Genusses oder das Resultat äußerer Eindrücke, ein Resultat des ungestörten körperlichen Befindens, sie bedingt die normale oder, je nach ihrer Größe, eine erhöhte Function des Organs, sie bedingt damit den normalen oder beschleunigten Ablauf der Metamorphosen des dem Organismus dargebotenen Nahrungsmateriales. Der dauernde Schmerz, der Kummer wirkt im entgegengesetzten Sinne, und ob rein geistigen, ob körperlichen Ursprungs, in Depressionen der einzelnen und der Gesammtfunction des Organismus, in Retardationen der Stoffmetamorphosen spricht er sich aus. — Aber nicht allein das Seelenleben influirt auf das Nervensystem. Abhängig von seinem Bildungsmateriale, von dem Blute, entspricht es in seinen Actionen zugleich der Qualität dieses und, je nach der Qualität und Quantität der einzelnen Blutbestandtheile selbst, resultirt bald eine Depression, bald eine Excitation jener Thätigkeit. Retardationen oder Beschleunigungen der Metamorphose eben

des Materiales, dem der Nerv die krankhafte Thätigkeit verdankt, stellen sich wieder als endliches Resultat heraus.

Nur selten wird die Gelegenheit geboten, den Einfluß geistiger Bewegungen auf den Ablauf chemischer Vorgänge im Organismus zu beobachten; er kann überhaupt nur erschlossen, aber nicht thatsächlich bewiesen werden, wenn man der exacten Untersuchung der Resultate der Stoffmetamorphose nicht anhaltenden Fleiß und fortdauernde, tägliche Aufmerksamkeit zuwendet. — Sind die letztern aber vorhanden, so wird der Zufall sicher oftmals derartige Freuden oder Schmerzen herbeiführen, daß wir ihre Wirkungen mit Bestimmtheit in den fraglichen Resultaten nachzuweisen im Stande sind. — Nur ganz vereinzelte Beobachtungen stehen mir zu Gebote; allein in vollkommnem Einklange mit aller Theorie, mit aller Erfahrung liefern sie den thatsächlichen Beweis, daß der Kummer, die psychische Depression zur Retardation der Stoffmetamorphose und damit zur Oxalurie, daß die freudige Aufregung dagegen zu dem entgegengesetzten Resultate, zur Beschleunigung der Stoffmetamorphose und nicht zur Oxalurie führt.

In Betreff des ersten Satzes, so war ein Hospital-Patient Gegenstand einer Beobachtung. — Ignaz G....., 24 Jahr alt, Schriftsetzer aus Rußland, war zunächst 1849 vom 27. Juni bis zum 13. Juli im Hospital. — Er fühlt sich seit mehren Wochen unwohl; klagt namentlich über allgemeine Schwäche, Husten und Druck im scrobiculo cordis. — Ist in frühern Jahren stets gesund gewesen; litt nie an skrophulösen Erscheinungen; beide Eltern leben und sind gesund. — Das Hautcolorit ist fast blühend; Haar und Auge schwarz; Muskulatur mäßig kräftig, Gemüthsstimmung gedrückt, Gesichtsausdruck ruhig und ernst. Organische Leiden sind nicht nachweisbar. — Objectiv sind nur die Erscheinungen eines Magencatarrhs vorhanden. Retardirte Stuhlausleerung, saurer Geschmack, Appetitlosigkeit fehlen dabei nicht. — Oftmaliges Gefühl von Zusammenschnürung des Halses und (wahrscheinlich in ähnlicher Weise veranlaßter) Hustenreiz sind zugegen. — Bei einer knappen Diät, dem Gebrauche von Mineralsäuren, leichten Purgantien und fleißiger Bewegung in freier Luft erholte sich Patient; an dem oben bezeichneten Tage wird er aus dem Hospital entlassen.

Während dieser Zeit bot der Urin des Patienten die folgenden Verhältnisse dar:

Datum.	Tempus mittendi.	Specif. Gewicht	Quantität	oxalsf. Kalk.	Erdphosphate in 1 Unze.	äuß. physikal. Beschaffenheit
					Gran	
26—27. Juni	Abends u. Morgens	1027	30 Unzen	wenig.	appr. 0,70	tiefgoldgelb.
27—28. „	d°.	1016	15 „	sehr wenig.	„ 0,40	hellgoldgelb.
30—1. Juli.	Gesammtmenge von 24 St.	1010	60 „	Spur.	„ 0,3	goldgelb.
2—3. „	d°.	1012	72 „	Spur	„ 0,35	„
4—5. „	d°.	1010	60 „	mäßig viel.	„ 0,60	tiefgoldgelb.
6—7. „	d°.	1014	30 „	d°.	„ 0,60	hellgoldgelb.
8—9. „	d°.	1008	60 „	wenig.	„ 0,50	hochgoldgelb.
10—11. „	d°.	1010	40 „	wenig.	„ 0,45	mittelgoldgelb

Am 20. September kehrte Patient ins Hospital zurück. Seit zwei Tagen war er krank. Er klagt über Zittern und Kälte in den Gliedern, Brennen in der Brust, unbedeutenden Husten, allgemeine Hinfälligkeit. Die Erscheinungen des Magencatarrhs sind stark ausgeprägt. Die Zunge, mit rothen Rändern, ist stark gelb-weißlich belegt; Stuhl stark retardirt, Geschmack pappig, oft sauer. Der Puls klein, schwach, 84. — Es lag eine einfache Dyspepsie vor; die Gemüthsstimmung war sehr gedrückt. Neigung zur Hypochondrie spricht sich in vielfacher Weise aus. — Einige Dosen Calomel mit Jalappe, dann wieder Mineralsäuren, einige Gaben Chinin werden verordnet; die Diät besteht dabei durchschnittlich täglich in 8—10 Unzen Brod, zu Anfang Mittags nur aus einer Bouillon-Suppe, später aus 4 Unzen Fleisch und etwas Gemüse; Abends wird eine Wassersuppe gereicht. — Besondere Erscheinungen treten im Verlaufe der folgenden Tage nicht auf. Die bestehenden bessern sich nur in geringem Grade. — Da Patient jedoch durchaus arbeitsfähig ist, die Unthätigkeit seiner Genesung aber nicht förderlich sein kann, wird er am 28. September wieder entlassen. — Der Speichel hatte während dieser Zeit stets eine leicht saure Reaction. Der Urin bot folgende Verhältnisse dar:

Datum.	Tempus mittendi.	Specif. Gewicht.	Quantität	oxalf. Kalk.	Erdphosphate in 1 Unze.	äuß. physikal. Beschaffenheit
					Gran	
20—21. Spt.	24 stündige Gesammtmenge.	1012,5	43 Unzen	mäßig viel	appr. 0,55	hochroth.
21—22. „	„	1014	30 „	ziemlich viel	„ 0,75	hochroth.
22—23. „	„	1019	30 „	dº.	„ 0,70	dº.
23—24. „	„	1017	40 „	wenig	„ 0,55	hochgelb.
24—25. „	„	?	40 „	dº.	„ 0,50	dº.
25—26. „	„	1022	34 „	ziemlich viel	„ 0,75	geldgelb. crystallklar.
26—27. „	„	1020	45 „	mäßig viel	„ 0,65	dº.
27—28. „	„	1018	45 „	wenig	„ 0,50	dº.

Am 4. October wurde Patient von Neuem ins Hospital gebracht. Die sämmtlichen frühern Erscheinungen waren mit erneuter Heftigkeit hervorgetreten; eine heftige Gemüthserschütterung hatte G. in einen an Tiefsinn gränzenden Zustand geworfen; seine Braut war ihm mit einem mühsam erworbenen kleinen Vermögen davongegangen. — Patient saß stumm und theilnahmslos an seinem Bette; kein Versuch der Aufheiterung gelang seinen Kameraden; der Appetit war gänzlich gewichen; vom 4.—7. October wurden kaum 12 Unzen Brod und etwas Thee genossen; vom 7.—14. täglich etwa 6—8 Unzen Brod, einige Tassen Thee und Mittags eine Tasse Bouillon. — Die gastrischcatarrhalischen Erscheinungen traten mit größter Intensität hervor. Mit Purgantien und Clystieren mußte der Stuhlgang befördert werden. Eine allgemeine Abmagerung schritt täglich sichtlich vorwärts. — Einige Unzen Wein, eine Mischung von Mineralsäuren, Chinin und Tinct. Valerian. aeth. blieben erfolglos. Patient verfiel zuletzt in vollkommne Melancholie und es blieb kein andrer Ausweg, als ihn in eine Anstalt für Geisteskranke zu schicken, wo er, am 15. October aufgenommen, ein halbes Jahr verweilte. Ich habe ihn dann wieder gesehen; heiter, lebensmuthig und bei Weitem kräftiger, als in der eben bezeichneten Periode. — Während derselben, vom 6.—14. October, bot sein Urin aber, bei gegen früher bedeutend erhöhtem Säuregehalte, die folgenden Eigenschaften dar:

Datum.	Tempus mittendi.	Specif. Gewicht.	Quantität	oxalf. Kalk.	Erdphosphate in 1 Unze	äuß. physikal. Beschaffenheit
5—6. Oct.	24 stündige Gesammtmenge.	1027	30 Unzen	überaus viel u. große Crystalle	1,10 Gran	hochgeldgelb.
6—7. „	„	1023	18 „	dº.	1,20 „	„
7—8. „	Abends u. Morgens	1022	35 „	dº.	0,90 „	tiefgeldgelb.
8—9 „	Abends	1025	15 „	dº.	1,30 „	„
13—14. „	Gesammtmenge.	1024	30 „	dº.	1,30 „	„ Sediment von harns. Salzen.

Trotz der bedeutend verringerten Einnahme von Nahrungsmaterial nahm also das specifische Gewicht des Urins relativ und absolut gegen früher zu, die Quantität verminderte sich, die Menge des oxalsauren Kalkes erreichte eine enorme Höhe, die gewöhnlichen Octaeder waren mit vielen sanduhrförmigen Crystallen untermischt, die Quantität der entleerten Erdphosphate nahm absolut beträchtlich zu; die Farbe blieb constant tiefgoldgelb; früher nicht beobachtete harnsaure Sedimente treten auf und deuten mit Wahrscheinlichkeit auf einen vermehrten Harnsäuregehalt des Urins hin. In allen diesen Erscheinungen aber und insonderheit in der eminenten Zunahme des oxalsauren Kalkes liegen schlagende Beweise für eine beträchtliche Hemmung der Stoffmetamorphose und bei dem Fehlen jeglichen andern ätiologischen Momentes für dieselbe, muß die deprimirende Gemüthserschütterung allein als solches betrachtet werden. — Die Zunahme des specifischen Gewichts entspricht nicht der Zunahme an Oxalaten; dürfen wir annähernde Schlüsse aus denselben auf den Harnstoff machen, so ist die etwaige Vermehrung desselben jedenfalls nur sehr unbeträchtlich. Ein solches Resultat, das ich bedaure nicht durch genaue Analysen belegen zu können, muß sich aber und stellt sich auch stets bei den bezeichneten Retardationen heraus. Ein Theil der stickstoffhaltigen Blutbestandtheile erreicht in seiner Metamorphose nur die Stufe der Harnsäure; wir finden diese vermehrt und den Harnstoff öfter vermindert als vermehrt. — Die aus einem Zerfallen der Harnsäure hervorgehende Oxalsäure scheint aber alsbald den Organismus zu verlassen und

nur zum sehr geringen Theile eine Oxydation zu Kohlensäure zu erfahren. Bei Retardationen und Beschleunigungen der Stoffmetamorphose tritt stets der wichtige Unterschied hervor, daß dort der Harnsäuregehalt vermehrt, der Harnstoffgehalt aber selten vermehrt, oft sogar vermindert ist, hier jedoch, wie u. A. in Folge körperlicher Anstrengung, nicht nur die Harnsäure, sondern gleichzeitig auch der Harnstoff *) in vermehrter Quantität ausgeschieden wird.

Zu ganz entgegengesetzten Resultaten führen nun freudige Gemüthseindrücke; sie beschleunigen die Metamorphose in eben der Weise, wie sie der Kummer hemmt. Ein einziges durch die Analyse erwiesenes Beispiel dafür steht mir nur zu Gebote; es ist jedoch hinreichend, um einen Begriff von dem Einfluß einer geistigen Bewegung dieser Art zu geben. Ich danke die Mittheilung desselben Hrn. Dr. Böcker in Bonn und habe es a. a. O. schon erwähnt. — Mag es aus letzterm Grunde genügen, hier nur anzuführen, daß während Dr. B. bei einer fortgesetzten täglichen Untersuchung nur höchst unbedeutende 24stündige Schwankungen seines Körpergewichts beobachtete, und täglich 70 — 75 Gramm fester Urinbestandtheile und 37 Gramm Harnstoff entleerte, diese Verhältnisse eine bedeutende Veränderung erfuhren, als eine unerwartete, sehr freudige Aufregung Statt hatte. — Der Körper verlor in 24 Stunden 1159 Gramm an Gewicht. Die Quantität fester Bestandtheile des Urins stieg auf 87,318 Gramm; die Harnstoffquantität stieg von 37 auf 40 Gramm; die Erdphosphate dagegen waren „nicht vermehrt". Es wird durch diese kurzen Angaben eine beschleunigte Metamorphose der stickstoffhaltigen Blutbestandtheile erwiesen. Ueber etwaigen Oxalsäuregehalt des Urins ist nichts bemerkt; daß derselbe jedoch nicht vermehrt war, wenn er überhaupt Statt hatte, glaube ich mit Sicherheit aus der nicht vermehrten Quantität von Erdphosphaten schließen zu können. Es ist interessant genug, und im Einklange mit allen Erfahrungen des gewöhnlichen Lebens, daß das endliche Resultat in beiden Fällen dasselbe ist. Abmagerung folgt der freudigen sowohl, als der deprimirenden Gemüthsbewegung. Allein die Art und Weise, in welcher beide zu Stande kommen, ist unendlich verschieden. — Dort findet ein absolut vermehrter

*) Cf. Lehmann, Physiol. Chem. Bd. I. 1850. pag. 169.

Stoffwechsel in Folge erhöhter Nerventhätigkeit (beschleunigte Circulation, Respiration u. s. w.) statt; hier liegt der Stoffwechsel darnieder; die Depression des Nervensystems führt zur Verringerung der organischen Thätigkeiten (Circulation, Respiration u. s. w.), diese Verringerung der organischen Thätigkeit zur Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile, die retardirte Metamorphose der stickstoffhaltigen Blutbestandtheile zur pathologischen Anhäufung von Oxalsäure im Organismus, diese zur Fortnahme einer für einen normalen Zellenbildungsproceß erforderlichen Quantität phosphorsauren Kalkes und eine Abmagerung, welche die Freude rasch herbeiführte, sehen wir endlich durch den Kummer schleichend und langsam hervortreten. Dort ist durch reichlichern Genuß von Nahrungsmitteln der Verlust sofort ersetzt, die Störung ausgeglichen; hier würde ein solcher Genuß die Abmagerung nur vermehren; die Diät muß quantitativ auf das sorgfältigste regulirt und vor Allem der Depression des Nervensystems entgegengearbeitet werden, welche die Retardation der Stoffmetamorphose veranlaßt.

Es kann nicht meine Aufgabe sein, von der Einwirkung aller einzelnen Eindrücke, die der Geist empfängt, zu reden. Sie alle wirken, je nach ihrer Qualität, in der bezeichneten Weise. Die schmerzliche Berührung deprimirt und retardirt, die freudige excitirt und beschleunigt die Stoffmetamorphose. — Hat das Sonnenlicht, hat das Grün der Felder, hat die Musik eine Besserung mancher Leiden herbeigeführt, so war es sicher die unbewußte freudige Bewegung, die sich dem Geiste mittheilte und in einer Beschleunigung der Stoffmetamorphose reflectirte, welcher der größte Theil solcher Besserung verdankt wird; entwickeln sich dagegen in dunkeln Wohnungen, in einem monotonen, wenig freudig bewegten Leben, in einer tristen Umgebung Leiden der verschiedensten Art, die gemeinsam den Charakter retardirter Stoffmetamorphose tragen, so mögen daran jene ständigen Eindrücke nicht ohne Schuld sein, und eine erfolgreiche Therapie wird nicht in etwaigen Arzneimitteln, sondern in einem Ortswechsel, in einer neuen Beschäftigung und geistigen Zerstreuung gefunden werden. — Es wurde oben eine retardirte Metamorphose der stickstoffhaltigen Blutbestandtheile als dem Wesen des tuberkulösen Krankheitsprocesses eigenthümlich bezeichnet. — Kann deshalb der Kummer, der andauernde Gram zu einer solchen Retardation

führen, so kann er damit auch ein wesentliches Moment des tuberkulösen Leidens bedingen, und den Gemüthsleiden wird mit Recht eine hervorragende Stelle in der Aetiologie der Tuberkulose angewiesen. — Die Tuberkeln werden in dieser Beziehung nur zu treffend „die nach innen geweinten Thränen" genannt; ist doch ihre Ablagerung nur das endliche Resultat einer excessiven Bluterkrankung, deren Wesen zum hauptsächlichen Theil eben in der bezeichneten Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile und deren oftерwähnten unmittelbaren Folgen zu bestehen scheint! — Und sollen wir nicht in derselben Weise jene Thatsachen der Erfahrung erklären, die uns, insonderheit bei Verwundeten, mit der Bildung von Abscessen u. s. w. in Folge psychischer deprimirender Affekte bekannt machen? Mr. Solly, Wundarzt am Thomas's-Hospital in London, sagt in seiner Lecture on purulent absorption (Lancet. 1851. March 15. pag. 289.): „I am quite sure, that mental anxiety has carried moore poor fellows to the grave, than any single cause that the surgeon has to contend with. I have repeatedly, and for a long series of years, observed a connexion between anxiety of mind and the formation of abscesses. — I have also observed purulent absorption more frequent in patients, who, to use their own phrase, have „had something on their minds"." — Ist in der Erklärung dieser Facta nicht auf eine retardirte Metamorphose der Albuminate, eine eventuelle pathologische Ausscheidung derselben mit Eiterbildung, oder auf eine retardirte Metamorphose und Ausscheidung resorbirter pathologischer Producte zu recurriren? — Ein pathologisches Quantum der Albuminate scheint ebensowohl, wie ein pathologisches Zerfallen resorbirter Eiterflüssigkeit innerhalb des Gefäßsystems (Ammoniak-Verbindungen?) bei jenen Erscheinungen in Frage zu kommen, die unter dem Ausdruck „Pyämie" zusammengefaßt werden; von genauen Blut- und Harnanalysen sind weitere Aufklärungen zu erwarten: a priori läßt sich nur vermuthen, daß Oralurie bei jener „anxiety of mind" als Symptom des Gesammtkrankheitszustandes auftritt.

Primäre, das Nervensystem deprimirende Eindrücke führen in der angegebenen Weise zur Oralurie. — Einen ganz gleichen Effect müssen jedoch jene Bluterkrankungen haben, die die Erregbarkeit des Nervensystems herabsetzen, die der Reaction, den Mitbewegungen den Charakter der Trägheit, die der Sensibilität den Charakter der Unempfindlichkeit aufprägen. Tausendfältig ist die Verschiedenheit solcher Bluterkrankungen. Je nach ihrer verschiedenen Qualität liegt unendlich oft schon in ihnen selbst ein primäres ätiologisches Moment der Oralurie; aber durch ihre Einwirkung auf das Nervensystem bedingen sie immer ein weiteres Moment derselben und das in ihnen selbst begründete Leiden wird in dieser Weise potenziirt. — Wenn man bei jenen verschiedenen Krankheitszuständen, die im gewöhnlichen Leben unter dem Namen der „Unterleibsleiden" zusammengefaßt werden, Oralurie als eine der gewöhnlichsten Erscheinungen vorfindet; wenn sich diese Zustände durch Leberhyperämie, Magencatarrh, Anschwellungen der Hämorrhoidalgefäße u. s. w. zu erkennen geben, und durch die Blutuntersuchung selbst, so wie durch einen hohen Betrag der festen Bestandtheile des Urins, Vermehrung der Harnsäure, oft auch der Schwefelsäure, mitunter selbst durch Zuckergehalt des Urins auf Störungen in der Zusammensetzung des Bildungsmateriales des Organismus hingewiesen wird; so findet sich bei eben denselben Zuständen auch meistens eine Reihe dem Nervensystem zugehöriger Erscheinungen und jenen Störungen, wie diesen Erscheinungen muß in der Aetiologie der Oralurie ein Platz eingeräumt werden. Bald klagen die Patienten über allgemeine Mattigkeit, bald spricht sich die allgemeine Depression in einer Verstimmung des Gemüths aus; bald ist eine beständige subjective Hautkälte (Einwirkung des Nervensystems auf die Production thierischer Wärme. Cf. Golding Bird, Lectures on electricity and galvanism. London 1849. Lecture III.), bald die größte Trägheit in den Functionen des Darmkanals zugegen; bald findet man einen schwachen, weichen, kleinen Puls, bald eine schlaffe Muskulatur, eine schläfrige Haltung. — Bei dem beständigen Wechselverkehr zwischen Blut und Nerv ist es oft überaus schwer zu bestimmen, wo die ursprüngliche Quelle des Gesammtkrankheitszustandes liegt, ob in dem Blute, ob in primären Erkrankungen des Nervensystems. So viel jedoch steht fest, daß eine Therapie, welche sich, wie es so häufig der Fall ist, allein bei diesen Zuständen gegen die Bluterkrankung richtet, selten zum Ziele führt. Mag man die Diät auch auf das sorgfältigste reguliren, mag Bewegung, Arbeit ꝛc. ꝛc. in genügend-

stem Maaße vorhanden sein, mögen Kaffee, Spirituosa und Alles, was man sonst als „schädlich" bezeichnet hat, gemieden werden, die Oralurie, ein Symptom der Gesammterkrankung besteht fort, und die erheblichste Ursache derselben scheint auf Seiten des Nervensystems gesucht werden zu müssen. Man wendet vielfach salinische Purgantien an; die Patienten fühlen sich schlechter darnach, als zuvor, und die Oralurie nimmt oft zu, statt ab *); man vermindert die stickstoffreiche Kost und setzt die Patienten auf vegetabilische Diät; man vermindert damit allerdings die stickstoffhaltigen Blutbestandtheile, aber es fehlt dem Nervensystem jener Stimulus, den ihm die animalische Diät giebt; man empfiehlt dem Patienten oft stundenlange Märsche, aber die Besserung erfolgt nicht; die der Anstrengung folgende Ermattung ist zu groß und die von uns augenblicklich nur berücksichtigte Erscheinung, die Oralurie, sie mindert sich nicht. Kann man dagegen dem Nervensystem dieser Individuen, neben der sorgfältigen Regulirung ihrer Diät, einen geeigneten Reiz geben, eine gesunkene Erregbarkeit wiederverschaffen, so sind die Erfolge günstiger, und die Erlaubniß des mäßigen Weingenusses, der Antrieb zu diesen oder jenen Lieblingsbeschäftigungen, die Excitation des Nervensystems durch Schauerbad, Electromagnetismus u. s. w. u. s. w. sichern dem Arzte oftmals und mit Recht einen Dank, auf den er bei allen evacuirenden oder ableitenden Mitteln, bei allen „auflösenden Extracten" u. s. w. vergeblich wartet. — Einem Patienten, der an den bezeichneten Erscheinungen in hohem Grade litt, der sich aus Sorge um seine Gesundheit in ein Privatzimmer des Hospitals einquartiert und den ich seit längerer Zeit genau beobachtet hatte, empfahl ich einmal einer Abendgesellschaft von Freunden beizuwohnen und dem Weine ad libitum zuzusprechen; der momentane Erfolg davon war günstiger, als ich ihn bei irgend einem andern Heilversuche gesehen hatte. Am 21., 22. und 23 October wurde eine gewöhnliche Hospitaldiät eingehalten; Arzneimittel wurden nicht genommen; am Abend des 23. nahm Patient an der Gesellschaft Theil und

*) Es bedürfen hier die Untersuchungsresultate von C. Schmidt der Erwähnung. Nach ihnen vermehrt sich der Gehalt des Blutes an Albuminaten bei Transsudationsprocessen der Darmschleimhaut. (Vgl. Cholera-Charakteristik pag. 54. u. 92.)

trank dabei circa 1 — 1½ Flaschen Bordeaux-Wein, und ¼ Flasche Champagner. Die Befunde der Urinuntersuchung waren dann folgende:

Vom 20—21. Oct. Abends wurden 24 Unzen Urin gelassen; specif. Gew. = 1029,2; Säuregrad = + 43. Erdphosphate: in einer Unze nahezu 0,55 Gran; oxalsaurer Kalk war in beträchtlicher Quantität, Zucker nur spurweise vorhanden. — Die Tags über genossene Quantität von Fluidis belief sich auf 38 Unzen.

Vom 21 — 22. Oct. Abends wurden 28¾ Unzen gelassen; specif. Gew. = 1025,5; Säuregrad = + 33. Erdphosphate: in einer Unze nahezu 0,50 Gran; oxalsaurer Kalk war in etwas geringerer Quantität zugegen; Zucker konnte unzweifelhaft nachgewiesen werden. Die Tags über genossene Quantität von Fluidis belief sich auf 30 Unzen.

Vom 22 — 23. Oct. Abends wurden 27¼ Unzen gelassen; specif. Gew. = 1023,7; Säuregrad = + 35. Erdphosphate: in einer Unze nahezu 0,55 Gran. — Oxalsaurer Kalk fand sich reichlicher, als am Tage zuvor. Zucker war nur spurweise aufzufinden. Die genossene Quantität von Fluidis betrug 49 Unzen (inclusive des Weins am Abend).

Am folgenden Tage traten nun folgende wesentlichen Veränderungen ein. Die vom 24. Oct. Morgens — Abends gelassene Quantität Urins belief sich auf 63 Unzen, von 1015,7 spec. Gew. Säuregrad = 15; Erdphosphate in einer Unze = 0,25. Oxalsaurer Kalk war nur in sehr geringer Quantität vorhanden; Zucker fehlte ganz. — Die Tags über genossenen Fluida beliefen sich auf 35 Unzen. — Dabei war das Allgemeinbefinden bedeutend gebessert. Patient fühlte sich leicht und heiter; ein allgemeines Wohlgefühl trat an die Stelle großer Mattigkeit und Abgeschlagenheit.

Am 25. blieben die Erscheinungen noch nahezu dieselben; es wurden 52½ Unzen von 1022,3 spec. Gewicht, + 21 Säuregrad; mit gleicher Quantität Erdphosphate und oxalsaurem Kalk, wie am 24. entleert; dabei aber 46 Unzen Fluida genossen. — Am 26. näherten sich die Verhältnisse dagegen schon wieder denen, vor dem 23.; es wurden 45 Unzen Urin entleert; dieselben hatten ein spec. Gew. von 1022,3; einen Säuregrad von + 15; die einzelne Unze enthielt 0,40 Gran Erdphos-

phate; der oxalsaure Kalk war dagegen noch nicht wieder vermehrt

Bei einer Reduction der sämmtlichen gefundenen Zahlenwerthe auf eine Durchschnittsquantität von 41,5 Unzen *) ergiebt sich für das specif. Gewicht eine bedeutende Zunahme nach dem 23. Okt. (Reducirt war das spec. Gew. am 21. = 1014,6; am 22. = 1017,6; am 23. = 1015,5; am 24. = 1023,8; am 25. = 1028,2; am 26. = 1023,9.); für den Säuregrad keine merkliche Schwankung, nur am 25. eine Zunahme (am 21. = + 25; am 22. = + 23; am 23. = + 23; am 24. = + 22,9; am 25. = + 27,4; am 26. = + 16,5); für die Erdphosphate keine Abnahme, eher eine unbedeutende Zunahme; für die im Urin entleerte Oxalsäure dagegen eine beträchtliche Abnahme. Es stellt sich demnach heraus, daß die am 23. Abends stattfindende Excitation des Nervensystems, die Quantität des entleerten Urins beträchtlich, und durchaus nicht etwa in Proportion mit dem Mehrgenuß an Fluidis, steigerte (Excitation der vasomotorischen Nerven); daß die Quantität der festen Bestandtheile des Urins zunahm; daß, der entleerten Gesammtquantität von Erdphosphaten nach zu schließen, eine absolute Verminderung der Oxalsäureproduction und eventuellen Ausscheidung von Erdphosphaten nicht statt hatte, dagegen die Oxalsäure in weit geringerer Menge als zuvor, den Organismus verließ, d. h. zum größten Theil schon in demselben ihrer normalen Metamorphose zu Kohlensäure unterlag. — Die Excitation des Nervensystems beschleunigte demnach in auffallender Weise die Stoffmetamorphose, und es geht aus derartigen Beobachtungen nur zu bestimmt die Indication hervor, bei den betreffenden Individuen jenen Haupt-Coefficienten des Stoffwechsels, jene vom Nervensystem abhängige typische Kraft des einzelnen Organs, wie des Gesammtorganismus nicht zu übersehen. — Es bezieht sich das nicht nur auf jene mit den bezeichneten „Unterleibsleiden" behafteten Individuen; es gilt in gleicher Weise von allen Patienten, deren Gesammtkrankheitszustand durch eine gehemmte Metamorphose der Blutbestandtheile ausgezeichnet ist. — Erscheinungen der Depression, wie sie sich bei der Chlorose, beim Typhus, bei der sich in Folge von schweren Gemüthsleiden entwickelnden Tuberkulose finden, sie erheischen einerseits die strengste Berücksichtigung der vorliegenden Bluterkrankung, sei sie eine auf quantitativen oder qualitativen Alterationen beruhende; aber die durch die Bluterkrankung selbst herbeigeführte Depression des Nervensystems erfordert gleiche Aufmerksamkeit, sie retardirt die Metamorphose der Blutbestandtheile und wird damit eine neue Ursache materieller Störungen. Deshalb werden reine Nervina beim Typhus, deshalb leichte Excitantia oder s. g. Roborantia (?) bei der Chlorose oder Tuberkulose stets ihren unbestreitbaren Werth behalten; die Wirksamkeit des Kohlensäuregehaltes der Mineralwässer von Ems, des Eisengehaltes der Wässer von Schwalbach, Soden u. s. w. mag in dieser Weise eine Erklärung finden.

*) Diese Durchschnittsquantität resultirte aus der Addition der an noch mehren Untersuchungstagen gelassenen Quantitäten.

Wir haben somit im Allgemeinen die Ursachen der Oxalurie kennen gelernt; wir sahen sie eines Theils auf Seiten des zu metamorphosirenden Nahrungsmaterials, andern Theils auf Seiten der die Metamorphosen bedingenden Agentien liegen. Welche von diesen Ursachen in dem einzelnen Krankheitsfalle concurriren, muß das genaue Studium desselben ergeben. Selten wohl wird sich eine Ursache als die ausschließliche und allein vorhandene nachweisen lassen. — Fassen wir aber die Resultate der vorstehenden Betrachtungen zusammen, so stellen sich in Kürze folgende Sätze heraus:

1. Die Oxalurie, eine Erscheinung, welche die verschiedenartigsten, leichtern oder schwereren Krankheitszustände begleitet, hat ihren nächsten Grund in einer gehemmten Metamorphose, in einer unzureichenden Oxydation der im Organismus producirten Oxalsäure zu Kohlensäure.
2. Die Oxalsäure hat, wenn nicht ihre ausschließliche, jedenfalls ihre durchaus vorwiegende Quelle in den stickstoffhaltigen Blut- oder Nahrungsbestandtheilen; und Alles also, was die Metamorphose dieser Bestandtheile retardirt, wird zum Erscheinen der Oxalsäure im Urin, zur Oxalurie Anlaß geben.
3. Eine solche Retardation der Metamorphose der stickstoffhaltigen Blutbestandtheile kann bedingt werden:
   a) durch absoluten Mißbrauch stickstoffhaltiger Nahrungsmittel (directe Retardation);

b) durch Mißbrauch zucker- und mehlhaltiger Nahrungsmittel (indirecte Retardation);
c) durch einen Mangel gefärbter Blutkörperchen und eventualiter verminderten Oxydationsproceß;
d) durch mangelhaften Genuß einer reinen, frischen, ventilirten Luft;
e) durch organische Leiden, die in irgend welcher Weise die Respiration oder Blutcirculation beeinträchtigen;
f) durch Zustände des Nervensystems, welche den Charakter der Depression an sich tragen, sei es daß sie in psychischen Leiden oder in Bluterkrankungen ihren letzten Grund haben.

4. Ein Ueberschuß des Blutes an alkalischen Basen spielt nach vielfachen Beobachtungen eine weitere wichtige Rolle unter den ätiologischen Momenten der Oxalurie; und es ist nicht unwahrscheinlich, daß die in Folge eines pathologischen Plus an alkalischen Basen stets beschleunigte Milch- und Buttersäurebildung im Digestionskanal irgendwie die Bildung gefärbter Blutkörperchen beeinträchtigt und damit spontan jene chlorotischen Zustände herbeiführt, die wir als oftmalige Begleiter und ursächliche Momente der Oxalurie kennen.
5. Catarrhalische Zustände der Darmschleimhaut haben, falls sie von Oxalurie begleitet sind, mit dieser meistens ein und dieselbe Quelle. — Sie können in Folge der durch sie bedingten Verdauungsstörungen die Oxalurie potenziiren; als letzte Ursachen derselben scheinen sie aber nicht in Rechnung gebracht werden zu dürfen.
6. Die Aufgaben der Therapie gestalten sich je nach den im einzelnen Falle vorliegenden Momenten verschieden. Die Hauptaufgaben werden aber immer darin bestehen, den Gehalt des Blutes an stickstoffhaltigen Verbindungen und alkalischen Basen zu verringern oder diejenigen physikalisch-chemischen Vorgänge, von denen die Metamorphose der stickstoffhaltigen Blutbestandtheile abhängt, zu bethätigen.

Längere Zeit mit ein und demselben Gegenstande beschäftigt, stets auf neue und interessante Seiten desselben hingewiesen, läuft man nur zu leicht Gefahr, den Werth des Gegenstandes selbst zu überschätzen. — Ich habe mir bei der Bearbeitung der Frage nach den ätiologischen Momenten der Oxalurie diese Gefahr nicht verborgen. Aber dennoch kann ich nicht anders, als sie als eine der wichtigsten, für Diagnose, Prognose und Therapie bedeutungsvollsten Erscheinungen bezeichnen. — Das Vorhandensein oder Nichtvorhandensein der Oxalsäure im Urin giebt uns stets einen festen Anhaltspunkt für die Beurtheilung des gehemmten oder nicht gehemmten Ablaufs der Metamorphosen der Blutbestandtheile an die Hand; es erlaubt uns, wenn auch nur einen einzelnen, dennoch einen überaus lehrreichen Blick in das Wesen des vorliegenden Krankheitszustandes. — Wenn sich aber Oxalurie in den verschiedenartigsten Krankheitszuständen und unter den verschiedenartigsten Bedingungen vorfindet, so raubt das der Erscheinung keineswegs einen Theil hoher Bedeutung; es kann nur eine Stütze für den obersten Satz der allgemeinen Pathologie abgeben, der in dem Krankheitsproceß nichts andres als eine Modification des normalen Lebensprocesses erblickt.

Die Gesetze, nach denen die Lebenserscheinungen ablaufen, ändern sich nicht; nur ihre Substrate, ihre Bedingungen; und finden sich Störungen in jenem Ablauf, so müssen die Ursachen dazu eben in diesen Substraten oder Bedingungen liegen. Bei aller Complication, bei aller Verwicklung und gegenseitigen Abhängigkeit der einzelnen jenen Ablauf bedingenden chemisch-physikalischen Vorgänge sehen wir nun aber dennoch im gesunden, wie im kranken Organismus nur eine geringe Anzahl von Endgliedern aller Metamorphosen resultiren. Trotz des verschiedenartigsten Wesens des Krankheitsprocesses selbst stellen sich die Resultate desselben in kleinern oder größern Mißverhältnissen dieser heraus, und das genaueste Studium eben dieser Verhältnisse wird damit zur dringenden Aufgabe. Findet sich dann irgend eine Erscheinung, wie die Oxalurie, als den verschiedenartigsten Krankheitsprocessen gemeinsam vor, so giebt sie zunächst allerdings den Beweis, daß diese sämmtlichen Krankheitsprocesse einen Charakterzug gemeinschaftlich an sich tragen. Aber die Pathologie, und mehr fast noch die Therapie, haben

die weitere, dringende Aufgabe nachzuforschen, aus welchen Modificationen des normalen Lebensprocesses dieses Resultat hervorging, in welchen der vielseitigen Coefficienten derselben die Ursache der uns zugänglichen Erscheinungen im einzelnen Falle liegt. Durch zunehmende Kenntniß der Anfangs- und Endglieder der Metamorphosen-Reihen, durch zunehmende Kenntniß der die Metamorphosen bedingenden Agentien wird der Schluß auf unzugängliche Mittelglieder nach und nach die Sicherheit gewinnen, die ihm zur Zeit noch fehlt, die uns aber allein den Besitz einer rationellen Pathologie und Therapie in Aussicht stellt. — Es ist dabei niemals zulässig, wie es so oft geschieht, von einer höhern oder geringern Dignität dieses oder oder jenes Organs, dieses oder jenes Blutbestandtheils zu sprechen. Jeder, auch der kleinste Blutbestandtheil, jedes, auch das geringste Organ, hat eine gleiche Dignität, wie das andre, und wenn wir die Folgen eines geringen Ueberschusses oder Mangels einer alkalischen oder erdigen Basis, wenn wir die Folgen einer geringen und selbst beträchtlichen Structur- oder Functionsveränderung eines Organs nicht gleich mit Händen greifen können, so sind wir nur berechtigt, die Grobheit unserer Hand oder die Kurzsichtigkeit unseres Auges anzuklagen, nicht aber jene pathologischen Verhältnisse, jene kleinen Anfänge zu großem Ende, als der sorgfältigsten Beachtung unwerth zu halten. — Mißverhältnisse in den unorganischen Bestandtheilen des Bildungsmateriales bilden sich immer nur langsam heran; sie entstehen bald in Folge von Mißverhältnissen in den Einnahmen, bald in Folge pathologischer Vorgänge im Organismus selbst, in Folge von Erkrankungen gewisser Organe (Herz, Leber), oder in Folge von Bluterkrankungen (Morb. Bright., Cholera etc.). In ihren kleinen Anfängen selten erkenntlich greifen sie aber auf das Tiefste in die Bildungsvorgänge ein, und bedingen damit stets Störungen der Metamorphose stickstoffhaltiger und stickstofffreier Blutbestandtheile, die sich in den mannigfachsten Erscheinungen kund geben. Dafür eben liefert die Oxalsäurebildung im Organismus wieder einen wichtigen Beweis. Ueberschuß des Blutes an alkalischen Basen, Mangel an Eisen verändern die Oxalsäure-Oxydation, vermehren die Oxalurie; einmal aber zu Stande gekommen bedingt die Anhäufung von Oxalsäure im Organismus wieder Proportionsstörungen anderer unorganischer Bestandtheile, der Erdphosphate, und in neuen Störungen der organischen Bildungsprocesse findet diese Störung wieder ihren Ausspruch.

Man hat der Oxalsäure und ihren Verhältnissen im thierischen Organismus bisher nur eine geringe Aufmerksamkeit geschenkt. Ich zweifle nicht, daß ihre hohe Bedeutung für Physiologie, Pathologie und Therapie alsbald allgemeiner anerkannt und sie damit einer sorgfältigern Beachtung theilhaftig werden wird. Von dem Interesse, welches sie vom allgemein-physiologischen Standpunkte aus einnimmt, schweige ich hier. Sehen wir sie aber im Pflanzenreiche, überall verbreitet, als ein erstes Glied in der aufsteigenden Stufenleiter der Bildungsprocesse unter dem Einflusse des Lichtes aus Kohlensäure und Wasser hervorgehen, steht sie im thierischen Organismus am Ende der absteigenden Leiter, um in der Norm denselben als Kohlensäure zu verlassen, so kann auch von dieser Seite her unser Interesse für sie nur gewinnen. — Vereinzelt werden die Thatsachen der Physiologie und Pathologie entwickelt, vereinzelt wird die Erkenntniß dieser und jener Bildungsvorgänge im Pflanzen- oder Thierreiche gewonnen; aber allmählig reiht sich die eine Thatsache der andern an; in einem wunderbaren Zusammenhange unter einander stehen die einzelnen Erscheinungen da, und die Mühe und Arbeit, welche auf die Entwicklung der Einzelheiten verwandt wurden, finden in der tiefern Einsicht in die Gesammtheit der erhabenen Schöpfungen einer unergründlichen Macht einen unersetzbaren Lohn.

Nachtrag.

Erst nach Beendigung des Drucks vorliegender Arbeit kamen mir Liebig's chemische Briefe, 1851, zur Hand. Ich finde darin eine Stelle, die für die pag. 36 und 37 beregte Frage über die spontane Entstehung von Chlorosen in Folge eines pathologischen Plus an alkalischen Basen im Organismus von hohem Interesse ist. — Liebig sagt pag. 520: „Milch- und Traubenzucker entziehen bei Gegenwart einer alkalischen Base in

gelinder Wärme selbst Metalloxyden ihren Sauerstoff." — Wird nun durch den erhöhten Alkaligehalt des Magensaftes die Metamorphose der Kohlehydrate der Art beschleunigt, daß sie sich uns durch „Säurebildung in den ersten Wegen" kund giebt, so muß, da die Amylaceen, ehe sie zur Stufe der Milch- und Buttersäure gelangen, die Stufe des Traubenzuckers durchlaufen, bei jenem erhöhten Alkaligehalt im Magen auch eine vermehrte oder beschleunigte Zuckerbildung stattfinden, und es ist sehr wohl möglich, daß in dieser Weise, in Folge der Desoxydation des Eisens, die Bildung gefärbter Blutkörperchen beeinträchtigt wird.

Druck der Universitätsbuchdruckerei v. E. A. Huth in Göttingen.

To the Editor of the Medical Times & Gazette with the Author's respects

OBSERVATIONS

WITH

HUTCHINSON'S SPIROMETER.

BY

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Reprinted from the "Transactions" of the Provincial Medical and Surgical Association.

WORCESTER:
PRINTED BY DEIGHTON AND CO., JOURNAL OFFICE.

MDCCCLI.

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HUTCHINSON'S SPIROMETER.

BY

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Amongst the inferences drawn by Dr. Hutchinson from his admirable researches with the spirometer, the one wherein he states that there does not exist "any direct relation between the circumference of the chest and the vital capacity,"—implying, by that term, the greatest quantity of breath which can be expired at one expiration after the largest possible inspiration,—is especially calculated to arrest attention. Flowing from this inference, Dr. Hutchinson deduces several other conclusions equally at variance with our preconceptions. Thus, he is of opinion that "there is no relation between the volume of the lungs and the vigour of the constitution, nor between the size of the cavity of the thorax and the amount of air which can be respired; * * * that "there is good reason to believe that in a healthy man there is no relation between the extent of mucous surface in the lungs and the amount of aëration or oxidation of the blood;" * * * that, "in fact, aëration need have no relation to the thoracic dimension; and, for the same reason, the size of the chest no relation to the vigour of the whole man. Indeed," he continues, "we incline to the contrary, viz., that it is most likely the respiration is most vigorous in the narrow-chested man, when the mobility is greatest. The vigour of the lungs, like every other organ in the body, we believe, has no

relation to the dimension. One person may have a brain one pound lighter or one-third less than another person, and yet their capacity and mental qualities shall not appear different." * * * "The *function* of the chest, as indicated by the quantity of air which we can expel, is in strict relation to the minute difference of a single inch of stature, or to ten pounds of weight. It appears probable that the function of an organ may be exactly relative to the size of the body, increasing or decreasing with it, while the organ itself bears no visible relation of volume either to its own activity or to the dimensions of the body."*

From observations upon nearly 5000 men, Dr. Hutchinson obtained the average vital capacity for every inch of stature, ranging from five feet to six feet. He found that this average followed the remarkable rule of increasing by eight cubic inches of air for every additional inch of stature from five feet to six feet. In accordance with this rule, he has furnished a table entitled, "Vital Capacity Volume (temp. 60° Fah.) Necessary to Health at the Middle Period of Life."† The quantity stated in this table will be here referred to as the standard vital capacity for the given stature.

Desirous of ascertaining how far this mean quantity, or statistical average, was practically useful in its application to individuals taken indiscriminately as we meet with them in practice, and having my curiosity excited in respect to the physiological points above stated, in 1848 I examined with the spirometer the greater number of persons who called at my house professionally, whether themselves invalids or not. As I was then residing in an agricultural part of Cheshire, the majority of my cases were agriculturists, and all were persons accustomed to live much in the open air.

It is necessary to premise that the standard vital capacity is considered by Dr. Hutchinson to be modified by temperature, weight, and age.

Temperature.—When the temperature of the room is maintained at about 60° Fah., no correction is required; but when it departs considerably from this a correction becomes necessary. The change in the bulk of the air, however, is only $\frac{1}{300}$ for every degree Fah.

* Cyclopœdia of Anatomy and Physiology, Art., "Thorax;" also, an Epitome of Dr. Hutchinson's Paper, from Medico-Chirurgical Transactions for 1846.

† Table V., Art. "Thorax;" Cyclopœdia of Anatomy, p. 1072, vol. iii.

of variation of temperature. For eight months out of the year, Dr. Hutchinson states, there needs no correction.

Weight.—Dr. Hutchinson has given a table of the mean weight for heights ranging from four feet six inches to six feet, founded upon 3000 cases.* At the height of five feet eight inches, the weight should be about eleven stone. It is expected that more extended observations will prove that the weight increases by 7lbs. for every inch of stature. When the mean weight at a given stature is exceeded by 7 per cent., the vital capacity decreases one cubic inch per pound for the next 35lbs. above this weight. Dr. Hutchinson justly remarks that the usual weight of an individual when in health may be taken to represent his mean weight,—an observation which goes far to lessen our confidence in the actual value of the calculated mean weights as guides in practice.

Age.—According to Dr. Hutchinson's table on the "Effect of Age,"† the vital capacity increases by five cubic inches during the period of from 15 to 35 years of age; diminishes by 19 cubic inches from 35 to 45; by 11 cubic inches from 45 to 55; by 13 cubic inches from 55 to 65. This conclusion is founded upon observations on 1775 healthy men, yet says Dr. Hutchinson, "although this appears by calculation, yet we do not strictly follow this ratio, as we find by experience that the effect of age may be more diminished;" and, in his own tables, Dr. Hutchinson has not "brought in the effect of time before the age of 55 years." From 55 to 65, five cubic inches per cent. are deducted from the standard quantity, and from 65 to 75, eight cubic inches per cent.

The modifying influence of temperature, and of weight and age, may be borne in mind, but in practice they are commonly neglected, and, with the exception of temperature, are, perhaps, not sufficiently ascertained to be trustworthy when applied to individuals.

In my own observations the temperature was correct, but no correction was made for either weight or age. In the few instances of children, and of persons below five and above six feet in height, I have affixed as the standard vital capacity a quantity calculated at eight cubic inches for every inch of stature, although Dr. Hutchinson restricts this rule to middle age, and to the stature ranging from five to six feet. The same standard vital capacity

* Tables X. and Y., p. 1074, l. c.

† Table CC., p. 1077, l. c.

also is applied to females as to males, which probably is not intended, as it is not mentioned by Dr. Hutchinson.

The total number of cases detailed in the tables which follow is 121. Of these, 90 were males; 31 females. Two of the cases, that of an American giant and that of the Devonshire pedestrian, have been observed recently, and are added as being interesting in themselves.

In age, 92 were from 20 to 55 years; 9 from 55 to 69 years; 17 from 10 to 20 years; 3 under 10 years.

The vital capacity was precisely the standard quantity in one case only, that of a tall shoemaker, 18 years of age, scrofulous, and having anchylosis of the right knee; was higher than the standard in 25; lower than the standard in 95.

Of those who had a vital capacity above the standard, all were males; 21 were in good health; 4 unhealthy.

Of those who had a vital capacity below the standard, 56 were in good health; 39 unhealthy.

Of the four unhealthy cases above the standard, one was a case of convalescence after pleurisy, with an excess of merely one cubic inch; another, hypochondriasis, with an excess of thirty cubic inches; the third, congestive headache, with an excess of thirty-four cubic inches; the fourth, thoracic pains two years subsequent to an attack of pneumonia of the right lung, with an excess of six cubic inches.

Of the thirty-nine unhealthy cases below the standard, one was a case of anæmia; one of dyspepsy; one of dysentery; two of pneumonia; one of chronic gastritis; one of pleurisy; fifteen of disease of the liver; five of bronchial irritation; one of peritonitis; nine of pulmonary tubercles; one of constipation; one of chronic metritis.

Of the healthy cases below the standard, 44 were males. If we take the mean of their vital capacity, we find it to be $197\frac{9}{10}$ cubic inches. This, taking Dr. Hutchinson's calculation that 230 cubic inches is the average vital capacity for all heights taken together, shows a deficiency of $32\frac{1}{10}$ cubic inches for each of the 44 individuals. Not one happened to present precisely this amount of deficiency. To take an impartial view, we add the 21 healthy males, whose vital capacity was above the standard, the healthy males, now 65 in number, give a mean vital capacity of $215\frac{7}{10}$

cubic inches; which is still below the standard, but to the smaller amount of $14\frac{7}{10}$ cubic inches. Now, this mean vital capacity, correct as regards the gross number of the sixty-five, is not correct in a single instance when applied to each individual of the gross number. This may serve to illustrate the radical distinction between an average quantity in figures, which is perfectly true for all purposes of calculation on a large scale, but absolutely untrue, or, if ever true, only so by accident, when applied to an individual case, and one of those proper facts on which medical experience is built, where the conclusion is modified by considerations too inexact to be reduced to figures, but too real to be left out of consideration.

A mean quantity drawn from a very large number must represent truly the share theoretically belonging to each; but the standard so obtained, although an important guide, may be expected in practice to be widely departed from on each side. In my own experience I found this to be the case in regard to the spirometer.

Dr. Hutchinson states, that "a man below 55 years of age, breathing 193 cubic inches instead of 230 cubic inches, unless he is excessively fat, is probably the subject of disease;" and he is of opinion that a deficiency of vital capacity to the extent of 16 cubic inches per cent. should induce a suspicion of disease. In proportion to the importance of these statements, if correct, is the desirableness of investigating their accuracy. The largest calculated deficiency given above ($32\frac{1}{10}$ in 230 cubic inches) is just above the line of 16 per cent., but many of the individuals who furnished that average presented a much greater deficiency in reality, and yet were, as far as we generally signify by the expression, in good health. Even supposing that unknown disease may have existed in a few, the result would not be greatly altered; and such a supposition is less probable than ordinary from the class of persons in question.

In an adult male, I believe that we are not in a position to decide from the vital capacity as compared with stature alone, what precise amount of deviation from the standard on the side of defect implies disorder or disease. Excess of vital capacity, of course, never indicates disease.

In 10 healthy adult females, who presented deficiencies varying from 36 to 98 cubic inches, the average deficiency, using the same standard as for males, amounted to $62\frac{9}{10}$ cubic inches.

In 14 healthy males, under 20 years of age, using the same standard, the real deficiency observed varied from 4 to 60 cubic inches, whilst the mean deficiency amounted to $32\frac{4}{10}$ cubic inches.

Dr. Hutchinson's conclusion, that size of chest, as measured by its circumference and vital capacity, are not necessarily connected, seems to be unquestionable, if size of chest be viewed apart from all other circumstances. Thus (although no proof can be needed in addition to that adduced by the discoverer), of two healthy men at six feet, one, with a chest of 41 inches, had an excess of 18 cubic inches in his vital capacity; the other, with a chest of 45 inches, a deficiency of 42 cubic inches. One giant, of 7 feet (mentioned by Dr. Hutchinson, "D"), 27 years of age, with a chest of 50 inches, had an excess of 106 cubic inches; whilst another (Table XIV., No. 13), of 7 feet 3 inches, 18 years of age, with a chest of 51 inches, had a deficiency of 12 cubic inches.*

"There is a certain rude relation," says Dr. Hutchinson, "between the thoracic dimensions and the vital capacity; if, for instance, one man has a chest 35 inches in circumference and three inches mobility, and another man has a chest 40 inches in circumference and four inches mobility, then the latter will surely displace a larger volume of air than the former, but, omitting this, we expect as large a vital capacity from a man with a thin and narrow thorax, as from a man with a broad and deep thorax." But has not size of chest a more extensive influence than this? Of two chests equal in circumference (say each 35 inches) but unequal in mobility (say one three inches, the other four,) the more mobile will have the larger vital capacity; and, therefore, for the larger chest with the larger mobility to have no more, is still to ignore size as an element, since the larger mobility by itself will account for the augmented vital capacity. We find, indeed, that

* Thomas Stanhope Goode, of Rochester, New York, aged 18, 7 feet 3 inches in height, is stated in his advertisement to be 7 feet 8 inches; was 6 feet at 13 years of age. Circumference of chest is 51 inches. Weight, 20 stone. Vital capacity, 370 cubic inches. Is straight and well-built; his limbs feel wiry, hard, and free from fat. Pulse strong, steady, 60. Heart's action strong and regular. Has always had excellent health. Does not eat more than his companion, who is a thin, bilious-looking man, of 5 feet 7 inches. Father is of middle height; mother, 5 feet 7 inches; maternal grandfather was 6 feet 9 inches; maternal uncle is 6 feet 5 inches. He is strong, fond of exercise, and before he exhibited as a show-giant he worked as a ship carpenter.

although a small chest combined with large mobility will surpass a much larger chest in vital capacity, yet it does so only when the larger chest possesses but a small mobility. Moreover, we find that in cases of excessive vital capacity, the chest is usually large, and scarcely ever of the minimum size. A glance at the following table will show that of 21 healthy males who had a vital capacity higher than the standard some had large chests; with one exception, none had small ones. The exception was a country clergyman, perfectly healthy, very active, six feet two inches in height, with an excess of 25 in his vital capacity, and a chest of 35 inches in circumference.

TABLE A.

Twenty-one Healthy Males above the Standard in Vital Capacity.

Stature		Excess over standard vital capacity	Circumference of chest	Standard circumference of chest for same stature*			Largest chests with normal vital capacity at same stature	Deficiency of these in vital capacity	Remarks
				Minimum.	Medium	Maxm.			
Ft.	In.	Cub. in.	In.	Inches	Inches	Inches	Inches	Cub. in.	
5	6	6	34	32⅞	37½	40⅝	37	4	Above minimum size.
,,	,,	6	41	...	...	...	...	...	Above maximum size.
,,	,,	56	37	...	...	...	...	...	Nearly medium size.
,,	,,	106	38	...	...	...	...	...	Above medium size.
5	7	10	34	33½	38½	41½	37	22	Above minimum size.
,,	,,	22	35	...	...	...	...	...	Ditto.
5	8	20	37	33⅞	38⅝	42⅛	39	45	Ditto.
,,	,,	10	35	...	...	...	...	...	Ditto.
5	9	4	37	34½	39½	42⅝	42	38	Ditto.
,,	,,	12	36	...	...	...	...	...	Ditto.
,,	,,	2	38	...	...	...	...	...	Ditto.
5	10	12	38	34⅝	39⅝	43½	40	46	Ditto.
,,	,,	4	37	...	...	...	...	...	Ditto.
,,	,,	4	36	...	...	...	...	...	Ditto.
,,	,,	19	39	...	...	...	...	...	Nearly medium size.
5	11	41	41	35½	40½	44	37	114	Above medium size.
,,	,,	36	42	...	...	...	...	...	Ditto.
6	0	18	41	35⅞	40⅝	44⅜	45	42	Ditto.
6	1	10	42	...	...	...	...	...	Ditto.
,,	,,	30	38	...	...	...	...	...	Above minimum size.
6	2	25	35	...	...	...	...	...	Below minimum size.

Before the completion of growth, the size of the chest appears to have a more direct relation to the vital capacity, although here still, as in the adult, the influence of stature is paramount. During

* See Dr. Hutchinson's Art. "Thorax," in Cyclopædia of Anatomy and Physiology, p. 1040.

infancy and youth the mobility of the thorax is relatively greater than in mature age, notwithstanding that in quiet respiration it is less called into request, because breathing in the child is less costal than in the adult. Now, whilst the chest is not yet consolidated, its expansion indicates more exactly the extent of the lungs than in after-life, when *cæteris paribus* the rule seems to be that large lungs have less mobility of chest than small ones. Yet, taking the standard vital capacity as 174 cubic inches for five feet of stature, children less than five feet, even if they had that standard capacity at five feet, (which they have not, as it is calculated for the adult and not for the child,) would only possess a vital capacity after the rate of $2\frac{9}{10}$ cubic inches for every inch of stature below five feet; whilst in man the vital capacity is eight cubic inches for every inch of stature above five feet up to six feet. Consequently, the thoracic mobility is relatively greater in childhood, whilst the vital capacity is relatively smaller than in manhood, in the proportion of $2\frac{9}{10}$ to eight. For example, a healthy boy, four feet three inches in height at nine years of age, having a thoracic mobility of $2\frac{1}{2}$ inches, and a chest 24 inches in circumference, may present a vital capacity of 94 cubic inches. At 27 years of age his chest may measure 36 inches, his stature be five feet eight inches, his thoracic mobility be three inches, and his vital capacity 230 cubic inches. He has gained only one-sixth in mobility, and one-fourth in stature, but one-third in circumference of chest, and one-half in vital capacity. The gain in mobility does not keep pace with the increase in height and size. The gain in vital capacity is the largest; that of circumference of chest the next. Here, then, there does seem to be a tolerably direct connection between size and vital capacity.*

* In children, the ordinary quiet breathing is abdominal; in men, chiefly abdominal, but with more movement of the lower thoracic ribs than in children; in women, chiefly costal, with more movement in the higher ribs than in men. Hence, in the movement of the thorax of quiet respiration there is a sliding scale upwards, according to age and sex. Most movement below in children, progressing upwards in men, highest in women; descending again in old age, so that in second childhood, as in the first, respiration is abdominal. In both sexes, and at all ages, the upper portion of the chest is the part whose activity is most increased by energetic voluntary respiration. Hence the different physical effect of the joyous cries of children at play, and of the dull, monotonous reading or repeating of lessons at school; of speaking with a will and speaking against the will. The deformity called *chicken-breast* is referred by Mr. Shaw and by Dr. Hutchinson to the existence of some cause of deficient ingress of air, occasioning forcible inspiratory efforts, which, not meeting with sufficient air, suck in the walls of the chest. Even enlarged tonsils, when they occasion dyspnœa, are found to be an adequate cause. Undoubtedly, when dyspnœa and chicken-breast co-exist, the cause of the one may occasion the other also, but chicken-breast is very frequently seen in delicate children under a certain age, without anything abnormal in the respiration to account for it. The deformity is most noticeable when the child is breathing quietly; when the child breathes vigorously, as in active play, the hollow on each side of the lower part of the sternum is greatly lessened, or even obliterated for the time. As the thorax becomes stronger and more developed with age, and as the lower thoracic ribs enter more into action in ordinary inspirations, the chicken-breast almost or entirely disappears. A child which was very chicken-breasted at three years of age, is often perfectly well-formed at 13. On the other hand, some without any disorder of the respiratory organs will remain chicken-breasted for life. The explanation is, that in feeble children the naturally very flexible costal cartilages, to which the diaphragm is attached, are more yielding than usual, the energy of the voluntary muscles is small, and in ordinary respiration the diaphragm is left almost alone to do the work of inspiring; when it contracts, it draws in the yielding cartilages, and causes a hollow on each side of the lower part of the sternum—chicken-breast, because the intercostal muscles do not elevate the ribs so as to antagonise the depressing influence of the diaphragm. But when the child makes its intercostal muscles act, by breathing vigorously, then the raising of the costal cartilages prevents their being drawn in by the diaphragm, and the deformity is diminished; and when the period arrives at which the ribs are habitually more raised in quiet inspiration, the deformity may vanish entirely. The indication is obviously to call into frequent play the voluntary action of the respiratory muscles, before growth is so far advanced that the costal cartilages are fixed in their abnormal shape, and until the age when ordinary inspiration naturally calls the elevating influence of the lower thoracic intercostal muscles into co-operation with the downward and inward traction of the diaphragm. The grand remedy is, not machinery, or even elaborate gymnastics, but unrestrained romping in the open air with playmates, and but little of school.

The universality of Dr. Hutchinson's great discovery, that of all elements taken singly, stature is incomparably the most important in governing the vital capacity, is well seen by comparing growing persons one with another. In Table B, 14 healthy males, under 20 years, are enumerated according to their age. So long as stature increases with age so long does the vital capacity increase, but no sooner does stature lessen than vital capacity also lessens. Thus, a boy aged 14, with a stature of five feet four inches, expires 160 cubic inches, whilst another of the same age, but seven inches lower in stature, expires only 90 cubic inches. Again, by reading the list in the order of the stature, the progressive augmentation of vital capacity is striking. There is some correspondence also between the size of the chest taken alone and the vital capacity, and none of those marked contrasts which are so noticeable in adults.

TABLE B.

Fourteen Healthy Males under Twenty Years of Age.

Age	Stature	Circumference of chest	Vital capacity	Deficiency	Sequence according to stature
Years	Ft. In.	Inches	Cubic in.	Cubic in.	
8	4 2	24	70	16	1
9	4 4	24	80	22	2
12	5 1	24	130	44	5
14	5 4	27	160	38	8
14	4 9	26	90	60	3
14	4 10	27	120	38	4
15	5 3	31	150	40	7
16	5 2	32	140	42	6
17	5 6	33	180	34	9
17	5 7	32	170	52	11
18	5 7	33	180	42	12
18	5 10	36	220	26	13
18	7 3	51	370	12	14
19	5 6	34	210	4	10

In females there appears to be a somewhat closer connection between the size of the chest and the vital capacity than in men; possibly, because their chests being smaller are proportionably more expansible. In Table C, 10 healthy adult females are enumerated in the order of the progressive increase of vital capacity, and we see that a tolerably progressive range of increase of girth of chest corresponds.

TABLE C.

Ten Healthy Females above Twenty Years of Age.

Sequence in order of deficiency	Deficiency	Standard quantity*	Circumference of chest
	Cubic inches	Cubic inches	Inches
1	86	198	25
2	98	238	27
3	72	182	26
4	72	182	27
5	64	174	29
6	62	182	31
7	50	190	26
8	48	198	30
9	41	206	37
10	36	206	31

* Calculated at eight cubic inches for each inch of stature, as directed for adult males.

As it is evident that health is consistent with a considerable and perhaps undetermined range of vital capacity, have we any right to conclude that the vital capacity is in health a correct measure of the extent to which the lungs exercise their function in ordinary respiration? We find very strong athletic men, with large chests but small mobility, who possess only a small vital capacity. Must we conclude that in such persons the lungs do not adequately perform their work? There is no proof of it. Professor Jackson, of Jefferson Medical College, contends that muscular power and not height governs the vital capacity*; but he is undoubtedly mistaken in this opinion. Muscular strength by itself does not appear to regulate at all the amount of vital capacity. In Table VII., case 9, is that of a strong muscular man, stature five feet six inches, with an excess in vital capacity of 56 cubic inches; case 10, that of a spare, compact, but not very muscular man, of the same stature, with an excess of 106 cubic inches. On the other hand, Truman, the Devonshire pedestrian, whose feats indicate his muscular vigour and power of endurance, with a stature of five feet four inches, presents a deficiency of 18 cubic inches.† Case 6, Table XI., was a man of herculean mould, very powerful, 5 feet 10 inches in height, with a deficiency of 46 cubic inches. Case 5, Table XIII., was another unusually powerful man, six feet in height, with a deficiency of 42 cubic inches. For the most part, the wide-chested men were powerful and muscular men, whatever their vital capacity.

* American Medical Examiner, 1851, p. 51.

† John Truman, on May 27th ult., the weather being dry and warm, walked 100 miles in 23½ hours. Three weeks previously, he had walked 400 miles in six days. Eight hours after completing the hundred miles in less than 24 hours, he felt free from weariness, had slight pains in the middle of each thigh, no soreness of feet; very slight swelling of feet and hands; pulse steady, firm, 80. He had slept for six hours. After a night's rest he felt perfectly fresh and as usual. He is 53 years of age, 5 feet 4 inches in height; circumference of chest, 33 inches; thoracic mobility, 3½ inches; vital capacity, 180 cubic inches; respirations 13 per minute; pulse firm, strong, steady, 72. Father died at 58. Mother is 82. Maternal grand-parents both above 80 at death. Parternal grand-parents died younger, but at what age he does not know. Is a house plasterer; has always drunk moderately, and occasionally gets intoxicated. Never feels the worse for it next morning; is always ready for work. Never had headache in his life; never was ill. Is married; has had nine children; eight are living and strong. Is a moderate eater; always has an appetite. Bowels act once a day. Never had diarrhœa, even during cholera-time. Is not a particularly good boxer, nor a good runner. His complexion is brown and sallow, hair black, eyes hazel. Never weighed ten stone.

By the vital capacity we only measure directly the quantity of breath which a person can give out, and indirectly the quantity of air which he can take in. Inasmuch as he never inspires up to his capability in ordinary breathing, this measure indicates not what a person *does* breathe, but what he *can* breathe. If then the quantity inspired were the quantity used, the vital capacity would only show what amount could by possibility be used in case of need, and not what actually was generally used. But the whole of the air inspired may not be used in ordinary respiration. If the quantity inspired did necessarily indicate the amount of activity of respiration, any person, by voluntarily deepening his inspirations, might increase the oxygenation of his blood to an almost indefinite extent. Considering that in ordinary quiet breathing we are supposed to inspire not more than from 12 to 20 cubic inches at each inspiration, and that the standard vital capacity is rated at 230 cubic inches, this arbitrary power of hyperoxidating the blood would be extreme. But we do not possess it. By general exercise we can, it is true, increase oxygenation, and for this end the inspirations are larger and more frequent, but it is because we have set in action Nature's chain of causation; we have worked the tissues faster, and the tissues have required more oxygen from the blood in consequence. The demand has evoked the supply. We may sit still and deepen our inspirations in vain, beyond the effect of the exertion required to do this, so far as adding oxygen to the blood is concerned. Merely filling the chest with air permits the breath to be held for a longer interval, but it does not impart more oxygen to the blood in a given fraction of that interval. Consequently, the man whose vital capacity exceeds his ordinary requirements by 100 cubic inches, may have all his pulmonary functions just as energetically and as completely fulfilled, so far as regards mere quantity of air, as the man with a surplus of 200 cubic inches.

The only difference would be that the latter in extraordinary circumstances will have a greater breathing power in reserve; he will last longest in a race. Individual differences in the functional capabilities of the textures of the lungs, and in the blood when circulating through them, have probably a greater influence on the activity with which ordinary respiration is performed, than has the vital capacity. An animal, after division of the *paria vaga*, provided an opening be made below the glottis, makes large and deep

inspirations, takes in much air, has a large vital capacity, yet it dies asphyxiated. Abundance of air is taken into the chest, but the lungs are unable to make use of it when there. Here the vital capacity is no measure of the activity of the respiratory function; and although there is no parallelism between this and the case of a healthy man, it will serve to illustrate the point, that a man may inspire more air than he can use. It is not assumed that he usually does so: but neither does he usually employ all his vital capacity.

There is no proof that persons with large vital capacity are in the habit of inspiring a larger quantity of air at once in ordinary respiration than those who possess a smaller vital capacity. Nor, again, that a man who possesses large lungs but only a small vital capacity, does not habitually make larger inspirations than another who possesses smaller lungs but a larger vital capacity. It must be that more air is constantly present in large lungs than in small ones, and if, as we have every reason to conclude, enough of fresh air be constantly inspired to maintain the contained breath sufficiently pure, whether the lungs be large or small, it will follow that more air is likely to be taken into large lungs than into small ones, in quiet breathing, quite irrespective of the vital capacity. The vital capacity represents a latent, and not a constantly employed power. The functional excellence of the lungs and of the blood being the same, more aëration goes on in large than in small lungs, notwithstanding that their vital capacity (in its present signification) may be less. In a word, energy of respiration depends upon the quantity of air which is used, not upon the quantity which can be inspired. The vital capacity measures only the quantity which can be taken in, not that which is used. Consequently, the vital capacity is not necessarily a criterion of the energy with which the function of respiration is habitually performed.

On this ground, then, there appears to be no reason for exempting the lungs from that law of nature which affirms that, other things being equal, size indicates power; quantity indicates force. There is nothing in nature of the figment that by lessening quantity we can increase power. A large healthy lung is a powerful breathing lung; a large healthy brain is, whether judiciously employed or otherwise, a powerful brain. So far, if not in their strange circular mode of developing an organ from a function, and a function from an organ, the phrenologists certainly seem to be correct.

But although there be no physiological ground for denying to a man with spacious healthy lungs the perfect and energetic performance of the function of respiration, merely on the score of his possessing a small vital capacity, yet such a man does lose certain advantages which a large vital capacity always confers. He is, of course, less fitted for those emergencies which demand unusual extent of respiratory movement, less capable of temporarily enlarging the superficies of his chest for the more advantageous action of his muscles during extraordinary muscular exertion, and has less available space to compensate for any encroachment on his chest by disease. Clearly, to possess a large vital capacity is a great advantage. The question is, however, how great is the disadvantage entailed by a small vital capacity? To ascertain this, we must review the conditions on which smallness of vital capacity depends, and some one or more of which its presence indicates.

Laying stature aside, these conditions in the adult of both sexes appear to be the following:—

1. Large healthy lungs with small mobility.
2. Small healthy lungs with average mobility.
3. Small healthy lungs with small mobility.
4. Lungs whose expansion is obstructed by disease either of their own tissues or of other parts, without any necessary diminution of thoracic mobility, as usually measured with the tape.
5. Disease which occasions obvious lessened mobility of the walls of the chest.
6. Morbid impatience of holding the breath sufficiently to do justice to the actual capacity of the chest.

1. The greatest bulk of lung lies behind the front part of the fourth intercostal space during expiration. A tape drawn around the chest, just below the nipples, covers this space in front. The circumference of the chest so obtained, regard being had to the fatness or spareness of the individual, indicates the size of the lungs; not accurately, for the perpendicular bulk of the lungs may differ, but with sufficient correctness. The degree in which the person can extend this tape from the completest expiration to the fullest inspiration, is the measure of the thoracic mobility. A source of fallacy to be avoided in making the observation consists in the patient separating and approximating the inferior angles of

the scapulæ out of correspondence with the expansion and contraction of the walls of the chest. The rule of health, according to Drs. Hutchinson and Sibson, is, that a person of ordinary stature and weight, and of middle age, should be able to extend the tape three inches. It may be that this is the best standard we can have, but in every healthy man whose chest was large and vital capacity small, I found the thoracic mobility less than this. When the mobility amounts to three inches, and the chest is large, the person healthy and of adult age, the vital capacity is always good. On the other hand, the chest may be large, the mobility less than three inches, the vital capacity small, and yet the person may be quite healthy. We must, therefore, check the vital capacity by the mobility. Large lungs with full thoracic mobility, but small vital capacity, show that something is abnormal. But does not the vital capacity test the thoracic mobility as well as the capability of inspiring? It does so positively, but not negatively. A large vital capacity necessarily indicates a large mobility; but a small vital capacity does not necessarily imply a small mobility; and it is the small vital capacity alone that is the subject of protracted inquiry. The giant, seven feet three inches in height, with a chest of 51 inches, and a thoracic mobility of six inches, possessed a vital capacity of 370; whilst the other giant, three inches lower in stature, having a thoracic mobility less by two inches, and a chest one inch less in circumference, presented a vital capacity of 464. The former was only 18 years of age, the latter 27. The largest vital capacity was possessed by the man with the smallest thoracic mobility. Whenever, then, an apparently healthy individual, with a large chest, presents but a small vital capacity, we must ascertain, before we attach significance to this, whether or not he has a full amount of mobility. If he has, the defective vital capacity is suspicious; if he has not, the next question is, whether or not the small mobility be normal. If so, the small vital capacity in itself does not indicate anything wrong; it merely indicates a smaller mobility than usual.

In healthy children the mobility is always large and the vital capacity small.

2. In females, as in males, before the completion of growth of the viscera, there may be three inches of thoracic mobility and even more, and yet deficient vital capacity, owing to the lungs

being small, though healthy. In a gentleman about to insure his life, examined whilst writing this, I find, with a stature of five feet six inches, and a thoracic mobility of 3½ inches, a deficiency of 14 cubic inches in his vital capacity. His health appears to be perfect, but his lungs are small, the circumference of his chest being only 33½ inches.

3. If large lungs with small mobility may present but a small vital capacity, small lungs with small mobility necessarily cannot present any other. It appears, however, to be more common for large-chested than for small-chested men to possess small thoracic mobility. The latter can less spare the power of obtaining extra room for breathing purposes. Here also the practical question recurs, is the small mobility normal?

4. It is the rule that any disease within the chest which mechanically opposes the performance of complete respiration, prevents the full movement of the walls of the chest. But chronic pneumonia may exist and lessen the vital capacity without diminishing the expansion of the walls of the chest, as ordinarily measured by the tape. Here we may have small vital capacity conjoined with large chest and large mobility. Indeed, a smaller amount of deficiency is suspicious when the chest is ample and the mobility large—conditions which in health would insure a large vital capacity—than when the thoracic mobility is normally small.

5. Disease which obviously lessens the thoracic mobility will necessarily lessen the vital capacity. Phthisis furnishes an example the commonest and best. But the significance of a given amount of deficiency should be estimated by comparing it with the capacity which the patient once possessed, rather than with any fixed standard. For instance, if a man of five feet eight inches in stature, could once expire 270 cubic inches, and can now expire only 190, the alteration is full of moment; but if his vital capacity never was greater than now, he may be in perfect health, and yet have a vital capacity not exceeding 190 cubic inches. The important point, then, to be ascertained is rather,—has the vital capacity undergone a change on the side of deficiency, than,—is the vital capacity smaller than a given standard? As this can rarely be known from prior examination with the spirometer during health, it is to be inferred from the mode and degree in which the walls of the chest now move in respiration. Ample directions for this

purpose are given in the works of Stokes, Williams, Walshe, and others, but I cannot avoid referring to the ingenious chest-measurer of Dr. Sibson, by which we learn all that the tape can demonstrate, and distinguish abnormities of movement too local for detection by that ruder means of examination. In all cases of disease whatever in which the spirometer is useful, in my opinion, the chest-measurer is ancillary to it.

6. I believe that there are some cases in which the vital capacity, as shown by the spirometer, indicates neither the amount of thoracic mobility nor yet the quantity of air which the lungs can hold;—cases in which the patient is unable to retain his breath so as to "slowly make the deepest expiration," which Dr. Hutchinson recommends. The morbid sensibility of the air-passages in influenza exemplifies this. To a smaller extent, the same fact is manifested by the healthiest lungs when the expiratory effort to fill the spirometer is made either much too quickly or much too slowly. I have particularly noted this in case 3, Table XII., because this man possessed an excess of 41 cubic inches over the standard vital capacity. When he expired with moderate and equal effort, after the deepest inspiration, he reached 295 cubic inches, but when he expired either very quickly or very slowly, he could not reach this amount by 15 cubic inches. The same holds good with all. I select the above as being, from his large vital capacity, an unexceptionable instance. The explanation may be, that when a large expiration is performed with undue quickness, the bronchial muscular tubes do not contract with their normal regularity and completeness, so as to expel all the breathing air, and a larger quantity of residual air is left. Conversely, if expiration be too prolonged, the bronchial muscles may lose somewhat of their contractile force, and the same result ensues. Of course the walls of the chest are not drawn in to the same extent as in the more perfect expiration, since we cannot by volition, under any circumstances, prevent the thoracic walls from accommodating themselves to the state of the lungs. In addition to this, the impression which dictates an inspiration may become irresistible, when expiration is too slow, before the lungs have given out quite all the air which, by a more perfect performance of the expiratory act, they could expel.

I have frequently found a smaller vital capacity than ordinary in

cases of marked anæmia, without any apparent thoracic disease to account for the deficiency. Here there is impatience of holding the breath, owing to the morbid condition of the blood. Desire for breath is always a sign that the blood requires oxygen. Anæmic blood is badly oxygenated blood. It is, consequently, less able to bear a momentary interruption to the supply of air. Hence one of several reasons for the breathlessness of anæmic subjects, and one reason for the temporary diminution in their vital capacity.

In all cases of subacute and chronic disease of the liver attended with enlargement, I have found the vital capacity diminished. This result may obviously depend upon mechanical obstruction to the descent of the diaphragm, arising from enlargement and tenderness of the liver. But it has often appeared to be more than commensurate with the degree of hepatic enlargement, so far as this could be ascertained. Breathlessness on exertion is so common an accompaniment of deficient excretion of bile, that cholæmia may not improbably act like simple anæmia in rendering the lungs impatient of holding the breath. Indeed, cholæmia usually is anæmia *plus* the excrementitious elements of bile.

In conclusion, I must observe that no one can be more sensible than myself of the vast disparity in numbers between my observations and those of Dr. Hutchinson, nor more disposed to rely implicitly on the accuracy of his. If I have ventured to question the applicability of some of his inferences as strict guides in practice, it is because my observations, however insufficient as a foundation for new general principles, are numerous enough to test those already laid down; and because I cannot think that inferences which merely express the mean of a number of observations can ever prove trustworthy as standards for individuals. It is the province of statistics to build up individual matters of chance into gross matters of certainty. However certain the calculation on a large scale may prove, no dealing with the individual elements can by possibility render one of them less a matter of chance than before. An evil is always corrected by a counter evil. Overdrugging leads to homœopathy; neglect of the use of water to hydropathy; intemperance to tectotalism. The pendulum always at first swings too far in the opposite direction. It may be that the abuse of numbers is *the* evil necessary for correcting carelessness in observing and laxity in thinking; but an evil it undoubtedly

is to divert the average of numbers from calculations which concern numbers, and to apply it to an individual as an individual. The average concerns him only as a constituent unit of the mass of figures, whose excess covers some other unit's deficiency, or whose deficiency is covered by some other's excess. In him as a constituent of the mass, the excess or the deficiency is alike unimportant; to him as an individual, the excess or the deficiency is all-important.

Still there is no other mode of obtaining a standard for reference but that which Dr. Hutchinson has adopted, nor, regard being had to the classes which furnished the subjects of his observations, can any better standard for adult males be found. But, taking the standard given, I should rate its practical importance lower, and allow a much wider departure from it to be consistent with health, than Dr. Hutchinson does.

The following are stated as provisional propositions to be confirmed or refuted by further inquiry:—

1. The close connection between the vital capacity and the stature, which constitutes Dr. Hutchinson's greatest and very important discovery, throws into the shade its other relations, yet size of chest, as measured by its circumference, does appear to exercise an influence, when not nullified by co-existing with a small thoracic mobility.

2. Narrow chests more constantly possess a full mobility than broad ones, both being healthy.

3. Consequently, both size and mobility are to be taken into account in estimating the significance of the vital capacity; for, if a large chest with a large mobility present even a moderate vital capacity, this may virtually imply greater defect than a smaller vital capacity in a chest of the same size, but which normally possesses a smaller mobility.

4. The only *exact* standard for any individual being his own normal vital capacity when in health, and this being rarely ascertainable, we must be guided by what we can learn of departure from it, by examining the chest and its amount and kind of mobility, in connection with the vital capacity, rather than by the actual degree of deviation of the vital capacity from the standard quantity laid down. Thus, a given deficiency taken in conjunction with any other ground of suspicion adds force to the latter; but

in the entire absence of such, taken alone, it does not necessarily indicate anything abnormal.

5. The limit of deficiency from the standard quantity laid down, which is consistent with health, cannot be stated with precision. It is probably a variable quantity, special in every individual. I apprehend that the limitation to 16 per cent. will be found too low in adult males, and considerably too low in females.

6. Dr. Hutchinson may not intend his standard scale of vital capacity to be applied to females, or to males before the completion of visceral growth. For both these classes the standard is too high.

7. When a deficient vital capacity co-exists with any cause of impatience of holding the breath, or with hurried breathing on slight exertion, which is not on other grounds referable to the lungs, it does not by itself necessarily indicate disease of the lungs. This, which is but a truism in the case of ovarian dropsy and such like causes of dyspnœa, also applies to cases of anæmia and of chronic disease of the liver, in which a small vital capacity need not by itself induce suspicion of tubercle of the lungs.

8. When the evidence furnished by the spirometer is distinctly favourable, it is valuable, trustworthy when taken singly, and needs no qualification. But, when the evidence appears to be unfavourable, although still valuable, it is not trustworthy when taken singly, but requires to be carefully checked by every other means of diagnosis, more especially by the use of the chest-measurer, before we can justly deduce from it the existence of any deviation from health.

TABLE I.—*Stature*, 5 *Feet*. *Standard Quantity*, 174 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	J. M., female	Farmer	58	29	110		64	Healthy.
2	C. T., female	Lady	16	26	100		74	Anæmia. No pulmonary disease.
3	E. S., female	Milliner	30	25	100		74	Recovering from hepatitis.

TABLE II.—*Stature*, 5 *Feet* 1 *Inch*. *Standard Quantity*, 174 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	C. C., female	Schoolmistress	69	32	80		94	Congested liver; habitual constipation.
2	M.W., female	Farmer	63	34	100		74	Chronic hepatitis.
3	S. S., female	Farmer	39	24	80		94	Hepatitis.
4	S. W., male	Gentleman	12	24	130		44	Healthy.

TABLE III.—*Stature*, 5 *Feet* 2 *Inches*. *Standard Quantity*, 182 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	S. P., female	Innkeeper	20	26	110		72	Healthy.
2	S. B., female	Butcher	23	27	110		72	Healthy.
3	M. B., female	Farmer	20	31	120		62	Healthy.
4	T. B., male	Farmer	16	32	140		42	Healthy.
5	M.W., female	Lady	15	25	70		112	Bronchitis.
6	S. L., female	Farmer	20	33	120		62	Recovering from peritonitis.
7	J. C., male	Farmer	29	33	140		42	Healthy.

TABLE IV.—*Stature*, 5 *Feet* 3 *Inches*. *Standard Quantity*, 190 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	W. B., male	Farmer	15	31	150		40	Healthy.
2	S. P., female	Farmer	32	30	110		80	Bronchitis.
3	R. B., female	Miller	27	29	140		50	Latent tubercles.
4	S. J., female	Farmer	31	28	140		50	Congested liver.
5	W. J., female	Farmer	31	26	140		50	Healthy.

TABLE V.—*Stature*, 5 *Feet* 4 *Inches*. *Standard Quantity*, 198 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	J. C., male	Farmer	27	36	190		8	Healthy.
2	S. C., female	Lady	21	25	112		86	Healthy.
3	M. S., female	Schoolmistress	36	28	130		68	Phthisis. 2nd stage.
4	E. M., female	Cooper	46	30	150		48	Healthy.
5	W. W., male	Gentleman	14	27	160		38	Healthy.
6	J. T., male	House plasterer	53	33	180		18	Healthy. The Devonshire pedestrian.

TABLE VI.—*Stature*, 5 *Feet* 5 *Inches*. *Standard Quantity*, 206 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	H. A., female	Tailor's wife	43	30	80		126	Phthisis, 2nd stage, of eight years' duration; still living.
2	A. N., female	Farmer	40	37	165		41	Healthy; sanguineous
3	A. B., female	Lady	32	30	130		76	Chronic metritis; bilio-sanguineous.
4	F. S., female	Lady	33	28	150		56	Constipation; bilio-lymphatic.
5	E. F., female	Lady's maid	29	31	170		36	Healthy; bilious.
6	E. W., female	Farmer	19	26	80		126	Phthisis, 1st stage.

TABLE VII.—*Stature*, 5 *Feet* 6 *Inches*. *Standard Quantity*, 214 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	G. S., male	Farmer	45	36	200		14	Chronic hepatitis.
2	J. B., male	Farmer	19	34	210		4	Healthy.
3	J. T., male	Shoemaker	43	37	210		4	Healthy.
4	W. S., male	Farmer	29	34	220	6		Healthy.
5	J. G., male	Shoemaker	43	32	200		14	Healthy.
6	E. G., male	Gentleman	26	41	220	6		Healthy; sanguineous, stout and fat
7	W. B., male	Farmer	21	35	174		40	Phthisis, 1st stage.
8	J. R., male	Gentleman	17	33	180		34	Healthy.
9	J. H., male	Blacksmith	27	37	270	56		Healthy.
10	J. B., male	Farmer	32	38	320	106		Healthy; spare habit; is a good runner; father a famed boxer; his grandmother died at the age of 110.

TABLE VIII.—*Stature*, 5 *Feet* 7 *Inches*. *Standard Quantity*, 222 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	J. C., male	Miller	20	34	223	1		A month after an acute attack of pleurisy.
2	J. C., male	Farmer	28	34	232	10		Healthy.
3	J. H., male	Farmer	18	33	180		42	Healthy.
4	T. H., male	Farmer	67	34	180		42	Enlarged and indurated liver; tender epigastrium; spare habit.
5	J. S., male	Farmer	60	40	150		72	Very corpulent; congested liver.
6	T. J., male	Sailor	36	36	220		2	Dysentery.
7	W. B., male	Miller	25	35	244	22		Healthy.
8	W. C., male	Gardener	60	39	100		122	Bronchial catarrh (influenza)
9	S. B., male	Butcher	21	34	180		42	Bronchial catarrh (influenza)
10	P. K., male	Farmer	17	32	170		52	Healthy.
11	R. T., male	Farmer	26	37	200		22	Healthy.
12	S. B., male	Butcher	26	37	120		102	Recovering from pneumonia. A week afterwards, 180 cubic inches.
13	J. C., male	Farmer	61	39	250		28	Chronic gastritis.
14	S. P., female	Farmer	25	28	54		168	Pneumonia.

TABLE IX.—*Stature*, 5 *Feet* 8 *Inches*. *Standard Quantity*, 230 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	J. C., male	Farmer	31	37	250	20		Healthy.
2	G. R., male	Farmer	59	36	180		50	Healthy.
3	J. A., male	Tailor	47	36	194		36	Healthy.
4	S. W., female	Lady	36	26	100		130	Latent tubercles in both lungs.
5	J. A., male	Farmer	49	38	200		30	Died a year afterwards, from hydatids in liver.
6	C. O., male	Farmer	50	39	260	30		Hypochondriasis. No local disease ascertainable.
7	G. S., male	Bookbinder	30	35	240	10		Healthy.
8	J. P., male	Farmer	39	39	185		45	Healthy.

TABLE X.—*Stature*, 5 *Feet* 9 *Inches*. *Standard Quantity*, 238 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	F. H., male	Miller	23	34	200		38	Healthy; spare habit
2	R. J., male	Farmer	38	37	200		38	Recovery from jaundice from congested liver.
3	P. R., male	Farmer	56	38	180		58	Healthy.
4	W. L., male	Farmer	65	37	210		28	Healthy.
5	J. W., male	Farmer	64	37	242	4		Healthy.
6	S. B., male	Farmer	43	38	230		8	Healthy.
7	H. L., male	Gardener	30	36	250	12		Healthy.
8	J.P.W., male	Gentleman	44	37	200		38	Healthy; stout, full, sanguineous habit.
9	J. C., male	Farmer	28	37	200		38	Bronchial catarrh (influenza)
10	W. S., male	Butcher	40	35	160		78	Congested liver
11	S. D., male	Mason	27	38	240	2		Healthy.
12	S. B., female	Farmer	40	27	140		98	Healthy; spare habit
13	W. H., male	Farmer	51	42	200		38	Healthy.
14	W. L., male	Cheesefactor	45	34	166		72	Phthisis, 1st stage; died two years afterwards.
15	J. T., male	Carpenter	49	41	230		8	Healthy.
16	J. J., male	Station-Master	30	37	200		38	Congested liver.

TABLE XI.—*Stature*, 5 *Feet* 10 *Inches*. *Standard Quantity*, 246 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	D. A., male	Farmer	18	36	220	34	26	Healthy.
2	J. G., male	Farmer	21	38	280			Congestive headache. Sanguineous. Very stout and fat.
3	J. B., male	Grocer	24	35	200		46	Strumous constitution, but in health.
4	J. M., male	Farmer	40	37	140		106	Hepatic congestion and enlargement.
5	J. L., male	Farmer	30	38	260	12		Healthy.
6	G. A., male	Gamekeeper	50	40	200		46	Very athletic; had been noted as a pugilist.
7	A. J., female	Milliner	25	29	60		186	Phthisis, 1st stage; since dead.
8	J. D., male	Farmer	32	37	250	4		Healthy.
9	J. M., male	Farmer	24	36	250			Healthy.
10	W. D., male	Farmer	23	40	230	19	16	Healthy.
11	I. J., male	Farmer	29	39	265			Is in health. Has lost three brothers and two sisters from rapid phthisis.
12	J. F., male	Actuary	32	40	220		26	Healthy.
13	E. H., male	Farmer	25	35	200		46	Healthy.

TABLE XII.—*Stature*, 5 *Feet* 11 *Inches*. *Standard Quantity*, 254 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	S. P., male	Farmer	42	37½	150		104	Recovery from pleurisy
2	T. L., male	Farmer	47	48	190		64	Very stout and fat. Dyspeptic.
3	I. N., male	Farmer and blacksmith	41	41	295	41		Athletic. If he expired either very quickly or very slowly, only 280 cubic inches.
4	T. J., male	Farmer	47	37	140		114	In health.
5	I. B., male	Farmer	24	36	200		54	Healthy.
6	T. B., male	Farmer	48	42	290	36		Healthy; athletic.
7	J. S., male	Gentleman	45	44	235		19	Corpulent; congested liver; lithic gravel.

TABLE XIII.—*Stature*, 6 *Feet*. *Standard Quantity*, 262 *Cubic Inches*.

No.	Initials and sex	Rank	Age	Circumference of chest	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Cubic inches			
1	J. P., male	Farmer	48	39	250		12	Healthy; spare habit
2	J. A., male	Farmer	36	39	260		2	Healthy.
3	P. J., male	Farmer	41	41	244		18	Healthy.
4	C. P., male	Publican	38	37	240		22	Healthy.
5	J. S., male	Policeman	32	45	220		42	Healthy; stout, athletic, and fat; biliolymphatic.
6	J. O., male	Farmer	25	37	240		22	Healthy.
7	W. L., male	Farmer	53	39	224		38	Healthy.
8	W. P., male	Shoemaker	18	36	262			Scrofulous. Anchylosis of right knee.
9	S. S., male	Farmer	45	41	280	18		Healthy.

TABLE XIV.—*Stature, below 5 Feet and above 6 Feet.*

No.	Initials and sex	Rank	Age	Circumference of chest	Stature	Standard quantity	Vital capacity	More than standard	Less than standard	Remarks
				Inches	Ft. In.	Cubic inches				
1	M. R., female	Farmer	12	22	4 1	78	75		3	Healthy.
2	E. J., female	Farmer	36	31	4 11	166	100		66	Chronic hepatitis.
3	R. G., male	Farmer	14	26	4 9	150	90		60	Healthy.
4	J. G., male	Farmer	14	27	4 10	158	120		38	Healthy.
5	A. A., female	Tailor	10	24	4 6	126	80		46	Healthy.
6	W. B., male	Farmer	8	24	4 2	86	70		16	Healthy.
7	D. M., male	Farmer	9	24	4 4	102	80		22	Healthy.
8	W. N., male	Farmer	35	40	6 6	310	280		30	Healthy.
9	J. B., male	Farmer	31	42	6 1	270	280	10		Healthy; sanguineous.
10	E. W., male	Merchant	33	37½	6 1	270	276	6		Thoracic pains; two years before, pneumonia of right lung.
11	G. B., male	Farmer	32	38	6 1	270	300	30		Healthy; sanguineous.
12	W. D., male	Clergyman	34	35	6 2	270	295	25		Healthy; an excellent walker; sanguineous.
13	T. S. G., male	Show giant	18	51	7 3	382	370		12	Healthy, muscular, not fat; chest expands by tape measure 6 inches.



From his Grateful &
The Author.

HISTORY AND DESCRIPTION

OF AN

OSSIFYING ENCHONDROMA

CONNECTED WITH THE

TESTICLE OF A HORSE.

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ILLUSTRATED WITH A COLOURED PLATE.

LONDON:
PRINTED BY COMPTON AND RITCHIE, MIDDLE STREET, CLOTH FAIR.
1850.

HISTORY AND DESCRIPTION

OF AN

OSSIFYING ENCHONDROMA

CONNECTED WITH THE

TESTICLE OF A HORSE.

The diseased specimen which is the subject of the present remarks was removed from a horse in the operation of castration, and forwarded on the 28th May, 1850, to the Editors of The Veterinary Record, by Mr. Robert Cook, veterinary surgeon at Erith. He stated in an accompanying note, that "the testicle was taken from a four-year-old horse, which appeared to suffer no inconvenience from its presence, although he had always been in an emaciated condition. The scrotum on the side whence it was taken had the appearance of hernia, the gland being much more pendulous and pouched than its fellow, which was quite free from disease, and very large. On making an incision into the diseased testicle, nearly an ounce of dirty-looking serous fluid escaped."

The testicle (see *coloured plate, fig.* 1) measures six inches in its long axis, and three inches from above downwards. It is soft and flabby, except at the posterior part, where a moderately hard tumour, A, about the size of a small orange, is connected with it. The gland is invested by the reflected or visceral portion of the tunica vaginalis, T, whose attachment, by a considerable amount of areolar tissue, is much looser than in the healthy condition, as it could readily be stripped off; moreover, the membrane is abnormally thick and opaque. Its general colour is a dead white, but pinkish at the supero-anterior part, near the commencement of the cord. The anterior extremity of the epididymis and the cord appear to be of normal size, and to preserve their usual relative position. The intermediate connecting portion of the vaginal tunic has been

torn asunder, and the extremity of the cord has been seared by the hot iron in the operation of castration. The spermatic artery measures $\frac{3}{16}$ of an inch in circumference; in its interior is a small quantity of coagulated blood, and in the structure of its coats no deviation from the natural condition can be detected by the aid of the microscope. The other constituents of the cord are apparently healthy. On dividing the testicle longitudinally, a hollow capable of containing a large pigeon's egg is exposed. The normal structure of the gland is nowhere presented to the naked eye; but from before backwards the tissue, indistinct in character and soft, gradually loses its red colour, becomes less in amount, and eventually degenerates into a dense white fibrous tissue, B, intimately connected with the diseased mass, A, which measures $1\frac{3}{4}$ inch from above downwards, $2\frac{1}{2}$ inches from before backwards, and, when cut longitudinally in the middle, $3\frac{1}{8}$ inches in the maximum transverse axis. The interior of the tumour being thus exposed, it presents two halves, of which the right is crescentic in shape, and measures $2\frac{1}{2}$ inches from before backwards, and $1\frac{1}{8}$ inch in breadth at the widest point; $\frac{6}{8}$ of an inch from its anterior extremity it is marked by a transverse groove. The left half is divided by one very deep and one shallow groove into three parts, of which the anterior and outer one is triangular, with its base turned forwards, united by a narrow and short slip of tissue, like the rest of the tumour, to the left half. The posterior portion is irregularly circular, and measures $1\frac{3}{8}$ inch transversely, and $1\frac{6}{8}$ inch from before backwards; between these two divisions of the left half is a third, much smaller, its longest axis not exceeding $\frac{7}{8}$ of an inch

The cut surface of the tumour has a pale yellow colour, and is marked by numerous shallow grooves which irregularly intersect each other, so as to divide the mass into irregularly-shaped lobules. It presents additionally three cysts, C, i. e., closed membranous sacs, each about the size of a large bean; two of them are situated on the left half and one on the right. They were distended with a transparent reddish fluid, the albuminous nature of which is proved by its conversion into a coagulated mass, after preservation of the specimen in a saline solution containing a small quantity of corrosive sublimate.

The physical appearance of the surface is neither that of genuine

adipose nor fibrous tissue, but is what would, *à priori*, be anticipated from an intersection of a mass of fat by bundles of fibres. The consistence of the tumour, in the greater part of its extent, is that of ordinary muscle; but in the centre of the exposed surface are two narrow crescents, *b b* (with their convexities in mutual contact), similar to cartilage in consistence, and in their pale bluish-white or greyish colour. This cartilaginous material may be seen and felt extending laterally into each half of the tumour, imbedded in the surrounding yellow adipo-fibrous tissue, to which it is so closely adherent as not to be detached by the aid of the forceps. By grasping with two fingers the right or smaller half of the tumour, an osseous mass is felt in its interior, and more particularly towards the outer surface, near the tunica vaginalis, from which it is only separated by a small quantity of fibrous tissue: this being dissected off, a dirty white surface is exposed; which is of bony hardness in a considerable part of its extent, but at one part yields to the knife. In this situation, a small piece of the apparently imperfect bone being removed, a cavity capable of containing a large filbert is exposed; its boundary is constituted by a thin layer of bone resting on cartilage, and lined by a closely adherent white fibrous membrane.

By microscopic examination, the tissues entering into the formation of the tumour are ascertained to be, firstly, fat and fibrous tissue (white and yellow); secondly, cartilage; thirdly, bone.

The yellowish tissue visible on the surface consists of fat cells (in no way differing from those of healthy adipose tissue) and fibres, which take a wavy and in some parts very irregular course between the fat cells. Some of the fibres are extremely fine and delicate, and may occasionally be seen placed side by side in considerable number to form fasciculi, while others present a well-defined outline, and are wider. The addition of acetic acid confirms the inference deduced from observation of the anatomical characters, *that the fibres are of two kinds*, white and yellow; for, while the majority of them are rendered indistinct by the chemical re-agent, a few remain intact, are very tortuous, and may in some instances be seen to divide, and to terminate abruptly.

Imbedded in the fibrous tissue above described, and very closely adherent to it, is cartilage. A thin perpendicular section of this texture, with some of the adjoining fibrous tissue (see *Fig.* 2)

being placed under the microscope, exhibits the true organization of cartilage; "nucleated cells disseminated in a solid mass or matrix." Those cells which are deeply seated, i. e., at a distance from the free surface, are irregularly oval and spherical, and their outline is indistinct. The nuclei are for the most part coarsely granular, and have a well-defined margin; their extreme measurements in the transverse direction are $\frac{1}{1000}$th to $\frac{1}{700}$th of an inch. The remark made by Dr. Sharpey* may be here with justice repeated, "that it is often difficult to say whether a body contained within a cartilage cell is its nucleus, or merely the granular contents which have shrunk away from its sides and formed a mass of the same shape as the cell itself, in which the true nucleus is concealed." In some parts an eccentric nucleolus (from $\frac{1}{4000}$ to $\frac{1}{3000}$ of an inch in diameter) is seen within the nucleus; its outline is well defined, and the interior bright, bearing a resemblance to a particle of fat. It may be well to observe that those nuclei which possess nucleoli are less granular than others (see *fig.* 3). On approaching the surface—which is in contact with the fibrous tissue—the cell walls are scarcely visible, and some of the nuclei present an elliptical, others a fusiform and almost linear shape. In some parts the fibres may be seen extending in a transverse direction from the adjacent tissue, for a short distance, into the cartilage. A very large proportion of the fibres being rendered indistinct by the action of acetic acid, the cartilage nuclei then come into view; but their shape is so altered (linear), that their real nature can only be inferred by tracing the gradual transition from the oval and spherical shape.

The intercorpuscular substance or matrix is in some situations homogeneous and transparent, in others granular; and at wide intervals a few delicate fibres may be seen winding their course between the cells.

A transverse section of the bone seen under the microscope, by transmitted light, exhibits somewhat imperfectly the ordinary structure of osseous tissue. The Haversian canals are surrounded by lacunæ, which have a tendency to concentric arrangement, but are not so regularly disposed as in normal bone, and the canaliculi appearing as dark tortuous lines, spread out from the sides and extremities of the lacunæ.

* Elements of Anatomy, by Jones Quain, M.D. Edited by W. Sharpey, M.D. and F.R.S., and R. Quain, F.R.S. Vol. I, p. cxxvi.

With a view to ascertain whether the bone owes its origin to intra-cartilaginous ossification, a perpendicular section of the cartilage has been made, including at one extremity a very small portion of the partially ossified structure. Under the microscope the walls of the cartilage corpuscles (see *fig.* 4) are observed to become very indistinct in proportion as the boundary of ossification is approached; they appear, in fact, to have become confounded with the matrix, which has undergone a change from its original character: it is more opaque and coarsely granular than in specimens of the unaltered cartilage (see *Fig.* 5), and the tissue appears mapped out into oval or irregularly-shaped figures, the boundaries of which are dark, while the interior is bright, and contains one or more nuclei, that are for the most part arranged in groups, are smaller, and more closely approach the circular form than do the nuclei at a greater distance from the seat of ossification. At the confines of the cartilage and bone, the opaque earthy deposit is evidently encroaching upon the matrix of the cartilage, and surrounding the corpuscles: some of these are completely enclosed by an opaque material arranged in concentric lines, within which the nucleus of the cartilage corpuscle is still visible. Some corpuscles, however, are not completely enclosed, but the earthy deposit is perceived advancing on each side of the cell, leaving it free at one part. On bringing a deeper and older portion of the bone into view, little oval cavities with dark boundaries are observed; their appearance and arrangement are very similar to that of the lacunæ in the piece of bone already alluded to, but no canaliculi can be detected. The quantity of ossifying cartilage obtainable in a section being extremely small, and the transition being apparently very sudden in the structure of the osseous tissue, from mere cartilage with earthy matter impregnating the matrix, to an imperfect appearance of Haversian canals and lacunæ, no sound inference can be deduced as to the mode of origin of the latter, or as to the eventual metamorphosis of the cartilage elements.

The precise structure of the morbid growth having been ascertained, the question arises, To what class of tumours does it belong? Insomuch as the naked eye characters differ from Müller's original enchondroma*, and microscopically the cartilage in the

* On the Nature and Structural Characteristics of Cancer, &c., by J. Müller, M.D. Translated by Charles West, M.D., 1840, p. 97 *et seq.*

tumour above-described presents the adult and not the embryonic type of that structure, reasons might be advanced for not citing the growth in question as an example of enchondromatous formation. It is, however, remarkble that, of all the cartilaginous tumours described by Müller, the only one which resembled the adult cartilage with isolated cartilage cells and intervening firm substance, was an enchondroma of the testicle*.

As Dr. Walshe observes†, "adventitious cartilage, at one time believed to take rank among the most common, is now known to be one of the rarest of new formations." True cartilage is undoubtedly the characteristic structural element of the tumour above-described, since positive proof is obtained of its conversion into bone; and appearances presented by a perpendicular section of the cartilage force on the observer's mind the idea that the deep-seated cartilage corpuscles, in their approach to the surface, are undergoing a gradual metamorphosis, eventually to form a part of the surrounding fibrous tissue. The difference in the naked eye characters does not appear to be a sufficient reason for not regarding this tumour as enchondroma. Though colloid, encephaloid, and scirrhus, are very different in their physical aspect, they are nevertheless regarded as varieties of cancer, since their anatomical characters and effects on the constitution are fundamentally alike.

Considering that the tumour above-described agrees with Müller's enchondroma in process of ossification, 1stly, in the essential identity of the structural elements, and, 2dly, in its benignant character‡, rather than create new pathological distinctions (always inconvenient and unprofitable, unless established upon very weighty reasons), we regard the tumour as an ossifying enchondroma, though, in the present state of knowledge, it must be considered a very rare, if not isolated, example.

* Op. cit., p. 125.

† The Cyclopedia of Anatomy and Physiology. Article, Adventitious Products.

‡ The benignity of the tumour is proved by Mr. Cook's statement (in reply to our inquiries), "that the wounds in the scrotum healed in the usual time, and the horse appears to have improved in condition since the removal of the testicle." Mr. C. adds, "the horse is the sire of several colts."

Explanation of Plate.

Fig I. Represents the testicle, D, connected by the fibrous membrane, B, to the tumour, A; *b*, cartilage; *c*, cysts, of fibrous tissue mingled with fat; T, tunica vaginalis.

Fig. II. A perpendicular section of the cartilage; *c*, is the cell wall; *n*, the nucleus. On approaching the surface the cells are seen to be flattened, their sections being oblong, and in some parts almost linear. Magnified 300 diameters.

Fig. III. A section of the cartilage exhibiting well-marked nucleoli *n'*, also the granular matrix, *m*; the latter, however, is represented too granular and dark. Magnified 300 diameters.

Fig. IV. A section of the cartilage in process of transformation into bone. The cells are seen arranged in groups, and masses of granules to pervade the matrix. At *b*, the cartilage is impregnated with earthy matter, which at *c*, is seen advancing around a cartilage corpuscle Magnified 300 diameters.

Fig. V. A section of the ossifying cartilage seen with a lower power: it has a mapped appearance, in consequence of the grouped arrangement of the corpuscles, and the irregularly shaped masses of granules pervading the matrix. Magnified 150 diameters.

If the author is desirous of correcting an unintentional impression by acknowledging that he did not prepare a section of the above described cartilage in process of ossification, until after Dr Harvey had shown and fully explained to him a similar section obtained from a Ossified Enchondroma which Dr Lucas removed from the shaft of the

OBSERVATIONS AND REPORTS

ON THE

TREATMENT

OF

INTERMITTENT FEVER

BY

PHOSPHATE OF LIME AND SULPHUR.

[Extracted by permission of the Madras Medical Board from their Volume of Medical Reports for 1852.]

MADRAS:
PRINTED BY D. P. L. C. CONNOR, AT THE CHRISTIAN KNOWLEDGE SOCIETY'S PRESS, CHURCH STREET, VEPERY.
1852.

OBSERVATIONS ON THE TREATMENT OF INTERMITTENT FEVER, BY PHOSPHATE OF LIME WITH SULPHUR.

BY ASSISTANT SURGEON A. BLACKLOCK,
23d Regiment, W. I. I.

"If any one, in the present case, promises demonstration, his undertaking is certainly too much: if he desires or expects it from another, he requires too much. There are, indeed, very few demonstrations in philosophy, if you except Mathematical Sciences, that can be truly and strictly so called; and, if we inquire narrowly into the matter, perhaps we shall find none at all; nay, if even the Mathematical demonstrations are examined by the strict rules and ideas of Aristotle the greatest part of them will be found imperfect and defective. The saying of that philosopher is, therefore, wise and applicable to many cases." "Demonstrations are not to be expected in all cases, but so far as the subject will admit of them."

ARCHBISHOP LEIGHTON.

As the following considerations on Intermittent Fever and its treatment by Phosphate of Lime may at first sight appear to some minds more curious in their nature than profitable in their tendencies, I would predispose the reader to a lenient criticism by premising a few words on the difficulties which beset us in entering upon an investigation like the present.

The mental power of the wisest of our profession, in every period, from a very remote antiquity, has been freely employed on the subject of fever, yet our knowledge of the primary causes of many of its phenomena is still very limited, while the actual mode of action of most of our febrifuges is likely to remain an open question till important particulars in organic chemistry, with their bearings on animal vitality, are more satisfactorily determined than they are likely to be for some years to come. We have very little positive knowledge of the mode of origin of fevers. Some are clearly occasioned by local or by general irritation; others are, with

some show of reason, ascribed to the introduction of an animal ferment from without, or to its spontaneous formation within the system, through some error of secretion. Miasmata and Electricity assist us in part in endeavouring to solve the mystery, and partly render the mystery still more mysterious. Even a cryptogamic origin has been assigned to certain fevers. But much of our present knowledge of this subject is entirely of a negative nature, and the amount of really well ascertained and positively valuable information on the nascent state of febrile affections appears to be but small and insignificant indeed.

Medicine has been well described as the most uncertain of the Sciences, though it is based in part upon truths which pertain to other sciences and admit of most rigorous demonstration. We are to some extent acquainted with the molecular and atomic constitution of the human body in health, and with the changes which cells and cell products are subject to in the progress of disease; but we are almost entirely ignorant of the nature of the errors, be they vital or be they chemical, in which the simplest morbid processes are originated. Much of this ignorance or uncertainty is occasioned by the peculiar inherent difficulties attending all such investigations, or rather in the impossibility of subjecting the prime movers of morbid action, or even morbid actions themselves, to the test of experiment. The nature of the hidden spring is, to begin with, in general a subject of mere conjecture, while the train of subsequent morbid processes can only be made out at some few points whose interspaces must be filled in by the mental ingenuity of the observer, from attention to the few phenomena which present themselves under the name of symptoms at the surface. There has thus ever been great latitude for ratiocination on the subject of disease in general and of fever in particular. And the extent of supposition will not be diminished, and there will be abundance of doubt and uncertainty in the etiology of fevers, till their mode of origin and the leading governing principles of the morbid processes accompanying them are

more clearly defined and accounted for than they are at present.

The whole train of reasoning in the present paper is founded on observations which have led to the belief that loss of phosphates from disintegration of nervous tissue, or their deficiency in the circulating fluid, is a predisposing cause of fever. Whether or not this loss can be with truth assigned as one of the causes of fever we shall have to determine as we proceed. Organic chemists have drawn important, and I think fair, conclusions from data which appear to me not so perfect as those which my experience has provided me with for the construction of the present view. Alterations in the relative amount of cerebric acid in fevers, and the probable effects of the nitrogenized compounds in favoring the production of that acid for the repair of the nervous tissues, are neither very appreciable to the sense of the chemist nor of easy demonstration to the medical profession; yet Liebig has remarked, while speaking of the relation of vegetable alkalies to nervous matter, that "The time is not long gone by when we had not the slightest conception of the cause of the various effects of opium, and when the action of cinchona bark was shrouded in incomprehensible obscurity. Now that we know that these effects are caused by crystallizable compounds, which differ as much in composition as in their action on the system; now that we know the substances to which the medicinal or poisonous energy must be ascribed, it would argue only want of sense, to consider the action of these substances inexplicable."* Yet notwithstanding this hearty feeling of reliance on the capability of chemical science in banishing the obscurities of therapeutics, and notwithstanding the profound knowledge and depth of thought which Liebig has brought to bear on the chemistry of organization, he has still left the mode of action of the alkaloids to a certain extent unaccounted for. And if Liebig with his large intelligence and numerous advan-

* Animal Chemistry by Justus Liebig, M. D. London 1842, p. 187.

tages be considered by some to have made only an approximation to a true theory of the action of nitrogenized vegetable products, and to have left it yet far from complete, though it may be true to nature so far as he has carried it, I may well trust the reader will be indulgent should I fail to explain myself satisfactorily or to enter into every minute particular connected with the easily proposed but not easily answered question of "*How does phosphate of lime with sulphur appear to cure some forms of intermittent fever.*

During the last 100 years, or more, bark has been a medicine of acknowledged efficacy in many kinds of fever, and for the last 40 years quinine has enjoyed a reputation as an antiperiodic greater than ever was attached to any other medicine; yet during these long periods we have never been furnished with any truly complete and satisfactory explanation of the manner in which they prevent or arrest febrile disturbance. And, if I may be allowed to compare small things to great, it is quite possible that such will be the case with many remedies yet to be introduced, my own among the number. While I would, therefore, dwell strongly on what I believe to be a fact, that phosphate of lime with sulphur is capable of arresting certain forms of fever, and imagine that in time many will see fit to place confidence in the combination, I would ask for but a moderate share of regard for the theory I have here attempted of its mode of action, as the fact I have stated may be a very true and valuable fact, while the theory may be a very poor theory, and may fall very far short of correctly elucidating the *modus operandi* of the medicine.

We sometimes meet with intermittents which cannot for years be made to yield to either quinine or climate. If there be any good foundation for the modern opinion that quinine arrests certain fevers by supplying a compound similar to cerebric acid to the nervous tissue, it is plain that we presuppose some proportion of that acid to be absent in fever, though we cannot obtain any direct experimental proof. But if we allow that uncomplicated intermittent and remittent fevers are occasioned by the predisposition of the body to fever through the nervous system being deficient in tone from a diminution of the cerebric acid, we must also admit that a deficiency of any other constituent equally characteristic of nervous tissue may likewise predispose to febrile derangement. Now, in that tissue there are two acids, the Cerebric acid and Oleophosphoric acid, *both* peculiar to the tissue, and each of so much importance to its integrity of function that a deficiency of either is likely to be followed sooner or later by constitutional disturbance, the symptoms varying according to the nature and amount of the deficiency. No very accurate line of demarcation can be drawn between the parts of the nervous system which contain the largest proportions of cerebric acid, and those containing most oleophosphoric acid; but the former seems to abound most in the sympathetic ganglia and in the grey matter of the cerebro spinal system, while the latter is in greatest quantity in the white nervous substance. Quinine, which most resembles cerebric acid, has always been found most efficacious in those fevers which are attended with foul secretions and great liability to congestion of important organs, and which may, therefore, be regarded justly as having their origin in some defect in the constitution of the nerves of organic life; but cases are frequently met with where there is no decided derangement of important organs, and which yet experience only temporary relief from quinine, or refuse to yield to it altogether. It is fair then to infer that quinine, where it fails to benefit, is not the principle required to be supplied, and that there is a deficiency still to be made good by some other medicine. In fine, I am of opinion that, while quinine admirably fulfils the indications afforded by its chemical constitution, it can only supply one of two principles which appear to be deficient in the bodies of fever patients, and, therefore, cannot be of decided benefit in every case of fever; and I am inclined to

the belief that phosphate of lime will be found to make good the deficiency which most frequently leads to fevers among a large proportion of our native population. Quinine has been so long and so loudly extolled as an infallible remedy in the majority of the fevers of this country that it seems almost fair to ridicule the idea of its being equalled as a febrifuge by phosphate of lime in the majority of the cases which occur among natives, and of its being in many cases even superior to that alkaloid. But when we find some of the older and more experienced members of our service candidly confessing they have met with many cases in which bark and quinine have had but little satisfactory influence, it is time to inquire whether there may not be two classes of cases, the one requiring alkaloid remedies, the other earthy phosphates.*

During the last four years I have been gradually led by what I have observed in practice to consider the primary cause of intermittent and remittent fever to be a loss of balance between the nervous tissue and the other soft tissues of the body, and to ascribe the loss of balance in a large proportion of native cases to a deficiency of phosphates in the nervous matter. I had noticed many facts in the origin of these fevers and their amenability to treatment which appeared to be very discordant facts indeed; but which were rendered perfectly reconcileable on the supposition that they originated in a deficiency of phosphates in the nervous tissue or in the blood. I saw that these fevers were peculiarly prevalent in populations subsisting on a diet deficient in phosphates, and that even the people of districts well supplied with food abounding in these phosphates were also very subject to fever, notwithstanding the quality of their diet, if the atmospheric peculiarities of the district were favorable to the passing off of phosphates. I knew, what is known to most people, that the inhabitants of low lying places where the dew point is usually high, or persons passing through or residing in damp apartments or jungles are very subject to intermittent and remittent fevers; and I had noted as a coincidence that wherever the dew point is commonly high the tendency to the passing of phosphates by the kidneys is nearly as remarkable as the liability to fever.

* Madras Medical Reports for 1850, p. 106, et Seq.

These observations were sufficient to lead me to suppose that the use of phosphates might be of some advantage in those fevers, and I therefore determined to make cautious trial of some phosphatic preparation. I was the more induced to experiment in the matter as I was at the time passing considerable quantities of phosphates during the early parts of the day, alternating with febrile disturbance and red sedimentary deposits during the night. I began accordingly to take drachm doses of phosphate of lime and magnesia in the hope of deriving much benefit from it; but, though the red deposits and the attendant fever were almost immediately arrested I found the relief was only very temporary, as the phosphatic sediments continued to appear as usual, and even seemed to be increased in quantity. I had long known that the performances of the kidneys depend very much upon the condition of the mucous surface of the lungs; and, as I was living on the east coast in an atmosphere peculiarly conducive to this alteration of renal secretion, through impressions on the respiratory surface, I surmised that, unless the action of the mucous membrane of the lungs were altered for the better, there would be no prospect of improving the function of the kidneys. I give this as the beginning of my reasoning on this subject. It is enough for my purpose to state at present that, as the air of certain parts of the low country adjoining the east coast appeared to me to encourage phosphatic deposits by its depressing effects on respiration, it became necessary to find a method of heightening the respiratory power in those cases where it was of consequence to retain in the body the phosphate which might be administered as food or medicine.

As I have among a few of my medical friends, earned a small reputation for empiricism by ascribing to *sulphur* an

important power which no one but myself has ever believed it to possess, namely, that of checking morbid actions in the nerves of organic life, while it at the same time increases the nervous power of the spinal respiratory surfaces, the reader may be inclined to smile at my simplicity in again bringing forward this neglected drug as a powerful agent in helping us to render the system tolerant of earthy phosphates; but I must hope he will ere long give sulphur with this view that he may be practically convinced as I am, that I am not disposed to over-estimate its value in controlling or arresting phosphatic deposition in cases where the deposit does not owe its origin to long standing disease of the kidneys or bladder, or to injuries of the spinal chord. Sulphur, however, possesses that virtue in a surprising degree; and I now found in my own case I had only to combine it with the earthy phosphates to ensure their being retained in the system, even under the disadvantage of continuing to reside in an atmosphere which there can be no question has at certain seasons the property of leading to their deposition.

I next proceeded to make trial of phosphate of lime mixed with sulphur in others having somewhat similar symptoms, and then ventured to test its powers in the milder forms of intermittent fever among natives. The success in these ordinary private cases was so satisfactory that I would have tried the remedy in any severe hospital case that might then have offered, had it not been for the necessity I would have been under of being prepared with satisfactory proofs on paper to convince my medical superior in the Division that I had good and sufficient reasons for introducing it into my public practice, and that I was not idly tampering with the lives of the public servants by ill-devised or ill-directed experiments. Not that there was any necessity for hesitating to give the phosphate on account of any direct injury so harmless a remedy could possibly occasion; but because the fevers of the Northern Division are so decided, and tend in so many instances rapidly to a fatal termination, that any medicine, however harmless in itself, cannot but be regarded as negatively injurious in those fevers if its administration occupy the precious time which ought to be sedulously employed in arresting the fever by medicines of well tried and acknowledged efficacy. Sufficient and satisfactory reasons, however, for such an innovation in hospital practice I could not be expected to have ready at the call of higher authority at a distance, and therefore I had to wait for the occurrence of a severe case in private practice to test the real value of my remedy. No one has long to wait for the sight of a bad case of fever in the neighbourhood of the northern part of the east coast of India, and it happened that as I was soon after travelling past the Condapilly hills, the base of which is so notorious for severe agues, a healthy looking coolie in my employment was seized with intermittent in the severe form so common among the labouring classes of the Northern Division. He had intense shivering about three o'clock one afternoon followed towards evening by correspondingly increased heat of skin and delirium. At that time I gave him only an ordinary purgative of calomel and jalap, with warm tea to encourage perspiration. The following day he made his way to the next bungalow after being many hours on the road, and had about three o'clock in the afternoon a still more trying recurrence of the fever. I now, while he was delirious in the hot stage, forced him to swallow three drachms of phosphate of lime, with one drachm of sulphur, and in the course of an hour I was pleased to find the man sensible, and the perspiration coming with more than usual rapidity. Next day he got over his march in good time, and had the dose repeated early in the day. Two days afterwards he was carrying his burden and eating as usual, and had no return of the fever from the time he took the first dose of phosphate of lime till he quitted my service about a fortnight afterwards. Other cases of a milder character occurred while I was on this march and yielded as readily and satisfactorily to the same kind of treatment without the assistance of a single grain of bark or quinine.

Soon after this I happened to be detained for about four days in the jungle at a post station about 28 miles to the north of Neermul, and as I again began to suffer from phosphatic deposition and to experience the debility and irritability which usually precede fever, I again had recourse to the phosphate of lime with sulphur with the benefit I had derived on first using it. Another opportunity was afforded me at the time of trying its effects in a nearly similar case and with an equally favorable result. I made no scruple, therefore, when I assumed medical charge of the 23d regiment, W. L. I., and temporary charge of part of the 8th Light Cavalry, to employ the phosphate of lime with sulphur in the treatment of intermittent fever cases in both hospitals, and took an early opportunity of bringing its medicinal value to the notice of R. Davidson, Esq., the superintending surgeon of the force, who was pleased to countenance a continuance of the trial of its virtues as a substitute for quinine in certain forms of fever. Through his encouragement a very extensive trial was then made of the power of the phosphate of lime and a very fair body of evidence collected in its favor.

The majority of the cases of intermittent and remittent fever which have occurred in the hospital of the 23d regiment, W. L. I., since the 3d April 1849, have been treated more or less decidedly with this medicine, to the exclusion of bark, quinine, arsenic and the other antiperiodics; and it will be seen by a reference to the cases, taken from the hospital books, that though it was used with caution at first, more and more reliance has been placed upon its virtues till at last I may safely say it has enabled us to dispense with much of the emetic, purgative and alterative medicine usually regarded as essential to the management of fever.

Before proceeding further, however, it will be necessary to explain how the loss of balance, to which I have alluded, is likely to be brought about; and how a deficiency of phosphates in the circulating fluid may give rise to fever.

The diet may be deficient in phosphates, though to all appearance sufficiently nutritious for the requirements of all the tissues, and the blood may not be rich enough in materials to repair the waste of the nervous substance, or to supply it with healthy stimulation. To any one who has paid ordinary attention to the value of different articles of food for the various purposes of the body, as indicated by their constituents, the following table, drawn up, after careful inquiry, by that highly intelligent 2d Dresser P. M. Hyasawmy of the Kamptee civil dispensary, is calculated to afford some useful information, though I cannot turn it to as profitable account as I desire, owing to my want of statistics to show the comparative liability of the different classes of natives to fever.

Malabar or Tamil Sepoys.

10 A. M.	7 P. M.	
Rice lb. 1, Tyre oz. 12, Meat oz. 4 or Dholl oz. 4, Vegetable 1 pice, Mussaula, Chillies, Coriander, Saffron, Cummin seeds. Mustard, Onions, Garlic, Tamarind about oz. ½ Ghee 4 pice weight, Greens.	Rice lb. 1, Mullagatunny, or Dholl curry, sometimes fish.	Meat curry about 3 days in the week.

Malabar Officers.

10 A. M.	3 P. M.	8 P. M.	
Rice 8 to 12 oz., Meat oz. 4, Mussaul (the same as above) Ghee oz. 4, Mullagatunny, Greens, Dholl.	Milk lb. 1, Rice oz. 8, Sugar oz. 2.	Rice 8 to 12 oz., Mullagatunny, Meat oz. 4, Vegetable 1 pice, Mussaul, Ghee oz. 4.	Meat every day.

Gentoo Sepoys.

10 A. M.	7 P. M.	
Rice lb. 1, Meat oz. 4 or Dholl oz. 2 with Rosal Green, Mussauls with an excess of Tamarind about oz. 2 or 3, Ghee 4 pice weight.	Rice lb. 1, Mullagatunny or Dholl curry, Rosal green, Ghee 4 pice weight, Sometimes fish.	Meat about 4 or 5 days in the week excess of Tamarind.

Gentoo Officers.

10 A. M.	7 P. M.	
Rice 8 to 12 oz., Meat oz. 4 or Dholl, Rosal green, Mussauls with an excess of Tamarind, Ghee 3 ounces, Mullagatunny.	Rice 8 to 12 oz., Meat or Dholl curry, Rosal green, Mullagatunny, Sometimes fish.	Meat every day, excess of Tamarind.

Mahomedan Sepoys.

8 A. M.	2 P. M.	8 P. M.	
Rice lb. 1, Meat oz. 4 or Dholl oz. 2 with vegetables, Mussauls with Tamarind about oz. ½, Ghee 4 pice weight.	Wheat flour (Bread) lb. ½ with Ghee and Sugar.	Rice lb. 1, Meat or Dholl curry.	Meat every day.

Mahomedan Officers.

7 A. M.	10 A. M.	2 P. M.	8 P. M.	
Soojee Conjee with Milk and Sugar.	Rice 8 to 12 oz. Meat oz. 4, or sometimes Dholl oz. 2, Vegetables, Mussauls, Ghee 4 pice weight.	Wheat Bread with Sugar and Ghee. Milk.	Rice oz. 8 Meat or Dholl curry.	Meat every day.

Rajpoot Sepoys.

12 o'clock.	8 P. M.	
Wheat flour lb. 2, Dholl lb. ½, Ghee 6 pice weight, With a little Saffron and Salt, Milk lb. 1.	Sathoo lb. 1, consisting of roasted chena and wheat flour with Sugar, Water, or Chena lb. 1, and water.	No Onions, No Tamarind, Mutton very seldom, and never eat fowl.

Rajpoot Officers.

12 o'clock.	8 P. M.	
Wheat flour lb. 1, Dholl, oz. 8, Sometimes Meat oz. 4, Vegetable 1 pice, Ghee 4 oz, Mussauls, Chillies, Saffron, Cummin seeds, Lime 1, Milk lb. 1.	Rice lb. ½, Milk lb. 1, Sugar oz. 4.	Meat curry about 3 days in the week, No Onions, No Tamarind, and never eat fowl.

All the diets here noted are tolerably well suited to keep a man in health, except perhaps the Malabar dietary, which is very poor in quality. The Mahomedan and Rajpoot diets are well supplied with phosphatic matter while those of the Malabars and Gentoos are so deficient in materials capable of affording an adequate supply of earthy phosphates, that one might safely predicate, merely from an inspection of these tables, that those classes will be found to be most liable to fever.* In their diets every tissue but the osseous and nervous is supplied with a sufficiency of its appropriate constituents, and nutritive deposits are ensured to the skin, muscle, adipose tissue, &c., without a corresponding nutrition of the nervous substance. This daily addition to the general periphery of the body without a corresponding daily repair of the nervous tissue, increase of bulk without the attainment of proportionate nervous material, appears to me to be a common cause of ephemeral and intermittent fevers in the generality of Hindoos in the south of India.

The nervous system of those who eat rice to the exclusion of a more highly phosphatized diet is seldom capable of any sustained effort. Their skeletons are deficient in earthy matter, as is shown by the great frequency of lateral curvature of the spine among those who are subjected to medical examination for enlistment, and their blood

* Vide remarks at the end of this report.—A. L.

is poor in earthy phosphates, as is proved by the small amount of them to be found in the urine, at whatever period of the day it may be examined. The diet, however, may contain a sufficiency of phosphatic matter for all the requirements of the nervous and osseous systems, yet the phosphates may be rendered unavailable by the use of tamarind in the food, as earthy phosphates form very insoluble salts with tartaric acid unless the acid is in very great excess. The tamarind is a very good condiment for food rich in phosphatic and azotized matter, as the tartaric acid which it contains prevents the absorption of an excess of earthy salts and leads to increased excretion of nitrogenized materials from lungs, skin, kidneys and intestines; but it is for these reasons extremely prejudicial to the nutritive quality of the food of the poorer classes, however, useful it may be as a relish to their otherwise insipid meals.*

The loss of balance of which I speak may in some instances be occasioned by defective secretion. The occurrence of any check to the usual perspiration, any unusual slowness of the bowels, or depression of the respiratory power may lead to fever by rapidly increasing the bulk of the body, without any corresponding increase of nervous material in the blood, and with a decided diminution of nervous power, though it be more than ever required to keep the tissues and their fluid contents under vital control. As phosphates which have once formed an organized portion of the body pass off to a marked extent only by the urinary channel, while the other effete constituents are continually passing off by extensive surfaces, as the skin and bronchial and intestinal mucous membranes, more extensive, indeed, in proportion to the tissues whose *debris* they carry off than is the

* The diets are good specimens of what natives are partial to and take when they can obtain them. But it must be remembered that the majority of the lower classes of natives cannot afford the kinds, and quantities of food noted in these tables. In native regiments, at least ten per cent. of the men are so burdened with families, and more distant relations, that rice with some mussaul is nearly all they can obtain.

secreting surface of the kidneys to the cerebro-spinal substance, so a complete or partial arrest of secretion from the skin or bowels ensures at once a great preponderance of those tissues, especially of their fluid parts, which ought to be daily expended in order that what remains of them may be kept in proper proportion and subjection to the more permanent and less absorbable nervous substance. In this way exposure to a dry hot wind or to a sudden chill a few hours after a meal, when the effete matters of the old tissues ought to be coming to the surfaces in the form of secretion, will suddenly throw more responsibility on the nervous system than it is capable of incurring safely; and this increased demand upon it to keep up secretion against the check to which the secernents have been exposed without an adequate increase of nervous power to meet the necessity for increased vital control over the unusual bulk of the periphery, will lead to fever by leaving part of the chemical constituents of the body free from the government of the vital energy, and amenable only to chemical action with one another and with the surrounding atmosphere.

Or, again, the loss of balance may be occasioned by the deposition of an excess of phosphates with the urine; and the deposition may be the result simply of a separation at the kidneys of an excess of phosphates from the blood, as appears to be the case in confined damp atmospheres; or the phosphates may first of all be absorbed from the spinal cord, as sometimes happens after great bodily fatigue, or may be derived from the brain after the excitement and detrition occasioned by great mental application. In this country those who are liable to become our patients are little observant of the daily state of their urine, so that less is known of its peculiarities than of the changes in other excretions prior to the advent of fever; but I have occasionally met with cases where white deposits in the urine had been noticed several days before the commencement of fever; and, in some instances, medical attention has been first directed to these deposits from the person becoming alarmed

at what he has supposed to be purulent matter in the utensil, though he has been in fair health otherwise and not expecting to have fever. As these phosphatic deposits are frequently accompanied with a corresponding degree of general emaciation, fever does not always follow their appearance, though it does so often enough in well fed persons to permit us to view the loss of phosphates and the fever in the relation of cause and effect.

If the phosphates have once been carried off to any marked extent and not efficiently replaced, either by phosphatic medicine or a diet adequately supplied with phosphates, the person so suffering is very apt to have an attack of fever and is liable for years after to a recurrence of the same variety of fever if he exceed in his amount of eating what experience has taught him to consider compatible with health, or, in other words, avoidance of attacks of fever. Intermittent thus often leaves a person so liable to its recurrence that a single error in diet, one more than usually good meal, will lead in the course of twelve hours, or when sufficient time has elapsed to allow extra food to be turned into extra blood, to a sharp onset of the old type of fever, even after a quarter of a century has elapsed from the time of the first attack. When a man has once had an attack of the intermittent fever which is attended with red edged tongue and been treated with any preparation of cinchona he is, though apparently restored to health by the treatment, still left very liable to a return of the same form of fever, and hence it happens that a very moderate number of men furnish a very large number of cases to the hospital by their coming again and again upon the sick report. In a regimental hospital the constantly recurring return of the same individuals with the same kind of fever is often painfully annoying. The men are suspected of having become fond of the hospital, or, at least, disinclined for duty, and the medical attendant is apt either heedlessly to ascribe bad intentions to them or to grumble to himself, while all the while it is the course of prescription during their former

treatment which ought to be called in question and submitted to remark. They are purged and vomited to reduce the bulk of their reducible tissues and tone is imparted to their more permanent tissues by quinine; but when they leave the hospital their bulk increases steadily while the tonicity of their nervous system as steadily disappears, till the loss of balance between the nervous system and the other soft tissues is so decided that the least excess of food, or a check to the free action of the skin or bowels, necessitates a febrile or rather a chemical process as a substitute for that weak nervous control which was insufficient to keep the component parts of the body in correct vital relation to themselves. Something, therefore, remained to be attended to in these cases when we last had them in hospital and we have failed to perform it. We have reduced and cleaned out the machine, as we are well qualified for doing, and we have tempered the main spring a little better; but we have been blind to the fact that though tempering may impart strength it is a strength which cannot be lasting, as it is merely temporary strength from some variety of stimulation or tonification or some new arrangement of particles and not the true healthy and comparatively enduring strength which results only from a judiciously applied addition of fresh materials.

To return from our digression, however, there is yet, another way in which the nervous tissue, or rather I should say the blood on which it depends for its energy, may become deficient in phosphates. The air of damp close apartments too numerously inhabited is well known to those who have practised much among the poorer classes in England to be a common cause of great daily loss of phosphates by the urine. In this case the loss is usually followed sooner or later by fever of the remittent type, sometimes passing into intermittent, and phosphates continue to appear in the urine independently of mesenteric disease, though almost invariably followed sooner or later by derangement in that quarter. The air of jungles has the same effect on the

state of the urine. So has the air of all low lying land out of the free influence of the prevailing wind and covered with a steaming atmosphere, like that large extent of sunk ground behind the sand hills on the eastern coast, extending from the head of the Pulicat lake to near Vizagapatam. Nowhere perhaps are phosphatic deposits from the urine, even when it is not alkaline, more frequently observed than in our Northern Division and there are few parts of the country where intermittents and remittents are so common.

Apart from the fact itself that the earthy phosphates are passed off in excess by the kidneys when a person is living in damp close air, especially in the air of jungles or in the cultivated neighbourhood of marshes, we have some curious collateral evidence that the air of close damp places, abounding in living vegetation, must be highly favourable to the increase of urinary phosphatic secretion. The bones of tigers and other jungle animals become covered with exostoses after they have been some time in confinement as objects of exhibition. So far as I am aware, no explanation of the cause of this peculiar instance of the deposition of osseous matter upon bone has ever been offered. The animals are as well fed in confinement as they could have been in their native haunts. They have more constant exercise, from being kept in a constant state of excitation by the annoyance of their confinement, and this anxious nervous irritability of disposition is known to be highly favourable to the urinary excretion of earthy matter, from the depressing wear of the nervous system which accompanies that state, yet the phosphates are deposited abnormally within the body instead of being fully excreted. There must have been some agent operating on their system during their wild state to prevent this internal deposition by encouraging external elimination. On examining the skeletons of tigers in a museum we can tell at a glance which animal was killed in the forest and which died in captivity by the healthy smoothness of the bones in the one instance and their curiously rough encrusted appearance in the other.

What then is the essential and primary occasion of this marked difference? Other animals, whose natural place is the open plain, are little liable to these osseous formations seemingly because we do not bring them to an atmosphere their organization was not intended for when we reduce them to our service. While, however, the ox and the horse undergo no decided change of atmosphere in passing from the wild to the domestic state, the caged tiger is an instance of an animal suffering from an atmosphere which, though in reality purer than the air of its native woods, is no more suited for its lungs than is the cage for its idea of comfort. No air but the air of the forest will keep the functions of its lungs so in relation to its blood and kidneys as to enable the latter to eliminate any excess of phosphates from their circulation. In the jungle the feline tribe require to be possessed of high nervous energy, and it is ensured to them by the large quantities of condensed phosphates from the bone and muscle of their prey affording a constant supply of exciting material to their nervous system; but they obtain the phosphates to an amount which would be prejudicial to other parts of the body, after the requirements of the nervous and osseous systems are made good, were it not that the air they are intended to breathe induces a separation of the superfluous earthy matter from the circulating fluid, and finally from the body, through the agency of the kidneys.

It has often been noticed that those who have lived long in jungly and marshy situations and suffered from fever without showing any tendency to tubercle are almost certain to become affected with tubercles after their removal to a dry and airy open locality. This fact has often been disputed but has never been fairly controverted. As instances have fallen under my own observation, I am in favor of what appears to be the general belief on the subject, and I think it can only be explained by the fact that while the person was in the neighbourhood of the marsh, or jungle, the peculiar air of the situation, by its effect

on the circulation through the lungs, induced the deposit of an excess of phosphates by the kidneys, or so far prevented their assimilation, as to necessitate a constant febrile reduction of the other tissues to keep them, as already explained, in due relative bulk to the weakened nervous system; but that the better air, as it was erroneously supposed to be in this instance, to which the person was removed to enable him to get rid of the fever, having no power or property of affecting respiration, so as to induce phosphatic elimination and excretion, led to the blood depositing the earthy matter in weak organs liable to congestion.*

A similar change of respiratory and renal function to that already noticed as occurring in the tiger, when it is removed from the air of the jungle to what we erroneously consider a purer and more suitable atmosphere for all animals,

* It may at first sight appear very absurd to regard tubercle as an internal phosphatic deposit, but I have no hesitation in adopting this view, now that I have looked well to the matter. The most material difference between bone and old tubercle is that the one is organized and the other is not. The next important difference *connected with our present subject* is that while in bone the earthy carbonates and phosphates are united to gelatine formed directly by vascular action from chondrin, the same earths are deposited with a gelatinous medium (we cannot say dissolved in it). Neither need there be any difficulty in accounting for the formation of this gelatinous medium of the phosphates in tubercle. As we are endeavouring to show, the low state of the respiration in the confined air of a close room, or jungle, favors the deposit of an excess of earthy phosphates by the kidneys, and diminishes the azotized part of their excretion; while the better air of what are called healthy localities diminishes these urinary phosphates and increases the quantity of nitrogenous solids in the urine by the increased oxidating power of the lungs. If a patient be sent to a sea side station, after being some time subject to fever in an unhealthy district, the lungs and kidneys cannot at once regain their healthy relation of functions. The lungs form more urea and uric acid, but the kidneys are not at once adequate to pass them off, while the earthy phosphates which formerly were too readily separated are now detained in the blood. All this seems to be very far from my point; but as Liebeg has shown that gelatine may be produced by the addition, to the elements of Proteine, of water urea and uric acid(a) its formation in excess in the blood and its appearance as the agglutinating material of tubercular phosphatic deposits may not be so difficult to explain.

(a) Animal Chemistry by Justus Liebig, edited by W. Gregory, 1843, p. 143.

appears to take place in individuals who have in early life been subject to rachitis. Children who are rachitic from passing a superabundance of earthy phosphates in their urine, when living in a close damp unwholesome atmosphere, usually acquire in after-life a great increase of density in the skeleton, and in addition frequently become affected with tubercular deposit; though these are rare in rachitic cases owing to the free state of the kidneys in early life. This remarkable change from a great deficiency to a superabundance of phosphates in their tissues appears to be owing generally, to the increased care usually bestowed on the health of rachitic children: and to the animal diet upon which they are placed so soon as the extent of their ailment calls for medical interference and direction, but is chiefly to be ascribed to the peculiar air of sea side localities, which are generally selected by the dictates of experience as being not only the most suitable for their residence, but also more to be relied on for their restoration to health than all the recipes in our pharmacopœas. The result of experience, rather than any correct rationale of the mode in which the change ensures a favorable turn to such cases, has proved the value of sea air in their treatment; a marked change for the better, and that almost immediately, generally follows the location by the sea side of a patient who has been passing urinary phosphates: unless the deposit of the phosphates have been occasioned by disease of the spinal cord or bladder; or their loss have resulted in the establishment of some organic disease or general breaking up of the constitution; and this change for the better consists essentially in the improvement in the functions of the lungs, as is proved by the amendment in the urine. There can be no clearer proof of the virtues and value of sea air in arresting the abnormal passing of earthy phosphates with the urine, than the fact long ago pointed out by my old friend Copland Hutchinson, that sea-faring people and dwellers on the coast enjoy peculiar immunity from calculous deposits.

What then is the essential difference between pure ordinary air and that in the neighbourhood of a marsh, or jungle, which may contribute to the latter the property of originating a chain of actions in the system, resulting in the separation of an excess of earthy phosphates from the blood by the kidneys? It cannot be the moisture of the air alone which affects the system, else those who live on the sea or near its margin would be similarly affected. Nor can it be gaseous effluvia from vegetable *debris*, because living plants are generally capable of consuming these exhalations as they are produced; and, if they did exist to any marked extent, owing to the crowded state of a jungle, exposure to their influence would be more immediately followed by headache, nausea, or some other well known effect of mephitic inhalation. The carbonic acid from vegetable expiration might be supposed, with some shadow of reason, to occasion the headache, lumbar pain and lassitude which usually mark the commencement of febrile attacks, as it is well known to induce such feelings when breathed in a very diluted form in our laboratories, were it not that the innervation consequent upon a deficiency of phosphates in the blood accounts more satisfactorily for these symptoms;* and that the same symptoms when produced by the inhalation of air containing a small proportion of carbonic acid, in ordinary localities, may and do continue for long periods without materially affecting the health, and especially without being followed immediately, or within a reasonable limit of time, by any kind of fever. Even long confinement to apartments which though badly ventilated are still not over-tenanted, though it may enfeeble the constitution, is not so often followed by fever as to induce us to regard carbonic acid and fever as a cause to an effect; though we must admit that it, in common with

* As the phosphatized constituents of the blood are peculiarly adapted for the stimulation of the brain and spinal chord, a diminution of these constituents is likely to be followed by lassitude and pain in some part of the nervous centres; in the same way that headache is sure to follow the withdrawal of any stimulant, to which the nervous system may have been for a time subjected.

many other exhalations in jungles and marshes, may be an important adjuvant in determining the peculiar type of the fever.

Heat and moisture in the air were long regarded as the primary causes of fevers in tropical climates; but they cannot be considered as the real originators of intermittents, since we are aware that this class of fevers can be developed in temperate and even in cold countries, provided there be only a moist atmosphere with some extent of vegetation. An elevated temperature is doubtless a great promoter of that peculiar quality of the air which induces fever, but cannot of itself give rise to a first attack of intermittent. The electricity with which moist air is more or less charged at all temperatures would seem to be more concerned in the formation of a fever—producing atmosphere than any of the agents we have named, as it is present wherever there is evaporation, and always in greatest quantity where heat and watery vapour combine to promote that great electrical agent vegetation. Both animals and vegetables are known to pass off electricity with the vapour from their surfaces, and the quantity developed is always in proportion to the extent of growth and evaporation. The air in the vicinity of large masses of vegetation or of animals crowded together must, therefore, be loaded with it and must effect some animals injuriously, as it is as much a law of nature that all animals cannot safely breath an atmosphere which is in the same electrical condition as their own surfaces, any more than they can all safely use air vitiated by their own gaseous exhalations. The electric fluid cannot certainly be considered as in itself a poison; but moist air may be so charged with it that the breathing organs of animals may not be able to get rid of the electricity which ought always to be passing from them, and may thus be unable to maintain their proper electric condition. In the hydro-electric machine, which lately attracted so much attention, it was found that the excitation of the boiler could not be brought to any great degree of intensity if the atmosphere around it was allowed to become

charged with the positive electricity of the vapour; and, as the vapour from the lungs in health is known to carry off large quantities of positive electricity, it is not likely the lungs can be kept in their right state of excitation, if they are acting in a medium already loaded with the same positive electricity produced by evaporation and vegetation. Hence, the air of jungles and marshes would appear to be prejudicial to the health of some animals, primarily by its inability to carry off the positive electricity generated in respiration, so rapidly as to keep up sufficient excitation at the respiratory surfaces.

Intermittent is not confined to the vicinity of jungles and marshes; but is also found in many other localities destitute of these great favorers of positive electric development. Sea shores strewed with pointed rocks and bare isolated rocky hills become positively electrified when heated by the sun's rays, and give out their electricity during the night to the surrounding air, when it has become cool and damp; so that those who sleep in such places are equally liable to fever as those who spend a night in a thick badly ventilated forest. Crowded ships in stormy weather, when the hatches are kept on and the air between decks is not renewed sufficiently often, sometimes furnish intermittent cases owing to the number of men in a confined space vitiating the air with their electrical as much as with their gaseous emanations; and these cases are often of a very severe kind like those which occurred many years ago in the channel fleet under Lord Howe.

Defective aëration of the blood, owing to this absence of electric stimulation, must lead to immediate changes in the functions of the secreting organs, as well as to increased bulk of the non-nervous soft tissues from partial arrest of secretion; so that loss of balance between the nervous substance and the other soft tissues may be brought about even where there is no actual disintegration of nerve matter, nor consequent loss of earthy phosphates. The connection between defective respiration and alterations

in the urine has not obtained the attention it deserves, although the functions of the respiratory surfaces and of the kidneys are most intimately related. It is now generally understood that the kidneys have no part in the formation of the urine, but merely eliminate it from the blood, into whose current its constituents are carried as fast as they are formed by the respiratory destruction of organized matter. The quality of the urine thus varies according to the degree of respiratory oxidation to which the constituents of the body are subjected. When the system is thoroughly oxygenated in health the animalized portion of the urine consists almost entirely of urea, with a very small proportion of uric acid; but when the aëration is less perfect the uric acid is increased in proportion as the urea is diminished. A lower degree of oxidation, such as we see brought about by want of action of the skin during the continuance of a common cold, eventuates in the formation of urate of ammonia. While in the fœtus where the respiratory surfaces have not begun to act, and in epidemic cholera where they have almost ceased to act, the urine is albuminous, the Malpighian bodies appearing to afford increased facilities for the transmission of unorganized products from the blood in proportion as the cells of the tubuli urineferi cease to eliminate the highly oxidized debris of the system. The albuminaria of cholera is doubtless partly the result of the congestion of the kidneys, and, therefore, partly independent of the congestion of the lungs or the want of respiration; but the albuminaria of newly born children cannot be ascribed to any turgescence of the renal veins. Nor do we expect to find the kidneys congested in every recent case of simple albuminaria in the adult, any more than we look for disease of the liver or kidneys in every case of diabetes mellitus, though we may be almost certain that in every case of albuminous or saccharine urine we shall find the skin to be very inactive and the respiratory murmur feeble or masked by submucous rales, even when we cannot find any decided evidence of organic disease. And, again, when the urine has been long albumin-

ous or saccharine, it sometimes suddenly ceases to contain either of these abnormal ingredients and has its usual urea and uric acid restored 36 or 24 hours before death, owing to the function of the lungs being all at once morbidly increased by irritation of the roots of the respiratory nerves, preceding effusion at the base of the brain. As the quality of the fluid passed by the kidneys is therefore as much influenced by the extent to which the lungs perform their functions as it has ever been proved to be by the state of the stomach or liver, it follows that the constituents of the body passed by the kidneys may vary according to the quality of the respired air. We know very well that they can be altered merely by change of air, since we find that young persons passing excess of earthy phosphates and very subject to remittent fever, as already noticed, from living in close damp apartments, vitiated by their own gaseous and electrical emanations, cease to pass any such excess when they are placed in opposite circumstances, and their lungs are stimulated by fresh air in any place, but more especially at sea bathing quarters where the air is rendered more stimulating from containing saline particles. The phosphates, however, are always more slowly replaced than any other constituents of the body, as every kind of diet does not contain them in quantity sufficient to replace any deficiency very quickly. So that if a person has lost phosphates, by being any time in an atmosphere highly electrized by either plants or animals, he will be long subject to fever unless the phosphatic loss be speedily made good. From three to ten days is the period which usually elapses between the time of passing through a jungle and the coming on of fever, or between the time of losing phosphates and acquiring a redundancy of other constituents liable to chemical changes if the blood has become in any way less suited for the requirements of that nervous system, which can alone, by its energy, keep their chemical tendencies in control. Those who have but little tendency to plethora, or are careful in their diet, may have a longer immunity from fever after being in a predisposing atmosphere, just as we find in those who are subject to the constant recurrence of intermittent that it occurs at longer and longer intervals as their assimilating functions become feebler, either by the injurious effects of the fever on their constitution or by their advance in years. Some are sure to have an attack of their old fever when a trifling plethora follows a slight excess. Others again never have a visit from their old enemy till the rain season, or the setting in of the land wind, occasions a sudden plethora by diminishing perspiration. One man acquires in three days by virtue of good assimilating powers an amount of body which a feeble or badly stimulated nervous system cannot keep from chemical decomposition. Another makes blood more slowly and requires a fortnight or more to be fit to develope fever; while some would soon cease to have intermittent were it not that the moon's influence induces or adds to this plethoric condition.

Ever since intermittent fever has attracted medical attention endeavours have been unsuccessfully made to account for its regular recurrence after certain intervals. How far the phosphatic theory may assist us in the elucidation of the question, it is for the reader to determine.

First effects of loss of balance.—Increased bulk of the soft tissues of the body with the exception of fat, without a corresponding increase of nervous matter to keep it in vital control, is sure to result sooner or later in the conversion of the abnormal increase into effete matter; and, if no shock have been imparted to the nerve fibre by its presence, to its speedy separation and expulsion from the body, as we daily witness in copious sweats and movements of the bowels after excess of food and in the spontaneous perspirations and purgings which relieve the system in fevers, and which we endeavour to imitate in our treatment. In many severe cases of fever, which come under our notice, the effete matters which are passed off at the commencement of the treatment, or of the curative process set up by natural effort, appear never to have been properly vitalized: and the system seems to have

been suffering for days before the actual onset of the fever from the presence of this half vitalized or, as we may call it, half dead matter. The greater the accumulation of this effete matter in the system the more apt is the fever to take on a typhoid form, however simple it may be at its commencement; but as all fevers may take on this low form if the semivitalized matters which have led to the beginning of the fever prove too much to be thrown off at once by strong nervous influence, so it may be inferred that though many seemingly opposite circumstances give rise to fever, and we enumerate several seemingly distinct varieties, yet that the real immediate cause of fever is in every case the same, though the opening symptoms appear to be widely different, and every case would follow nearly the same course and terminate in a crisis either by stool, by perspiration, or by urine, and at nearly the same periods, were it not that the quantity of the semivitalized or effete matters thrown upon the nervous system, as well as the vital power of the nervous system itself in bearing up against the excess, varies as much in different cases as one individual differs from another in size or appetite or configuration or any other of his natural attributes.

However the loss of balance may be brought about, the result after a time is fever; or in other words, the result is the natural endeavour of the system to restore the balance in the only way it possibly can be restored without extraneous assistance, that is by the soft tissues entering into new and simpler combinations with one another or with the atmosphere, which every part of them speedily commences to effect when deprived in any way of the full vital control of the presiding nervous system. One leading feature in the chemical reaction at the commencement of fever, though not at the first of the commencement, is the absorption of oxygen by the lungs and skin, during the early stage, and the consequent slow burning off of the combustible constituents in order to bring the soft solids to their proper ratio to the nervous system,

so that when a sufficient reduction of the body has taken place in this way the nervous system is able once more to take the vital control of the tissues and assert its supremacy.

But, with some exceptions soon to be noticed, there is in every fever a stage preliminary to the hot stage or period, during which the body undergoes reduction by accelerated respiratory combustion. The hot stage is the period of oxidation, but before the hot stage, there is, strangely enough, as it at first seems, a cold stage or period of deoxidation. In the hot stage an excess of oxygen is drawn into the body, but in the cold stage oxygen is actually withdrawn from the greater part of the body. In the cold fit the abdomen is too warm, or at least of the natural temperature, while the skin and the air expelled in expiration are considerably below the natural temperature. In a well-marked cold stage, though the air appears to permeate every part of the lungs, and the chest yields clear sounds to percussion, the ear, even assisted by the stethoscope, fails to detect the respiratory murmur except over a small extent immediately below the clavicles. Inspiration and expiration are proceeding even faster than usual, but true respiration, or union at the pulmonary mucous surface of the carbon with the vivifying principle of the atmosphere, is in great part at a stand still. The lividity of the skin would lead us to infer there is pulmonary congestion, but this lividity is produced in reality by abdominal congestion and absence of true pulmonic respiration. The lungs sometimes become congested secondarily in the cold stage in very serious cases; but in the cases usually met with they are very poorly supplied with blood, which is during this period drawn into the abdomen by becoming venous and accumulated in the great venous receptacles. Now the blood being in these great venous repositories must be venous, as no property of holding or being intended to hold arterial blood has ever been imputed to them; and, as this great accumulation of blood

is venous, how has it become so. To reduce so large a bulk of blood to the venous quality, a large quantity above the proportion usual in health must have lost its arterial properties; and, if so, how has an unusual proportion of the circulating fluid lost these properties. *Has it failed to acquire* these arterial properties by want of vital power at the moment in the lungs, or has it *lost* these properties in passing through the arteries of the abdomen. As the shivering, fulness in abdomen, and pain in one or both sides come on before any failure in the respiratory murmur can be detected with the stethoscope, I would say that the great bulk of venous blood in the abdomen has parted with its arterial properties in the arteries of the whole system, but more especially in the abdominal arteries, and that it has not primarily become venous from any decided want of power in the lungs to render it arterial; though we must admit the amount of their calorifiant work is diminished by the great temporary depletion they have undergone in consequence of the great temporary abdominal congestion.

Now, how has this great degree of abdominal congestion been brought about, or in other words, what has led to so great a loss of the oxygen of the arterial blood as to reduce so unusual a preponderance of it to venous? we must now call to mind what has been already advanced concerning the want of nervous power, either from the accumulation of tissues other than the nervous or from non-assimilation of the earthy phosphates or their actual loss prior to an attack of fever, and what has also been adduced concerning the liability of the constituents of the body to undergo chemical rather than vital changes when the nervous control is feeble, and we will not be slow to perceive that if the nervous control over the tissues be weak, and especially if the fluids of these tissues be superabundant, there will be an immediate tendency to the development of their chemical affinities. And as all the constituents of the body have, either combined or separately, a more powerful affinity for oxygen than they have for one another, the oxygen of the arterial

system will be rapidly withdrawn by these half vitalized or rather half dead matters, or portions of tissues which have already been spoken of as giving evidence of their accumulation before the access of fever; much arterial blood will necessarily become venous; and, as the chemical process of the oxidation of these half vitalized tissues at the expense of the oxygen of the arterial blood will be most decided in the abdomen, which is at all times the great store house for them, so the greatest formation of venous blood and its most marked congestion will take place in the abdomen also.

This then appears to be the deoxidizing process which gives rise to the cold stage of fevers. The oxygen of part of the arterial system appears to be absorbed by particles of the body which through the absence of adequate vital control are allowed to exert their chemical affinities to a degree which is unusual to the general healthy oxygenation of the system. A large proportion of the arterial blood is deprived of its oxygen and becomes venous, and is accordingly hurried into the venous reservoirs; and the fall of the temperature of the body results, in this way, not only from the actual withdrawal of the oxygen from the arterial system by the chemical attractions of these portions of the body deficient at the moment in nervous controlling power to hold them together; but from the abdominal congestion consequent upon the inordinate formation of venous blood in this manner from arterial, leading to a great reduction of the quantity of circulation through the lungs.*

Gradually, however, the demand for oxygen ceases; gradually the congested venous blood makes its way to the lungs, and gradually and steadily, as the venous blood comes to be reoxygenated in the pulmonary vessels, is the temperature of the system raised; and as the temperature resulting from pulmonic respiration is within certain limits in direct proportion to the quantity of venous blood which is being

* The deoxidizing power of organic matter while undergoing certain changes is well illustrated by yeast reducing the oxides of mercury to the metallic state.

aërated, so the heat and excitement and duration of the hot stage of the fever are exactly proportionate to the chill, depression and degree of congestion of the cold stage. Occasionally, as is well known, this reaction is never brought about, the deoxidizing process towards the central parts, and with it the formation and accumulation of venous blood in the abdominal veins and viscera, proceeding to such an extreme degree of congestion that blood is withheld from the lungs till death ensues from the whole system being deprived of oxygen; or, as more frequently happens, the blood returns so rapidly upon the lungs that the patient is suffocated by their being in turn congested during the first part of the stage of reaction. The ordinary termination, however, of the fever is the gradual development of a hot stage, and the termination of the hot stage in copious warm perspiration and watery exhalation from the lungs, from the union of oxygen and hydrogen resulting partly from the chemical deoxidation of arterial blood in the body during the cold stage, and partly from the subsequent additional pulmonary and cuticular respiration during the reaction. With this, and as a consequence of the increased exertion of the function of respiration, we have also an increase in the quantity of uric acid secreted by the kidneys, forming another evidence of the increased low oxidation and detrition of the tissues; and also in some cases profuse spontaneous bilious vomiting and purging terminating the hot stage, and bearing evidence to the extent of the primary deoxidizing process, and to the great necessity there has been for a critical and salutary throwing off of carbonized matters.

By this spontaneous reduction of the body in fever a complete removal of the fever is frequently, though slowly, effected; and it is found that the entire cessation of the febrile tendency is most speedily brought about in those cases which have had most accessions of fever in a given period. Instances of the kind are common to every medical man. In these cases the subsidence of the fever is effected by the febrile wasting of the reducible tissues, leading to the restoration of the loss of balance which existed between the bulk of the soft tissues and the bulk or power of the nervous system, as I have endeavoured to explain in a former part of this paper. But it may be asked, how happens it that while the greater part of the bulk of the soft tissues of the body is being reduced during fever, the substance and power of the nervous system is not reduced also. The nervous tissue is always last to suffer reduction in all wasting diseases, and appears to preserve its texture and its power perfect and intact almost till the last period of life, unless the disease be attended with great determination of arterial blood to the brain so as to lead to early destructive oxidation of that organ. Ordinary cases of pulmonary phthisis and cases of starvation are instances in point; and in these delirium never appears till the emaciation has proceeded to a great degree and the brain has begun in its turn to undergo absorption.

Experimental physiology has proved that when emaciation is set up by any disease attended with febrile reduction or oxidation the fat goes first. This tissue, however, I do not consider has much connection with my subject, as it is a tissue in no way dependent on the nervous system; and, whether superabundant or deficient, has little, if any, modifying effect on tendency or otherwise to fever. The muscular and cellular tissues are next to be absorbed in emaciation, then the tendons and parenchyma of the viscera of organic life, and last of all the nervous substance. For this reason the mere induction of a certain degree of emaciation, the degree varying with the case, is sufficient to bring all the purely protein tissues under the control of the nervous system; or in other words, to give the nervous system a paramount preponderance over the rest of the organism. Practitioners know well that a spontaneous diarrhœa sometimes precedes the subsidence of every variety of fever, and cases of intermittent occur occasionally which defy all our most approved remedies and yet yield at length to the reducing effects of profuse colliquative discharges, the effect and the termination of a severe oxidizing process which leaves merely as

much of the body remaining as can be guaranteed by the vital force of the nervous system from any further febrile encroachment.

Our endeavours to restore a fever patient to health ought to be based, and, indeed, are based, on the spontaneous proceedings of the body in its attempts to rid itself of fever. We prescribe emetics, sudorifics and purgatives to bring the mass of the circulating fluids into better correspondence with the bulk of the nervous tissue. As the nervous system has been lowered in power by want of phosphates in the diet, or, as is sometimes likely to be the case, has the amount of its solids reduced by the passing off of the phosphates prior to the advent of the fever, we proceed to diminish the bulk of the other soft tissues and assist nature to re-establish the healthy ratio between these and the remaining power of the nervous fibre. Aware that the lowered or depressed nervous system is incapable of controlling the chemical actions of the usual mass of the body, we reduce the body till it be small enough to be fully pervaded by the remaining amount of nervous vital action; or, as the weakened presiding power cannot govern its kingdom without permitting intestine ferment and commotion, we take care the presiding power shall have the less to govern—no more, in fact, than it can fairly keep in subjection.

Or, we proceed in certain cases by increasing directly the nervous power itself by the administration of remedies which impart some peculiar principle to the nervous tissue and tend at the same time to prevent further disintegration. It is the opinion of Liebig and his followers that the good effects of quinine and other medicines of its class are owing to some constituent or elementary substance which is common to them and to the nervous system. "Indeed," says Liebig, "it cannot be considered merely accidental, that the composition of the most active remedies, namely, the vegetable alkaloids, cannot be shown to be related to that of any constituent of the body, except only the substance

of the nerves and brain."* And, again, "It is singular that we find medicinal agencies all depending on certain matters, which differ in composition; and if, by the introduction of a substance, certain abnormal conditions are rendered normal, it will be impossible to reject the opinion, that this phenomenon depends on a change in the composition of the constituents of the diseased organism, a change in which the elements of the remedy take a share; a share similar to that which the vegetable elements of food have taken in the formation of fat, of membranes, of the saliva, of the seminal fluid, &c. Their carbon, hydrogen or nitrogen, or whatever else belongs to their composition, are derived from the vegetable organism; and after all, the action and effects of quinine, morphia, and the vegetable poisons in general, are no hypothesis."† Long, however, before these views were promulgated most of the vegetable alkaloids had been found highly useful in fever, owing to their distinguishing property of heightening the power of the cerebro spinal system, and it seems remarkable that the analogy between the composition of some of our best vegetable antiperiodics and the composition of cerebric acid, one of the chief constituents of the brain, was not sooner looked to for an explanation of their remarkable powers over the organism. The great differences between the nervous substance and the other tissues of the body are, that the nervous substance contains oleo-phosphoric acid and cerebric acid which are not found in the rest of the tissues. The phosphorus in this phosphorized elaine is from $\frac{1}{20}$th to $\frac{1}{30}$th of the whole substance of the solids of the brain, while the cerebric acid, combined with soda, forms another essential part, though in what proportion has not, I believe, been as yet determined. According to the most recent views, the oleo-phosphoric acid must be supplied to the nervous system directly

* Liebig's Animal Chemistry, edited by William Gregory, M. D., &c. &c. 1842, p. 185.

† Idem, p. 188.

from the food, while the cerebric acid must be furnished indirectly by the choleic acid of the bile, as none of the ordinary constituents of our food at all resemble it in composition.

According to Fremy, whose analysis has superseded that of Vanquelin, cerebric acid consists in 100 parts of

Carbon,	66·7
Hydrogen,	10·6
Nitrogen,	2·3
Oxygen,	19·5
Phosphorus,	0·9

According to Dumas and Demarcay, choleic acid of bile consists of

Carbon,	63·77
Hydrogen,	8·821
Nitrogen,	3·255
Oxygen,	24·217

and according to Liebig quinine is composed of

Carbon,	75·76
Hydrogen,	7·52
Nitrogen,	8·11
Oxygen,	8·62

The analogies in composition are here sufficiently striking to incline us to the belief, that medicines of the quinine class are admirably adapted either to replace cerebric acid or to furnish the means for its ready formation, and we are therefore led to conclude that this class of medicines serves to arrest fever, partly at least, by supplying a principle in which the nervous system is at the moment deficient; but it can only be said that the vegetable alkaloids serve to arrest fevers *partly* by increasing cerebral nutrition, for there are differences in the proportional quantities of some of the constituents of cerebric acid and quinine, for example, which prove to us that they cannot, as a whole, be applied by the system to the requirements of the nervous substance; while

these differences enable us at the same time to throw some light on other important points in their febrifuge action. Quinine contains only half the proportion of oxygen contained in cerebric acid, while it contains four times as much nitrogen; so that while quinine as a whole is capable of contributing to the solids of the nervous matter, and thus imparting to the nerve texture greater vital controlling power over the other tissues of the body, it still affords an excess of nitrogen which cannot but be of essential service in preventing a further febrile reduction of tissues, by acting as an antagonist to their oxygen, the great promoter of the waste of the body during life, in health and disease, and of putrefaction after death, by its affinity for carbon and hydrogen. This antagonism of nitrogen to oxygen comes into effect to the immediate benefit of a fever patient, to whom bark or quinine is administered, long before the nervous tissue can receive any direct support from the alkaloid, and the oxidation of the tissues thus receives a chemical check, till the medicine can repair the nervous matter and afford to it such an increase of true vital power, as will enable it of itself to keep the chemistry of the non-cerebral tissues in proper vital subjection. This antiseptic property of bark and quinine has been little heeded of late years, though their primary febrifuge effects cannot be fully understood unless we give it due consideration. For this property it must ever, in all probability, remain unequalled in the treatment of all fevers where there may not be enough of vitality, to keep in subjection the chemical affinities of the tissues for their own and the surrounding oxygen, and where there is a decidedly septic tendency. Most of the alkaloids, from their containing a large proportion of nitrogen, are thus of more or less value in the treatment of fevers, and the value of each appears to depend very much on the relative proportions of nitrogen and oxygen. The greater the proportion of nitrogen the greater is the antiseptic and febrifuge power. Morphine contains double the per centage of nitrogen to what is contained in cerebric

acid; and opium, therefore, since the days of Trotter and Lind, has been found a first-rate febrifuge and antiperiodic, when given freely and early in the hot stage of intermittent, and owes its good qualities in these respects in all probability to its power of furnishing azotized matter to the nervous system; but, as morphine contains a great excess of oxygen, opium is worse than useless as an antiseptic, and may not be given where the vital power has become so weak as to permit the chemical affinities of the body's constituents to come into operation.

Or, if the case be assuming a grave form, the nervous power becoming much depressed, the secretions and excretions foul from the extent of chemical decomposition, and scanty from the want of nervous energy to move them to the surfaces and expel them from the body, we subject the patient to the influence of mercurial medicines. The salts of mercury alone in such a case will generally occasion dark watery offensive purging without being of service, but the reverse, to the patient; but if the mercury be given in very small and often repeated doses, or be so combined with other medicine that the now irritable spinal system may not be excited to pass it outward, we find after a time, which varies very much according to the depth of the typhoid stage, that very important changes are brought about. The pulse becomes decided and excited; there is more appearance of life about the patient and he moves his limbs more frequently, the secretions begin to be increased, as is first evidenced by the skin, and then copious expulsive movements of foul secretions take place which could only be occasioned by some new vigour communicated to the nervous centres through some improvement in the quality of the circulating fluid. Mercury of itself could not effect these wonderful changes, for mercury is not a constituent of the human body and has nothing in itself to make it capable of conferring power to a diseased body to imitate the vitality of health. It cannot then be from the mercurial salts employed in fever that we more immediately derive the wonderful accession of vital

energy which is expressed at the period they are seen to affect the system; but from some essential life sustaining agent being brought into circulation by their agency.

Now, as there is proof that the loss of the phosphates from the blood, or their being deficient owing to peculiarities in the diet, will lead to fever, so we have pretty clear proof that in those low fever cases which we seek to benefit by the constitutional effect of mercurials the benefit does not accrue to the patient, that is to say the spinal nervous system is not roused, nor does excretion and expulsory removal of the old debris of chemical decomposition occur, till the absent phosphates are returned to the current of the blood. If we have a typhoid fever patient with an old node on one of his shins, the slightest change in the surface of this phosphatic deposition will be the signal that the insoluble phosphates are about to be taken into the circulation and that the patient will soon be roused and the secretions and excretions be vigorously resumed. The poorer the blood may be in phosphates the worse will be the fever case, the greater will be the need of phosphates being drawn by the influence of mercurials into the sanguineous current, and the more decided will be the absorption of the spongy alveolar processes, and other loosely aggregated parts of bones, before any sign of improvement becomes apparent.

Mercurial preparations have two connected yet widely different actions, and unfortunately in some respects, we cannot keep these actions distinct and separate in practice. In ordinary states of the body, when all its constituents are in their normal proportions, mercurials at once encourage secretion by acting as general stimulants to the glandular surfaces; but when phosphates are deficient (as we may satisfy ourselves in any case by the urine) they have first to bring them from the fixed deposits into the general circulation before healthy secretion can be re-established.* Secretion in fact depends entirely upon the integrity of the constitution of the blood,

* Lancet, Vol. I. for 1845, p. 9.

and if any one constituent of that fluid be deficient the secreting organs cannot furnish their healthy products for want of the requisite material. But the earthy phosphates have a much more important part to perform than any of the other constituents of secretion, for not only do they enter freely into all the secretions, but the nervous matter which prompts and directs secretion is dependent upon them for the essentials of its highest development and vitality. So that phosphates are not only absolutely necessary to constitute an important element of the principal secretions, but are at the same time the prominent chemical feature in the constitution of the nervous system which has to govern the largest secreting bodies.

In protracted and obstinate cases we have recourse to arsenic, our most powerful remedy in constantly recurring intermittents. Where phosphorus is deficient, arsenic may be made to serve as its substitute or, as Sir Robert Kane has it, "the resemblance of function, so often alluded to, between arsenic and phosphorus is such, that the latter element, which characterizes the animal tissues by its almost constant presence, may be replaced as a constituent of our bodies by arsenic."* The preparations of this metal however are almost never required except in cases where there is a constant tendency to plethora and its utility appears to consist simply in the power it possesses of permanently lowering the assimilating functions. It cannot impart any superior vital quality to the system, except in so far as it may make up for deficiency of phosphorus, nor does it possess a power of bringing any necessary constituents of the body into circulation; but it so effectually lowers the powers of organic life, after a time, that the tendency to any undue increase of the volume of the blood is effectually arrested. No other medicine appears to possess in so remarkable a degree this power of lowering or even destroying the functions of assimilation and nutrition, and of arresting the development

* Elements of Chemistry by Sir Robert Kane, 2nd Edition, 1849, p. 542.

of the body, and the propriety of administering it, even in intermittents of long continued recurrence, ought always to receive very serious consideration. The vital power is always tolerably good so long as the body can develope a well marked intermittent, shaken and broken though the constitution may appear in cases of long duration, and we should be slow in exhibiting a remedy whose most marked effect is that of reducing the organic functions to so low a vitality that they are for a time or, it may be, forever, unfitted for ministering to vascular repletion. Arsenic has been found of great value in skin diseases, partly from its acting as a substitute for phosphorous,* and partly from its property of diminishing the assimilating activity of the intestinal surfaces, so that they take up no more nutriment than can be readily turned to healthy account by other organs; and it may be found hereafter, on this account, a valuable medicine for the prevention of plethora in individuals who are threatened from time to time with apoplectic and hemoptytic attacks, from the constant recurrence of this plethoric state; but we must consider it inadmissible for keeping down the bulk of the body in those who have a constant tendency to intermittents, if we can bring ourselves to believe that our philosophic course of proceeding in the treatment of these harassing periodic fevers is not to reduce the body generally and permanently, so much as to communicate a permanent power and tone to the nervous system, that it may be capable of preserving any bulk of body in uninterrupted and harmonious action.†

* An interesting paper on the value of phosphate of lime in certain leprous affections was read to the Medical Society of Kamptee, 10th October, 1849, by Assistant Surgeon Tribe, who brought before the Meeting a Cavalry Sepoy who had recovered under its employment.

† In advancing this opinion I am at the same time well aware of the great value of the white oxide of arsenic in strengthening the nervous system when judiciously administered. But, when given in sufficient quantity to replace absent phosphates, and long enough to eradicate an obstinate intermittent, I am convinced it diminishes the absorption of chyle, from the peculiar character of the intestinal evacuations during its exhibition and long after we have ceased to exhibit it, if we have been obliged to continue it for a considerable period.

Or, last of all our instances, we may set free, phosphoric acid in the blood, and so make it more available for cerebral nutrition, by administering some acid, as the sulphuric, having a greater affinity for the bases of the phosphatic salts. Liebig shows in his letters, that this arrangement must certainly follow the absorption of diluted sulphuric acid, though he does not appear to perceive the importance of a knowledge of this fact in the treatment of fever. Both sulphuric acid

The following accurate description of the effects of the white oxide of arsenic primarily on the secretions and excretions, and secondarily on the cerebro-spinal system, especially on the nerves allotted to respiration, renders my view of its action more than probable, though the writer has unfortunately failed to notice its *ultimate* effects on assimilation.

"The appetite and thirst are moderately increased, the secretion of urine becomes more abundant, and *the evacuations from the intestines often more frequent, and of a pulpy or pappy character.* From the intestinal canal the peculiar action propagates itself over the whole system. The heat of the surface is augmented, and the increased temperature is experienced particularly about the forehead and eyebrows, and the skin is bedewed with a bathing perspiration. At the same time an increased strength and frequency of pulse is felt. The whole muscular system acquires energy and elasticity; the involuntary muscles especially become more powerful and vigorous in their action; the respiration is gently accelerated. The nervous system partakes of the impulse communicated to the frame, and the spirits as well as the courage of the individual rise, liveliness and regularity characterizing the whole functions of the system."—*Penny Cyclopedia, article "Arsenic."*

This description would apply almost equally well to the effects of phosphate of lime, except that the stools during a course of the phosphate appear to be more the results of excretion than of non-assimilation, such as is indicated by the frequency and pappy peculiarity noticed in the quotation; and that the frequency of the pulse is diminished, and often remarkably, by phosphate of lime, while its strength is just as much improved by it as by arsenic. Healthy secretion cannot proceed while the pulse is frequent, and medicines which steadily lower the frequency of the pulse and impart greater power to it, by first of all increasing the energy of the cerebro spinal system, are the great essentials in the treatment of all fevers but the inflammatory.

The author of the article on arsenic already quoted speaks of its effects on the kidneys as preceding its effects on innervation and respiration. My own observations, however, would lead me to believe that the nervous system is first affected by arsenic in the circulation, next the pulmonary and cutaneous respiratory functions, and then the function of urinification, simply because the work of the lungs is according to the power of the nervous system, and the performances of the kidneys are, in great part, merely indices of the state of the respiration, that is to say while there is no organic lesion.

and the sulphates, which have, like sulphate of zinc or copper, no disposition to excite expulsory actions when given in moderate doses, may be made useful allies for the temporary arrest of intermittent. The persesquinitrate of iron most probably acts upon the same principle; though we must not lose sight of the importance of the iron to a patient who has become pallid from the long continuance of ague.

If it be true, as has been proved by chemical analysis, that the circulating earthy phosphates are increased in amount when the system is brought moderately under the influence of mercurials;* and if we find, as is really the case, that periodic fever ceases for a time when mercurial action has been fairly established, and proved by free salivary secretion, we may safely, I think, conclude that the cessation of the fever is due to the effects of the increased amount of phosphates absorbed from the osseous textures and set free for the repair of the nervous system. It may be objected to this view that the well known action of mercury as a stimulant is sufficient to account for the cessation of the febrile symptoms, as we know that stimulants in general are antiperiodics, and are powerful in proportion to the permanency of their effect on the constitution; but it must be remembered that the cases in which mercury most decidedly shows its stimulating effects by quickening the pulse and respiration, and rendering the person nervously excitable, are the very cases where its febrifuge effects are least observable. In these cases, moreover, we have great soreness and swelling of the mouth with little or perhaps no secretion, even when the mercury has been pushed to the limits of safety, which would lead us to conclude that the elements necessary for the salivary secretion, viz., the phosphates of soda, lime, magnesia, and phosphate of the peroxide of iron, with chlo-

* By salivation, the phosphates are nearly doubled in the blood, and are more than doubled in the urine; while the fibrin is decreased one-third, and the albumen one-tenth.

ride of sodium and sulphate of soda, are not at the moment in the blood, and, therefore, not ready for the repair or nutrition of the nervous system, which is in great part composed of them. We find also that when the morbid stimulating effects of mercury are at their height and no impression made upon the fever, we can lower the pulse, produce free salivary secretion, and dissipate the fever merely by the direct exhibition of these phosphates in the form of bone powder; and this is, I think, a tolerably good proof that the febrifuge effect of the mercurials is not occasioned by their stimulating action, but is owing almost entirely to the phosphates which they cause to be absorbed from those great phosphatic store-houses, the bones, and brought into circulation for the benefit of the cerebro spinal system.

If we admit, then, that the leading effect of alterative doses of mercurials in fever is the bringing of phosphates into circulation for the nutrition of the nervous system, so as to impart to it greater power of preventing the febrile oxidizement of the rest of the body, we may, perhaps, independently of all practical proof, be led to believe that in many cases of fever, where mercury is not specially called for as a stimulant to the dormant energies of the cells, the direct exhibition of the phosphates alone would be sufficient to serve our purpose of restoring the patient to health. But, if in addition to these hopeful anticipations afforded by theoretic induction, we find that the phosphates, as they exist in the bones, have actually the power of permanently arresting fever, when introduced into the system in a form to be retained in it, we must surely yield to the inductive reasoning and the practical proof together and acknowledge that earthy phosphates are really of no mean value in intermittent fever.

Though we can always bring phosphates into circulation by the action of mercury, and thus bring them to bear on the nervous system so as to eradicate an ordinary periodic fever, we cannot keep these phosphates for any time in the blood, as they are daily passed off by the secreting organs, and the body

may be thus in time almost cleared of available earthy salts. Intermittent cured by mercury is almost sure to return in an aggravated form, because though we have for a time enriched the blood with phosphates they are soon excreted or sink again into the osseous textures, from which they were taken up by the absorbents, and the blood is more impoverished than it was before we undertook the treatment of the case. This objection cannot apply to the direct administration of earthy phosphates, for if the blood be ill supplied with them, owing to their being deficient in the diet, or passed off in too great quantity by the kidneys, or used up for the requirements of pregnancy, or lost by adding to the density of the skeleton as we advance in life, there is every inducement to urge us to give the phosphates themselves in accordance with the exigencies and in obedience to the requirements of the body. By bringing the phosphates into circulation by absorbent medicines, we certainly effect a great and immediate temporary good; but the benefit is soon followed in many cases, among the poor at least, by a serious and lasting injury to the constitution, which might be avoided by simply prescribing the phosphates themselves.

In giving phosphates, our first care must be to stimulate the respiratory surfaces so that there may be no risk of their being passed off as fast as they are taken up. We can but seldom remove our patients to the stimulating air of the sea side, so that we must find a medicine which will have a somewhat similar effect on the pulmonary mucous membrane. Sugar has recently been proposed for this purpose by a French physician, whose views must be already known to the profession by the notice of them which appeared in a recent No. of the Lancet (about June or July 1850, I think); but it does not seem well adapted for this country where we find saccharine very often alternating with neutral or slightly acid phosphatic urine.* Sulphur will here be found to serve the purpose, if given in small doses to ensure its entrance into the

* But not with Ammoniaco Magnesian phosphate.

system and the conversion of part of it into sulphurous acid in the lungs. It will be found of great service in small frequently repeated doses in *beriberi* cases, with phosphatic and alkaline urine, as it acts more efficiently than the largest doses of mineral acids. Indeed it acts so efficiently in such cases that we must ascribe its virtues more to some effect it has upon the functions of certain organs, than to any neutralizing power the small amount of acid formed from it can exert upon the urine. In these cases the peculiar blueness of the gums and inside of the cheeks is but an indication of a somewhat similar state of the bronchial lining membrane, as is confirmed by the feebleness of the vesicular murmur, and we have to find a medicine which will ameliorate these symptoms before we can hope to prevent the kidneys passing excess of phosphates in true *beriberi* cases.

As to the best mode of administering phosphates in intermittent fever, I cannot do better than introduce in this place, with a few additions, the directions written by me some time ago and issued by the Medical Board, and then conclude with a few cases illustrative of the treatment; premising, however, that though I recommend phosphate of lime and sulphur only in fever cases where the tongue is red edged, still that cases occur with pale edged tongues which are benefited by this remedy, when they have continued to recur in spite of bark and quinine. Cases running into *beriberi* are instances of the kind. Where, however, the paleness of the tongue is accompanied with splenic enlargement this remedy will not be found of much service, unless given alternately with quinine.

1. Phosphate of lime (impure) is most easily prepared by calcining sheep's bones. It is then to be finely powdered and well mixed with one-sixth of its weight of sublimed sulphur. Washed sulphur is to be preferred.

2. This compound powder of phosphate of lime with sulphur may be given with advantage in most cases of well marked intermittent fever, and in any of its stages. If employed, it must constitute the basis of the treatment, and other medicines, when required, must be held as auxiliary to it.

3. When this powder is to be employed, emetics may be considered unnecessary, unless the tongue be unusually foul, or the conjunctiva decidedly yellow. A purgative of compound powder of jalap with calomel, or a pill of croton powder with calomel, should be given at the commencement of the treatment, unless the fever be present, or be expected to come on within a few hours, at the time the patient is first seen.

4. If the fever be expected soon, or be present in any stage when the patient is first seen, a dose of the powder ought to be given at once and the exhibition of the purgative delayed till next morning.

5. The dose of phosphate of lime with sulphur is two drachms, thrice a day, in treacle or sugar and water. It should be continued steadily till the usual period for the expected fever has passed without any febrile return. Four drachms may be given at the first if the patient be very weak when first seen, or if there be any urgent necessity for at once cutting short the fever. Half a drachm thrice a day, is considered a suitable dose between the ages of two and five; one drachm from five to twelve, and after that age two drachms, thrice a day.

Patients under 20 years of age recover more slowly on this medicine than those above 20.

6. This remedy acts moderately on the bowels, usually producing two stools per day; sometimes it seems to be very rapidly absorbed, and therefore acts very slightly on the bowels. It removes thirst and improves the secretions. The skin becomes steadily moist under its influence; and if the powder be given at the commencement of an attack of fever, headache is either altogether prevented or materially diminished, and perspiration is usually rapidly induced and pro-

moted by it. The patient also appears to lose the sense of prostration sooner on this than any other remedy.

7. The bowels are usually kept sufficiently open by this combination; but, if the tongue be foul after three days treatment, or the skin be too warm at all times and clammy, a smart purgative should be given, or muriatic or nitro-muriatic acid (in doses of from one drachm to two drachms, in five or six ounces of water) should be given with the morning dose of the powder, and repeated as often as the foulness of the tongue may indicate the need of it.

8. When the conjunctiva is yellowish, or there are any signs of congestion remaining between the attacks of fever, two or more grains of calomel should be given with each dose of the powder. Or a smart dose of calomel may be given with the last dose of phosphate of lime and sulphur at night, and a nitro muriatic acid draught with the first dose in the morning. Mineral acids, however, cannot always be procured; but any common purgative does sufficiently well in the morning, if given alone. When the tongue begins to clean, infusion of gentian or infusion of chereyta is of service as an auxiliary to the powder; and exercise should be permitted or recommended as early as possible.

9. Leeching and blistering must be used, of course, as they may be deemed necessary; but in my experience in the use of this remedy unusually few occasions have been found for the employment of leeches and blisters when this powder has been employed early and given steadily. When, however, any symptoms arise, to indicate the need of depletion in the course of intermittent, in which this powder may constitute the basis of the treatment, the patient, if a native, will be found to bear leeching or venesection better than is usual with natives, especially with Hindoos.

10. The cases most suitable for the remedy are well marked intermittents, where the tongue is either clean or very red, or foul with red edges, or raw and patchy, or has the pale boiled appearance usual in *beriberi*. The more

indeed, the tongue, pulse, feelings, and general appearance, indicate the transition of the case from intermittent to *beriberi*, the more necessity there is for giving this powder and persevering in its administration, as it seems to be of peculiar value in cases of long standing intermittents, threatening to terminate in paralytic, or dropsical appearances.

11. The only symptoms which contra-indicate the employment of phosphate of lime with sulphur are the changing of the tongue from the characters above enumerated, its acquiring a white shaggy or villous coating, and the fever appearing to be taking on the remittent form. Under these circumstances the powder ought to be at once intermitted, and quinine given till the tongue lose the shaggy coating.

12. As a general rule the patient ought to be restricted to low diet till he be fairly rid of the fever. When the pulse is feeble, between the attacks, chicken or mutton broth, or beef soup, ought to be allowed. Onions ought to be in the soup as their juice assists in the solution of the medicine, The use of tamarinds, as well as all preparations of tartrates, must be strictly forbidden, as they render the phosphate of lime insoluble. Garlic ought to be prohibited as it invariably keeps up febrile action.

Cases of Intermittent Fever treated with Phosphate of Lime and Sulphur.

INTERMITTENT FEVER.

P. Kristnamah, aged 24, 2d Dresser, No. 199.

18*th April*, 7 A. M.—Shivered about 6 o'clock yesterday morning, and had severe fever with frontal headache nearly all day. Took a croton and calomel pill in the morning; was well purged. Took phosphate of lime 1½ drs. with sulphur ½ dr. at 4 P. M., and began to perspire copiously at 5 P. M. At 7 P. M. the pulse was 100. Took another dose

of phosphate at 8 P. M., and perspired freely during the night. Pulse now 100 soft, full, much soft cough with copious expectoration, skin cool and wet, bowels open, no pain in any place but the forehead.

Calomel 4 grs. now.
Superphosphate of lime 3 drs. at 11 A. M.

19*th*.—Was obliged to attend to his duties yesterday, notwithstanding his debility, so that his improvement has been somewhat retarded; pulse 90 soft, looks clear, skin cool, cough easy and expectoration of clear mucus copious, tongue nearly normal, bowels open.

No medicine.
Chicken broth.

20*th*.—Is much disturbed by the constant moist cough which is also keeping up the pulse. Bowels open, no return of fever.

Cough Mixture.

21*st*.—Less cough, no fever, bowels open, pulse and tongue nearly normal.

Cont. mixture.
Phosph. of lime 50 grs.
Sulphur 10 grs. M. now.

22*d*.—No fever, pulse 80. No complaint but of cough.

24*th*.—No return of fever, cough nearly gone.—Discharged.

Tertian Intermittent Fever.

Duniah, Private, aged 20, No. 1674.

10*th March*, 1849.—Admitted. Fever comes on every 3d day about 2 P. M. Complains now of pain in head and limbs. Tongue rough, red-edged, bowels slow, had fever yesterday.

Compound powder of jalap 1 dr.
Calomel 4 gr. M. now.

11*th*.—Well purged, free from uneasiness, pulse 64, tongue red-edged.—Nothing.

12*th*.—Fever came on at 4 P. M. yesterday. He shivered and became hot; but did not perspire. Bowels freely moved, pulse 64, soft, tongue foul, very red at edges.

Phosph. of lime 50 grs.
Sulphur 10 grs.
Calomel 4 grs. M.—each night.

14*th*.—No return of fever, health good.—Discharged.

Quotidian Intermittent Fever.

Ramdeen, Private, aged 40, No. 508.

31*st July*, 1849, 7 P. M.—Admitted, had a slight attack of fever 5 days ago, but did not come to hospital. Shivered yesterday at 2 P. M., and had smart fever till the middle of the night, when it went off without perspiration; but the bowels moved twice spontaneously instead. Tongue foul, yellow with red-edges, pulse 100 soft full, skin dry and too warm. No headache, but has pains all over body, especially in loins.

Common emetic, now.
Phosph. of lime 3 drs.
Sulphur 1 dr. M. at 10 A. M.

1*st August*.—Had free vomiting and purging. No fever yesterday, but thinks he had fever about 11 P. M. Tongue nearly clean, red at tip, but pale at edges. Pulse 80, rather weak, skin cool but too dry. No pain any where. Repeat phosphate of lime and sulphur.

2*d*.—Had fever preceded by smart shivering for an hour yesterday afternoon. Tongue now thickly coated, with narrow red line at edges. Bowels moved three times yesterday, pulse 90, soft, full, skin too warm and clammy.

Compound powder of jalap 1 dr.
Calomel 5 grs. M. now.

3d.—No more fever, bowels moved four times. Tongue still foul and red-edged, pulse 80, soft, skin cool. Repeat phosphate of lime and sulphur now.

4th.—Improving, no return of fever. Continued phosph. and sulphur.

5th.—No return of fever. Pulse and skin normal, though he has been out of hospital, washing and eating at his own pleasure. Tongue still white and rather red-edged. Bowels open.

Phosph. of lime 1 dr.
Carbonate of potass ½ dr. M. now.

6th.—Tongue clean, health good.—Discharged.

Intermittent Fever.

Shaik Booran, Private, aged 30, General No. 635.

21*st April* 1849, 7 A. M.—Admitted last night with fever which commenced with shivering at 11 A. M., yesterday, and went off without distinct perspiration at 4 P. M. His tongue was dry and he had much thirst. Had an emetic last night which acted both ways freely. Pulse now 100, soft, weak, skin cool. Respirations 40 per minute, irregular, partly abdominal; chest sounds well and the respiratory murmur is distinct throughout, except at lower part of right lung where it is rather dull. Is very weak but free from pain.

Phosph. of lime 2 drs.
Sulphur ½ dr. M.

21*st.*—Complains of intense headache and seems almost beside himself from it, pulse 100 weak, tongue dry, bowels not moved since yesterday.

Compound powder of jalap 1 dr.
Calomel gr. 4 M. now.
Blister to each side of head.

23*d.*—Purgative and blister acted well; pulse 80 of better strength. No headache, tongue inclined to be dry.

Phosph. of lime 50 grs.
Sulphur 10 grs. M. twice a day.

24*th.*—Looks well, though the tongue is again dry and now brown, pulse 80 soft, blisters open, bowels very slow. Repeat the purgative powder. Continue the fever powders.

25*th.*—Improving. Continue phosph. of lime and sulphur.

28*th.*—Gaining strength. Nothing.

3*d May.*—Well in every respect though the tongue is still inclined to be dry. The skin and bowels secrete well. Repeated the last powders.

11*th.*—Strong and healthy.—Discharged.

This man had the tongue peculiar to sepoys who take too much garlic.

Intermittent Fever.

Mootoosawmy, Private, aged 30, General No. 715.

31*st July*, 1849, 7 A. M.—Had fever yesterday evening preceded by shivering at 4 P. M. Began to perspire at 11 P. M. Pulse now 80, soft weak, skin cool, looks heavy, eyes turbid, tongue coated yellow, red-edged, bowels moved once this morning. Has not taken medicine.

Common emetic now.
Phosph. of lime, 3 drs.
Sulphur 1 dr. M. at 10 A. M.

1*st August.*—Had free vomiting and purging, no return of fever, tongue clean, eyes clear, pulse 80, soft.

Phosph. of lime 3 drs.

2*d.*—No return of fever, pulse, tongue, skin and bowels normal.

3*d*.—No return of fever; well, but says he is rather weak, pulse 68.—Nothing.

4*th*.—Pulse very slow, quite well.—Discharged.

The five following cases were men who had been 10 days on duty at Seetabuldee, and were taken with fever from three to five days after their return. These cases were well marked.

INTERMITTENT FEVER.

Private, Govindoo, aged 28, General No. 1075.

Admitted 2d November, 8 A. M. with intermittent fever.

4*th*.—Has had one croton pill and several drachms of fever powder since admission, had fever yesterday afternoon.

Continue fever powder, 2 drs., twice a day.

5*th*, 2 P. M.—Tongue red-edged, pulse 120. Is in copious hot sweat, no pains, has been dieting imprudently.

Continue phosph. of lime and sulphur.

6*th*.—Looks well; tongue and pulse normal, except a small red triangle at tip of tongue, bowels moved twice yesterday evening and once this morning.

Continue phosph. of lime and sulphur, twice a day.

7*th*.—No fever yesterday, tongue red-edged and at tip, pulse normal, skin cool, bowels moved this morning.

Continue fever powder.

9*th*.—No fever since last report, tongue still a little red at tip, bowels open, pulse and skin normal.

Continue fever powder.

10*th*.—Quite well, though there is still slight redness at tip of tongue.—Discharged.

QUOTIDIAN INTERMITTENT FEVER.

Private, Soobroyen, aged 30, General No. 1112.

5*th November*, 1849.—Admitted at 2 P. M., in the sweating stage of intermittent: was discharged this morning, having been two days detained in hospital without any fever coming on. Began to shiver at 10 A. M., tongue now very red-edged and foul at centre, bowels slow.

Phosph. of lime 3 drs.
Sulphur ½ dr. M. now.

6*th*.—Tongue white and red spotted in centre, very red at edges, pulse and skin normal; bowels moved twice yesterday and to-day once.

Continue phosph. of lime with sulphur, 2 drs. twice a day.

1½ P. M.—Fever on now, pulse 100, soft full, tongue red, clean.

7*th*.—Tongue pale, rather red at tip, pulse normal, bowels moved once this morning.

Continue fever powder.

8*th*.—No fever yesterday, pulse, tongue and skin normal, bowels open to-day.

Continue fever powder.

9*th*.—No fever, tongue, pulse and skin normal. Discharged.

QUOTIDIAN INTERMITTENT FEVER.

Private, Shaik Hoossain, aged 30, General No. 1196.

5*th November*, 1849, 2 P. M.—Admitted in the sweating stage, began to shiver at 9 A. M. Tongue very red-edged, foul in centre, has severe pain in back and head.

Phosph. of lime 3 drs.
Sulphur ½ dr. M. now.

6*th*.—Pulse of normal number but too full, coughs a little,

skin temperate, tongue very red-edged and foul at centre, no stool.

Sulphate of magnesia 1 oz. now.
Continue phosph. of lime.

1½ P. M.—No fever.

7*th.*—Tongue slightly red at tip, bowels freely moved, skin rather warm and dry.

Continue fever powder.

6 P. M. Says he had shivering at 10 A. M., pulse now 100, skin too warm.

8*th.*—Tongue still too red at edges, clean; pulse and skin normal; bowels moved last night.

Continue fever powder.

9*th.*—No fever for two days, tongue a little too red, bowels open, pulse and skin normal.

Continue fever powder.

10*th.*—No fever, pulse and skin normal, tongue still a little red at edges, bowels open.

Continue fever powder.

11*th.*—Pulse slow, skin too warm and dry, tongue foul rather red at tip and edges, looks heavy.

Pulv. jalap compound 1 dr.
Calomel 4 grs. M. now.

13*th.*—Was well purged, tongue, pulse and skin normal, no return of fever. Discharged.

Quotidian Intermittent Fever.

Private, Syed Hoossain, aged 28, General No. 1833.

5*th November*, 2 P. M.—Admitted in the hot stage, began to shiver at 11 A. M. Tongue very red and foul at centre, pulse 120, no headache.

Phosph. of lime 3 drs.
Sulphur ½ dr. M. now.

6*th.*—Tongue very red-edged and foul, pulse normal, skin too warm and clammy, no stool.

Sulphate of magnesia ½ oz. now.
Continue phosph. of lime.

7*th.*—Says he had fever last night at 12, but there is no trace of it, had one ounce of fever powder yesterday, tongue has still very slight redness at tip, pulse 80 soft, skin cool and dry, bowels moved four times.

Continue phosphate of lime.

8*th.*—No fever yesterday, though he had a little heat of skin in the afternoon which he called fever, though not preceded by shivering. Tongue very slightly red-edged and clean now at centre, bowels open to-day, pulse normal, slight cough.

Continue fever powder.

9*th.*—Skin was rather warm last night but he had no fit of fever, tongue pale, pulse and skin normal.

Continue fever powder.
To take exercise morning and evening.

10*th.*—No fever, tongue free from redness and clean in centre, pulse and skin normal.—Discharged.

Quotidian Intermittent Fever.

Private Chickanah, aged 33, General No. 1538.

5*th November.*—Admitted 8 A. M., was detained yesterday and had fever, commencing with shivering at 8 A. M.; had croton pill, and phosphate of lime, 4 drs.

2 P. M.—Shivering came on at 10 A. M., to-day but he must have had very slight fever as the skin is now cool and dry. Had a dose of phosphate of lime with sulphur this morning, tongue now clean but rather red, pulse 88 of good strength, bowels moved this morning.

6*th* A. M.—Has just had a shiver, and vomited a large quantity of bile as he was becoming warm, pulse 140 soft, weak, skin moderately warm, no pain any where. To drink warm water and to have sulphate of magnesia one ounce when the sickness goes off.

Continue phosph. of lime.

1½ P. M.—Fever still on, tongue paler, pulse 120, skin warm and moist.

7*th*.—Tongue still red at edges, is free from fever, skin cool and moist, pulse normal, bowels moved freely.

Continue fever paste.

9*th*.—No return of fever, tongue still rather red-edged and marked by the teeth.

Continue fever paste.

10*th*.—Pulse rather quick, tongue a little red-edged, no return of fever, complains of weakness.

Continue fever powder; mutton diet.

11*th*.—Bowels freely open, skin moist, warm, tongue nearly normal, pulse normal, says he had fever yesterday with a cold fit; but the dresser saw him and there was no cold fit.

12*th*.—Quite well.—Discharged.

INTERMITTENT QUOTIDIAN FEVER, RUNNING INTO BERIBERI.

Seevacharum, Private, aged 25, General No. 1624.

3*d May*, 1849, 7 A. M.—Admitted. Is ghastly yellow, eyes unnaturally large, rather yellow, tongue very pale, slightly furred, skin cool, much thirst. Pulse 140 weak, incessant dry cough, coughs up clear mucus copiously at times, much giddiness, no pain nor uneasiness any where; walks pretty steadily, no numbness nor œdema of hands and feet, chest yields clear sounds; but the heart's action is very feeble,

though its bulk is normal and well defined to percussion. Bowels move regularly, passes plenty of urine, stools and urine not seen. Has had fever several days, it comes on every day about 2 P. M., and he perspires copiously about 8 P. M. Has very much the appearance of a beriberi patient, has never been in hospital.

Blister to each temple.
Phosph. of lime 2 drs.
Sulphur 40 gr. M.

to be taken now; mutton, dhal and coarse wheat bread for diet; rice and tamarind to be forbidden.

4*th*.—Pulse 100 soft, weak, tongue now red and moist, no stool, urine copious, red; blisters acted well, no giddiness now, no fever yesterday, skin only ordinarily moist last night, eyes still rather yellow but countenance improved.

Continue the fever powder and diet as above; castor oil one oz. now, that the character of the excretions may be ascertained.

5*th*.—Stools of good colour and natural odour, pulse 100 soft, tongue paler, urine scanty red.—Continue.

6*th*.—Passed about a quart of dark red urine in last 24 hours, pulse 82 soft, no stool, no return of fever, well in every respect.

Comp. p. of jalap one drachm.
Calomel 5 grs. M.
to be taken now.
Omit phosph. of lime and sulphur.

7*th*.—Bowels moved freely, urine copious but not seen, eyes nearly natural, pulse 68 soft; lungs seem a little congested but there is no cough.—Nothing.

8*th*.—Eyes clear, white, bowels moved three times; well in every respect.

11*th*.—Strong and well.—Discharged.

Intermittent Fever with Beriberi.

Condasawmy, aged 26, Genl. No. 1449.

27*th March*, 1849.—Has been in hospital, since June 1845, four times with intermittent fever, and twice with beriberi, or in the following order, twice with intermittent fever, twice beriberi, and then twice with intermittent fever. This time the beriberic symptoms predominate; complains of fever and giddiness with great debility but says nothing of numbness, skin now cool. Face peculiarly sallow and corpse like, eyes yellowish, dull and fishy, pulse 130 soft, tongue pale, white and clean as if boiled, bowels slow, no signs of effusion any where; lives mostly on rice.

Emetic of Ipecacuan.

28*th*.—No change, bowels not moved.

Pil. croton and calomel, No. 2.

29*th*.—Pills operated very slightly, pulse 130, skin cool, tongue pale white, slight effusion in abdomen.

R. Pil. hydrarg. 10 grs. quinine 5 grs. thrice a day, with 5 drops of tincture of iodine, and 5 grs. of hydriodate of potass in three oz. of water thrice a day; diet to consist of dhal, mutton and coarse wheat bread.

30*th*.—Pulse 100, bowels open, urine very scanty and almost colorless, not above 8 oz. in 24 hours. Other appearances as yesterday.

Continue the medicines.

3*d April*.—Pulse 130, evidently from mercurial excitement, no effusion any where, tongue not so pale, but now very tremulous, face swelled from blue pill, but no ptyalism.

Omit all the medicines.

Sulphur 1 dr. twice a day.

4*th*.—Left side of face still swelled, but no increase of secretion from mouth, tongue still pale and tremulous.

Continue sulphur with phosph. of lime 1½ drs. at night.

5*th*.—Tongue becoming decidedly pink at sides, pulse still 120.

Continue sulphur and phosph. of lime.

6*th*.—Is extremely active and very hungry to-day, tongue losing the boiled appearance and showing clear pink at sides, pulse 100 regular, bowels moved once since last dose; countenance losing the cadaverous appearance, swelling of face nearly gone, secretion from mouth free.

Omit sulphur.

Continue phosph. of lime.

8*th*.—Tongue red all over and streaked with a little dark fur but still tremulous, pulse 100 full, regular; countenance much improved, walks well.

Continue phosph. of lime, with animal and pea diet.

9*th*.—Tremulousness of tongue nearly gone, pulse 94 full, regular; stools light colored, urine neutral but free from phosphates.—Continue medicine.

10*th*.—Three watery stools this morning, no pain with them, urine neutral, tongue, skin and pulse as above.—Continue medicine.

12*th*.—Tongue paler, pulse 90.—Continue medicine.

13*th*.—Tongue of better color, less tremulous, bowels regular, pulse 80 of good strength, urine neutral.

Continue phosph. of lime night and morning.

15*th*.—Tongue has been pale during last 2 days, and it appears he has returned to rice diet notwithstanding daily cautions, pulse 100 soft weak; tongue tremulous; looks ill.

To continue phosph. of lime, to avoid rice, and adhere to dhal, coarse bread, mutton or eggs.

16*th*.—Tongue not tremulous and has more color, looks better, urine clear, but slightly alkaline to test paper though only recently passed into a glass vessel, pulse 80 soft, regular.—Continue.

17*th*.—Tongue improving, pulse 80 full, urine very slightly alkaline.

18*th.*—Pulse and tongue nearly normal, stools healthy, urine still slightly alkaline, as he will persist in using tamarind in his food, walks well, eats and sleeps well. Has gained a good deal of flesh since admission, wishes to go to duty.—Discharged.

7*th January*, 1851.—This patient has not been again in Hospital.

The next two cases were treated and reported by Assist. Apothecary Lima, doing duty 23d Regiment, W. L. I.

Quotidian Intermittent Fever.

Yenketkistnaraj, Private, 8th Company, No. 1627.

2*nd January*, 1851.—Admitted 6 A. M., complaining of fever which comes on daily, sometimes at noon and sometimes towards evening and at night, preceded by rigors for the last five or six days; last attack 6 P. M. yesterday; skin at present warm but moist, tongue coated at the base and red at edges, bowels said to be regular, has not been in hospital for the last year.

Phosph. of lime with sulphur 2 drs. thrice a day.

3*d.*—Return of fever at 7 P. M. yesterday, preceded by rigors, perspired and became cool at 11 P. M., bowels have not been opened yesterday.

Pil. calomel and croton, 6 grs.
Continue phosph.

4*th.*—Bowels have been freely opened, fever returned but slightly at 9 P. M., is at present cool, tongue cleaning.

Continue phosph.

5*th.*—No return of fever yesterday, no complaint.

Continue phosph.

6*th.*—Fever returned at 8 P. M., preceded by rigors and

left him during the night, has perspired freely, bowels not opened yesterday.

Pil. calomel and croton 6 grs.
Continue phosph.

7*th.*—Fever returned preceded by slight rigors at 9 P. M. last night, perspired at 12 and became cool afterwards, bowels have been freely opened.

Continue phosph.

8*th.*—Fever as usual but later at night, states that he felt chilly, which was followed by heat of skin about 11 last night, is at present perfectly cool, pulse moderate, tongue cleaning.

Continue phosph.

9*th.*—No return of fever, skin cool and moist, tongue cleaning, bowels regular.

Continue medicine.

11*th.*—Convalescent.

12*th.*—Discharged.

Quotidian Intermittent Fever.

Ramasawmy, Private, 3d Company, No. 1166.

4*th January* 1851.—Admitted 6 P. M., complaining of the usual symptoms of fever coming on daily for the last five days. Paroxysm generally about 6 A. M., preceded by rigors and leaving him about 12 at night, says that he does not perspire, skin at present warm and rather dry, pulse excited, tongue coated at the base and red at the tip, bowels confined; has not been into hospital for the last year.

Phosph. of lime and sulph. 2 drs. thrice a day.

5*th.*—Has had a return of fever as usual preceded by rigors at 6 P. M., yesterday, perspired during the night, is at present cool and moist, pulse moderate, tongue the same, bowels once opened.

Continue phosph.

6*th.*—Return of fever at 9 P. M., felt a little chilly but no rigors, bowels have been opened, tongue the same, the fever did not last for more than two hours.

Continue phosph.

7*th.*—Says that he felt a little chilly and warmth of skin at 11 last night, and perspired afterwards, tongue cleaning, bowels regular.

Continue phosph.

8*th.*—No return of fever.

Continue medicine.

9*th.*—No complaint, tongue still loaded at the base but cleaning at the forepart.

Continue remed.

10*th.*—No return of fever, tongue natural, bowels regular.

11*th.*—No complaint.

12*th.*—Discharged.

A. BLACKLOCK,
Assistant Surgeon,
23d Regt. W. L. I.

KAMPTEE,
9th February, 1851.

NOTE.—In the early part of 1850, Assistant Surgeon A. Blacklock, embodied some remarks on the treatment of fever with phosphate of lime and sulphur in his annual report of the 23d W. L. I. then under his charge, and the Medical Board being of opinion that they were possessed of much interest, and moreover that the subject was one of great importance, requested Mr. Blacklock to give his views in detail of the mode of action of this medicine.

At the same time under instructions from the Board, a Committee of medical officers was convened at Kamptee, to institute a careful practical investigation, as to the therapeutic properties of this remedy (phosphate of lime with sulphur) at the several hospitals under the immediate guidance and direction of the Superintending surgeon.

The foregoing most valuable and interesting paper has been the result; the reasoning is highly ingenious, but the correctness of the data in some particulars would appear to be doubtful; example at page 13, speaking of the greater prevalence of fever amongst the Hindoo portion of the population, owing to the different nature of their food, containing less of phosphates, the returns of the Native Army for a period of three years ending 1852, would show that the ratio of fever, or the liability to that disease is almost the same in both bodies of men; the ratio per 1000 of strength being 326· amongst the Hindoos, and 322· amongst the Mussulman in the infantry, while in the native cavalry the ratio for the same period has been, Hindoos 300· and Mahomedans 344·.

The cases that follow give also somewhat equivocal results; but there can be no question however that the fever powder will be very useful in the milder forms of distinct intermittent fever, such cases as those detailed by surgeon Drever, and it may thus enable us to husband our stock of quinine and bark, where fever is periodically endemic as at Seetabuldee, and in the out-posts of the northern division, &c.

The Medical Board are taking steps to give this remedy a more extensive trial, conceiving that its use should be encouraged at any station where it may be found to be advantageous; it is simple in its nature, cheap and easily procurable.

A. L. *Secy. Med. Board.*

CASES TREATED BY ASSISTANT SURGEON DR. ANDERSON.

FEBRIS QUOTIDIANA INTERMITTENS.

Abbas Ally, Driver, D Company, 23 years in the Service.

Admitted at 10 o'clock A. M. on the 7th November 1849, when he stated that he had a paroxysm of fever on the 5th. It began at 3 P. M., and recurred the following day at

the same time. The last paroxysm passed through the stages of cold, heat and sweating; during the first he says there was merely cold, tongue foul, bowels confined.

Can assign no cause for the attack.

Pulv. jalapæ com. 1 dr. statim.

Vespere, frequently moved by the jalap powder which he says has weakened him much. There has been slight pyrexia since 3 o'clock.

Phosph. of lime with sulphur 2 drs.

To be taken immediately.

8*th*.—Perspired slightly after the powder, when is not exactly known, three stools in the night, apyrexia this morning.

The fever powder to be repeated now, and again at 5 P. M.

9*th*.—The powders caused no sensible action, but there has been no return of fever, bowels open.

Omit powder.

11*th*.—Has remained well.—To duty.

FEBRIS QUOTIDIANA INTERMITTENS.

Chellivan, Driver, D Company, aged 25, General No. 1176, 7 years in the service.

This man is of spare habit of body. He was admitted into hospital on the 16th April 1850, but he presented himself the previous morning when he was detained under observation, as he said he did not feel well, had no appetite, and his bowels had not been moved for 48 hours, he had then no heat of surface; took calomel 5 grs. and pulv. jalapæ com. 1 dr. which purged him smartly; about an hour afterwards he became restless, his skin was hot and he complained of thirst. These symptoms remained till 3 P. M. when he began to perspire. This morning he states that he feels weak and is without appetite, tongue is coated with a whitish fur, the surface is cool.

Mist. salina simp. 1½ oz.

Every 3d hour until the bowels act.

17*th*.—Fever returned at 11 o'clock A. M. yesterday, there was first a chill, then heat of skin, thirst and general uneasiness, no headache; began to perspire sometime during the night, bowels were 4 or 5 times moved by the saline mixture. This morning apyrexia, but there is a white fur on the tongue, he is still without appetite and he complains of thirst and general debility.

2 drs. of phosphate of lime with sulphur thrice daily.

6 P. M.—Has taken 3 powders, but fever returned at ½ past 11 o'clock, there was no cold stage; but there were heat of skin, thirst, vertigo and a general feeling of soreness and debility; bowels scantily moved once, tongue is less furred, began to perspire slightly an hour ago, and he is now bathed in perspiration. Pulse 102.

Continue powder.

18*th*.—A good night, skin cool, pulse of natural frequency, tongue is white with a red-edge, no stool.

Continue powder thrice a day.

Vespere, fever returned at 2 P. M., skin remains hot; but on the forehead there is a slight moisture; vertigo, debility and thirst are what he complains of; four pale yellow feculent stools, there was no rigor, tongue furred.—Continue.

19*th*.—Heat of surface left at 8 o'clock P. M.; a good night, tongue is nearly clean, complains of nausea but there is perfect apyrexia, one scanty stool evidently containing sulphur.

Continue powder thrice daily.

Vespere, heat and dryness of surface; restlessness, thirst and vertigo since 2 o'clock P. M. Complains of a sense of internal heat and of griping, no tenesmus, two loose feculent stools of the colour of sulphur and containing two clots of blood. Pulse upwards of 100, skin hot and dry.—Continue.

20*th*.—Fever did not leave till late in the night, two stools, feculence of a pale yellow colour, apyrexia, no abdominal uneasiness, tongue is nearly clean and moist.—Continue.

Diluted nitric acid as a drink, 2 drs. to 24 ozs. of water in the day.

Vespere, fever since 4 P. M., symptoms much as in former attacks. It continues, no return of abdominal uneasiness, tongue is pretty clean, he complains of thirst, two consistent stools coloured by the sulphur.—Continue.

21*st*.—Fever left during the night, to-day apyrexia, tongue clean and moist, one feculent stool.

Continue powders and the acid drink.

22*d*.—Fever returned at 8 P. M. no cold stage, but there was a distinct hot one followed by sweating; two feculent stools, apyrexia this morning.

Continue powders and drink.

23*d*.—Fever returned as before at ½ past 8 o'clock, there were present heat of skin, vertigo, nausea, thirst and great muscular debility, feverish symptoms left during the night about 11 o'clock it is believed, three stools; there is a brown fur on the middle of the tongue, edges clean, pulse 78.

Pulv. Cinchonæ 2 drs. every 2d hour to the 4th time.

24*th*.—No return of fever.—Nil.

25*th*.—Convalesces.

1*st May*.—To duty.

Febris Quotidiana Intermittens.

Caulapen, Driver, D Company, aged 37, 14 years in the service, No. 722.

Admitted on the evening of the 19th May, 1850, with slight febrile symptoms which left at 8 P. M. Took on admission calomel, pulv. antim. comp. of each 5 grains, and 2 oz. of simple saline mixture, (a weak solution of salts and tartar emetic) by which his bowels were moved five times.

20*th*.—Apyrexia; by account on the 17th had a chilly feeling which returned yesterday at 3 P. M., and was succeeded by heat, &c.

Mist. salina simp. 2 oz.
ter in die.

21*st*.—Had a return of fever yesterday. It began by a chill at 2 P. M. afterwards there were heat and dryness of skin and headache. The attack terminated in sweating at 7 o'clock P. M., took three doses of saline mixture by which his bowels were moved twice. This morning perfect apyrexia, tongue coated with a whitish fur.—Nil.

Vespere, fever returned at 3 P. M. commencing with a slight chilly feeling; now at 6 P. M. his skin is cool but moist, neither headache nor vertigo were present, one stool; the attack has been slight.

2 drs. of the phosphate of lime with sulphur, immediately.

22*d*.—Apyrexia, weak and without appetite, bowels not again moved, tongue moist and furred.

Repeat 2 drs. of the powder thrice daily.

Vespere, slight fever from noon till ½ past 2 o'clock P. M., now apyrexia, one stool.

Continue powder.

23*d*.—No fever.—Continue.

24*th*.—Heat, dryness of skin and thirst from ½ to 4 o'clock to 6 P. M., tongue furred, weak and without appetite.—Continue.

25*th*.—Fever returned yesterday with rigor at 2 o'clock P. M. He began to perspire at ½ past 4 o'clock P. M., now apyrexia.

Pulv. cinchonæ 2 drs.
To take 4 such doses at the intervals of two hours.
Omit phosphate of lime and sulphur powder.

26*th*.—No return of fever.—Nil.

30*th*.—Remains free from fever.—To duty.

Febris Quotidiana Intermittens.

Shaik Abdoolah, Horse Driver, A Company, 1¼ years in the service.

Of full habit.

Admitted at noon on the 3d October 1849, complaining of fever, which he says attacked him three days ago at 5 o'clock P. M. and since has recurred daily. It does not leave him till early in the morning; tongue furred, vertigo, bowels confined, but there was no fever on admission.

Rec. calomel 4 grs.
Pulv. jalapæ comp. 2 scru. mft.
Pulv. statim sumendus.

Vespere, skin hot and dry, pulse accelerated, much vertigo and pain in the extremities, thirst and nausea, bowels once moved.—Nil.

4*th*.—A restless and sleepless night, fever remains unabated, skin hot and dry, pulse 84; what he complains of are vertigo and pain in the extremities, tongue furred.

2 drs. of the powder of phosphate of lime with sulphur thrice daily.

Vespere, it is said he perspired at noon but skin is now both hot and dry, he is restless, pulse 90, no stool.

Repeat calomel and compound jalap powder, and omit the third dose of the phosphate of lime powder.

5*th*.—Fever left at 5 o'clock this morning when he perspired profusely, two copious stools, at present no fever.

Repeat phosphate of lime powder 2 drs. thrice daily.

Vespere. Escaped fever.—Continue.

6*th*.—Fever returned at 11 o'clock P. M., it began with a chill followed by heat of skin and inability to sleep, is now restless and giddy, he complains of thirst and of a feeling of cold, pulse 90, two stools.

Continue powder thrice daily.

Vespere, perspired after the first dose of the powder, about 10 o'clock skin became hot and dry and so it continued till 5 P. M. when he began to perspire; at present skin is warm and moist, bowels once moved, pulse 96, a fur on the tongue, complains of vertigo and of pain all over his body.

Rec. calomel 10 grs.
Pulv. antim. comp. 5 grs.
Mft. pil 3 immediately, and
after two hours to take ol. ricini 1 oz.

7*th*.—Perspired at 8 o'clock and since has been free from fever, bowels twice moved, evacuations chiefly feculent, apyrexia this morning.

Repeat powder of phosphate of lime as before, thrice to day.

Vespere, has had much fever all day, restless; complains of universal pain, skin hot and dry, bowels not moved, no diaphoresis, took the last dose of the powder an hour ago.

Repeat pil et ol. ricini as yesterday.

8*th*.—Apyrexia since 4 A. M. 3 stools.
Rec. quinæ disulph. 6 grs.
Acid sulphur dil. 6 gts.
Aquæ—2 oz. ft.
haustus omni hora ad 3 tam vicem.

Vespere, slight fever from 10 A. M. till 6 this evening, bowels free.—Nil.

9*th*.—Apyrexia, has passed a good night.

Repet haustus ut heri.

10*th*.—Escaped fever yesterday.—Nil.

He had no return of fever, and on the 14th he was discharged to his duty.

Febris Quotidiana Intermittens.

Sundeogh, Driver, D Company, aged 25, 7 years in the service, No. 1179.

Admitted on the 18th August 1850, by account has had fever daily since the 14th, in a paroxysmal form; on that day

it began at 10 A. M. and ended at 6 P. M. On the ensuing morning at 10 o'clock, the fever returned and remained till noon. On the 16th a third paroxysm from 2 to 3 P. M.; he says he had fever from 3 to 5 A. M., this morning; on admission tongue foul, bowels open from medicine he had taken, complained of debility and loss of appetite, skin cool.

Mistr. salina simp. 2 oz.
ter in die.

19*th.*—Had a return of fever at 10 o'clock P. M. which did not leave him till 7 A. M., tongue still furred, bowels slightly moved by the saline mixture.

Powder of the phosphate of lime with sulphur 2 drs. thrice to-day.

20*th.*—Was attacked with fever at 10 o'clock yesterday evening. It left him at midnight when he perspired freely, bowels open, tongue clean.

Repeat powder as yesterday.

21*st.*—Perspired freely throughout the day but this morning at 1 o'clock there began a paroxysm of fever that lasted for 3 hours, tongue slightly furred, bowels twice moved; at present apyrexia.

Repeat powder.

22*d.*—No return of fever.—Nil.

25*th.*—Discharged to duty.

Febris Quotidiana Intermittens.

Michael Conway, Half-pay bugler, A Company, aged 14. Born in India.

This boy was detained in hospital under observation on the 16th May, he came to hospital stating that he had fever; that it came on, on the 15th about noon, on admission, however, skin was cool but tongue foul, he had no appetite

and said he felt unwell, bowels slow; he took calomel 4 grs. pulv. jalapæ comp. 2 scru. by which his bowels were twice acted on; at noon heat of surface, headache, pain in the eyeballs, and redness of conjunctiva with flushing of the face and restlessness supervened; saline mixture 1 oz. every 3rd hour was given and 6 leeches were put on the temples, he began to perspire about 9 o'clock after he had taken calomel and pulv. antim. comp. each 3 grs. The saline mixture was at that time omitted.

17*th.*—Apyrexia, has slept, bowels not again moved, tongue furred, white with a central reddish patch.—Spoon diet.

Pulv. phosph. of lime with sulph. 1½ drs. ter in die.

18*th.*—No return of fever, no evident action of medicine except that it moved his bowels once.—Stool not kept.

Repet ut heri.

19*th.*—One feculent stool of the colour of sulphur, he has remained well since last report.

Omit pulvis.—Low diet.

21*st.*—Well.—To duty.

Febris Tertiana Intermittens.

Patrick James Cassarley, Half-pay bugler, D Company, aged 11 years. Born in India.

Admitted on the 19th April 1850, at 6 o'clock A. M. but he came to hospital the previous day at 4 o'clock P. M. with his mother who gave this account; on the 14th an attack of fever, on the 16th a similar one at the same hour, and on the 18th another, all the attacks began about the same time and ended in perspiration, between 4 and 5 o'clock; when he was brought to hospital his skin was warm and moist, he had headache, general febrile uneasiness and thirst, tongue pretty clean, bowels said to be regular, pulse frequent.

Has had intermittent fever at this station in October 1849.

Rec. calomel 3 grs.
Pulv. jalapæ comp. ½ dr. statim.
Spoon diet.

19*th.*—Bowels once freely moved, his nose bled and he passed a good night, tongue pretty clean, skin cool and moist.

Rec. pulv. phosph. of lime with sulphur 1 dr. ter in die.

20*th.*—The only effects the powder had were to excite perspiration, which was very free at the evening visit yesterday, and to move his bowels once, stool was consistent and of the colour of the powder; he has had no return of fever.

Repet pulv. ter in die.

21*st.*—No return of fever, bowels slow. Omit pulvis.

Pulv. jalapæ comp. ½ dr. statim.

22*d.*—Bowels satisfactorily acted on, no return of febrile disturbance, a herpetic eruption on the mouth, it appeared on the 20th.—Nil.

23*d.*—Nil.

24*th.*—Low diet.

28*th.*—Well since last report.—To duty.

Admitted again on the 3d June 1850 at 5 P. M., with slight fever which by account attacked him at 9 this morning, bowels slow, he therefore was ordered calomel 4 grs. pulv. jalapæ comp. ½ dr. by which his bowels have been moved once; a good night, apyrexia since 7 P. M. abdomen full and tense.

Mistr. purgans 2 ozs. statim, post horas duas 1 oz. si opus sit.—Spoon diet.

5*th.*—Bowels were well moved, abdomen is now soft and free from uneasiness. There is no symptom of fever.—Nil.

6*th.*—Convalescent.—Low diet.

7*th.*—Well.—Discharged.

Febris Tertiana Intermittens.

John A. Dammermoth, bugler, D Company, aged 26.

Born in India.

Admitted on the 1st June 1850, at noon with slight symptoms of fever, which came on at 9 A. M. with a feeling of chilliness; knows no cause for the attack; his tongue being foul and bowels slow, he took 2 grs. of tartar emetic and 1 scru. of ipecacuan by which free vomiting ensued; and calomel 6 grs. and pulv. jalapa comp. 1 dr., which purged him several times.

2*d June.*—At the evening visit yesterday he was in a state of perfect apyrexia in which he remains.—Nil.

Spoon diet.

Vespere.—Bowels slow.

Extr. colocy. comp. 8 grs.

3*d.*—As yet bowels not moved.

Pulv. jalapa comp. 1 dr. statim.

Vespere, fever returned at 8 A. M., commencing with a chill, some headache, heat of surface and an aching in his loins and lower extremities with other symptoms followed, he began to perspire a little before noon, and skin is now cool, but headache and muscular soreness and debility remain, four stools not kept.

Pulv. phosph. of lime with sulphur 2 drs. h. s. s.

4*th.*—The powder caused thirst, tongue coated with a whitish fur, perfect coolness of surface, is weak but he has no pain.

Repet pulvis.

Vespere, no return of fever, one feculent stool of the colour of sulphur.

Continue.

5*th.*—Fever returned at 4 o'clock A. M. commencing with rigor, followed by headache, pain in the back and limbs, thirst, heat and dryness of skin; he has been and continues to be very

restless, although there is a slight moisture on the skin which is still hot, pulse 96, tongue has its papillæ raised and white.

Continue.

6*th*.—Fever left at 10 o'clock A. M. yesterday, he has no trace of fever this morning, except the fur on tongue. He feels weak however, skin cool and moist, two feculent stools of the color of sulphur.—Continue.

7*th*.—Apyrexia from the time of last report till 2 A. M. to-day, when he had a rigor followed by heat of surface and slight headache, began at 5 A. M. to perspire, skin is now at 6 A. M. cool and moist. The attack much milder than any previous one, no stool, tongue much as it was.

Continue pulvis ter in die.
Rec. acid. nitro-muriat. 2 drs.
Aqua, 24 ozs. mix. fiat
Potus, during the day.

8*th*.—Apyrexia since last report, two stools whitish feculence with a yellow tint, took the whole of the acid drink, feels weak, tongue is cleaner.

Continue pulvis.
Omit potus.

9*th*.—Fever returned at midnight commencing with slight rigor which was succeeded by restlessness, thirst, slight headache and slight pain in the lower extremities, skin hot and dry. These symptoms terminated in perspiration at 3 o'clock A. M.; now at 7 A. M. apyrexia, tongue has a whitish sodden appearance, he feels very weak, and looks worn and emaciated, bowels once moved, a feculent stool much of the same appearance as before.

Repeat as yesterday

10*th*.—No return of fever, bowels once moved evacuation feculent, tongue is less sodden in appearance, he feels very weak, but he slept well and his appetite is good.

Repeat powder.

11*th*.—He himself states that he had fever (heat of skin only) from 10 P. M. till midnight but he called no one, afterwards slept well, tongue is cleaner, appetite good, bowels open, I believe he had no fever.

Omit pulvis.—Low diet.

13*th*.—Has continued well since last report.—Nil.

14*th*.—Well.—Discharged.

Vespere, this man did not leave the hospital, for at 2 P. M. he had a slight chill followed by heat of surface, restlessness, accelerated pulse and slight headache; at 5 P. M., he began to perspire and felt relieved, now complains of headache only, bowels not moved to-day, tongue is clean.

Rec. Pilulæ hydrarg. 4 grains.
Ext. colocynth comp. 6 grs. for bed time.

15*th*.—A good night, apyrexia, two stools.

Mistr. quinæ 2 ozs. 2da. q. q. hora.
Ad. 3 tiam vicem.

16*th*.—No return of fever.

Repet mistr. quina ut heri.

17*th*.—Convalescent.—Low diet.

18*th*.—Half diet.

19*th*.—To duty.

Febris Tertiana Intermittens.

Hugh Kane, Gunner, D Company, aged 34.
Resident in India, 9 years.

13*th July*, 1850.—Admitted on the 12th at 2 P. M., in the cold stage of tertian ague, the 2d paroxysm; the first he had on the 10th it commenced at 2 o'clock P. M. That of yesterday set in an hour earlier at 1 P. M. He had on both occasions all the 3 stages of the disease well marked. On admission tongue being foul and bowels slow he was ordered calomel 6 grs. and pulv. jalapæ comp. 1 dr. by which his

bowels were well acted on once, the motion is dark loaded with bile and frothy, tongue furred at the base.

Pulv. jalapæ comp. 1 dr. statim.—Spoon diet.

14*th*.—Bowels once moved by the jalap; herpetic eruption on the lips, no uneasiness any where, tongue coated with a whitish fur, this is fever day.—Nil.

Vespere, cold stage of a paroxysm commenced at 1 P. M., and was followed by heat and sweating, the latter commencing at 3, and continues to be profuse; attack much less severe than the former, tongue whitish with a red margin, was ordered immediately on the accession of fever, pulv. phosph. of lime with sulph. 2 drs. 3 tis. horis ad. 3 tiam vicem.

Continue powder.

15*th*.—A good night, apyrexia, no stool for 24 hours, tongue in the same state.

Repet pulvis ter in die.

Rec. acid nitro-muriat 1½ drs.
Aquæ—24 ozs. fiat.
Potus-bibat in die.

16*th*.—This is fever day, at present except weakness no complaint, one feculent stool.

Repeat as yesterday.

17*th*.—Escaped fever yesterday.

Omit remedia.
Low diet.

18*th*.—Convalescent.—Nil.

20*th*.—Well.—Discharged.

Febris Tertiana Intermittens.

Mathew McNamarra, Gunner, D Company, aged 23.
Resident in India, 2 years.

Admitted on the 14th July 1850, at 10 o'clock A. M., with the second paroxysm of a tertian ague, which attacked him

on the 12th and on the day of admission at the same hour 10 o'clock; on both occasions, all the three stages, cold, hot and sweating were well marked, the latter commencing at 2 P. M., tongue was foul, bowels slow, much headache.

He took on arrival at the hospital 1 grain of tartar emetic and 1 scruple of ipecacuan by which his stomach was emptied and the powder purged him once.

15*th*.—A good night, this morning apyrexia is perfect, tongue quite natural.—Nil.

Spoon diet.

16*th*.—This is fever day.—Nil.

10 A. M.—Has just had a slight rigor and skin is becoming hot.

Phosphate of lime with sulphur 2 drs. ter in die.

17*th*.—The attack of fever was slight, he began to perspire about noon yesterday; slept well, and there is complete apyrexia this morning, tongue clean.

Repeat as yesterday.

18*th*.—This is the day for the fever to return. Is now well.—Repeat.

19*th*.—No return of fever, bowels open.—Nil.

Low diet.

20*th*.—To duty.

Febris Tertiana Intermittens.

George Maslon, Gunner, D Company, aged 31.
Resident in India, 3 years.

Admitted on the 22d July 1850, at 11 o'clock A. M. in the hot stage of what appeared by his account to be the third paroxysm of a mild tertian ague; the first paroxysm and the last, the one with which he was admitted commenced by slight rigor at 9 o'clock A. M., and the intermediate one which was the longest and most severe at 1 o'clock P. M. tongue furred, bowels slow; he took on admission an emetic

composed of 1 grain of tartar emetic and 1 scru. of ipecacuan and subsequently calomel 5 grains and pulv. jalapæ comp. 1 dr., both of which acted satisfactorily.

23*d*.—This morning perfect apyrexia.—Spoon diet.

24*th*.—Fever returned with rigor this morning and he has now slight heat of surface, pain in back and thighs, a moist whitish fur on the tongue, thirst, bowels twice moved, vertigo.

Pulv. phosph. of lime with sulphur.
2 drs. ter in die.

Vespere, began to perspire at 10 o'clock A. M., and there is now distinct apyrexia, bowels open.—Continue.

25*th*.—Did not sleep well, but this he attributes to his having slept during the day yesterday, a whitish slimy moist fur on the tongue, pulse 66 soft.

Repet pulvis ter in die.
Potus acid. nitro. muriatic.

26*th*.—Fever returned at 4 o'clock this morning, first there was rigor, then heat of surface, pain in the back and other usual but mild symptoms of the hot stage, is now at 7 A. M. beginning to perspire, bowels open, tongue is much as yesterday.

Repet pulv. et potus ut heri.

Vespere, took the first dose of the phosphate at ½ before 6 o'clock A. M.; perspiration broke forth about 20 minutes thereafter and with it the attack terminated, apyrexia this evening, bowels regular.—Continue.

27*th*.—A good night, an eruption of vesicles on the lips.—Continue.

28*th*.—Fever returned at 4 o'clock this morning commencing with rigor, the hot stage was of short duration and terminated in sweating at 6 o'clock, no uneasiness, complains only of debility, bowels open, tongue is nearly clean.

Omit pulvis.

29*th*.—Apyrexia now, but fever may be expected to return to-night or very early to-morrow morning.

Mistr. quinæ. 2 ozs. meridie et.
Repet at 4tam vicem 2da. q. q. hora.

30*th*.—No return of fever.

Omit mistr. quinæ.—Low diet.

31*st*.—No fever.

Repet mistr. quinæ. 8 ozs.

1*st August*.—Has again escaped fever.—Half diet.

2*nd*.—Well.—Discharged.

Febris Tertiana Intermittens.

William Prout, Gunner, D Company, aged 40.
Resident in India nearly 16 years.

Admitted on the 6th June 1850 at 6 P. M. with the usual symptoms of fever which by account attacked him at 3 o'clock this afternoon, had a previous paroxysm on the 4th at ½ past 4 o'clock P. M. tongue foul, bowels slow.

Took 1 grain of tartar emetic and ipecucuan 1 scru., and subsequently calomel 6 grs. pulv. jalapæ comp. 1 dr.

7*th*.—Emetic acted well, one stool from the purge.

Haust purgans 4 ozs.
Tinct. zingiberis 3 drs. mix fiat. haustus statim.
Spoon diet.

8*th*.—No return of fever.—Nil.

Vespere, fever returned at 4 o'clock without rigor; the symptoms are mild, he complains of his head only, bowels not open, his skin is hot and dry at present, pulse accelerated, tongue coated with a whitish fur, moist.

Pulv. phosph. of lime with sulphur 2 drs. h. s. s.

9*th*.—Fever terminated a little after 7 o'clock yesterday evening, he has slept indifferently, he complains of a feeling

of weight in the head this morning, the powder caused him to perspire freely, bowels not moved, tongue pale and coated with a white fur, there is however perfect apyrexia.

Repet pulv. ter. die.

Potus acid nitro muriat. 24 ozs. in die.

10*th*.—No return of fever, two feculent stools, tongue white and sodden in appearance, has slight uneasiness over the right eye brow.

Repet pulvis.—Omit potus.

11*th*.—No return of fever, bowels open, tongue whitish moist, appetite good.

Omit pulvis.—Low diet.

13*th*.—Has continued well since last report.—Nil.

14*th*.—Well.—Discharged.

FEBRIS TERTIANA INTERMITTENS.

Robert Wiggins, Gunner, A Company, aged 32.
Resident in India 12 years.

16*th*.—*August* 1850. Admitted at 8 A. M. on the 15th August. He is a court martial prisoner, a man of intemperate habits, spare form and sickly aspect.

Had on admission feelings of chilliness soon followed by heat of surface, headache, and other usual but slight symptoms of the hot stage, during which he took calomel 5 grs. and pulv. jalapæ comp. 1 dr. as his bowels were confined; the dose has operated well.

This morning apyrexia. By account had on the 13th a febrile attack commencing at 11 o'clock, a slighter attack on the 14th, and the paroxysm of yesterday at 8 A. M.

Nil.—Spoon diet.

17*th*.—It is said that he had heat of skin for an hour yesterday, but no one saw him while it was present, he seems to be quite well.

Mistr. salin simp. 2 ozs. ter quater in die.

8 A. M.—He is now in the cold stage of ague, it is a slight attack.

Pulv. phosph. of lime with sulphur 2 drs. ter in die.

Vespere, the hot stage followed and terminated at 11 o'clock A. M.—Continue.

18*th*—A good night, bowels not moved.

Repet pulvis.
Acid nitro muriatic 1½ drs.
Aqua.—24 ozs. mix.

19*th*.—Bowels have not been moved, no return of fever, but it is expected to-day.

Repet ut heri et.

Injic. enema purgans.

20*th*.—Bowels twice moved by the injection no return of fever.—Nil.

Low diet.

21*st*.—Continues well.

22*nd*.—Bowels slow.

Ol. ricini. 1 oz.

To duty in the afternoon.

FEBRIS QUOTIDIANA INTERMITTENS.

John Kirby, Gunner, D Company, aged 29.
Resident in India 7 years.

Admitted at ½ past 5 o'clock A. M. on the 22d November 1850, with pyrexia, slight headache, nausea, vomiting, and purging, hands tremulous, has been drinking lately. By ac-

count has had fever since the evening of the 17th, it returned every evening, no cause for the attack.

Rec. calomel 10 grs.
Opium 1 gr. mix, fiat pilula 2 statim.
Rec. mistr. purgans 4 ozs.
Pulv. jalapæ comp. 1 scru. after three hours.

Vespere, perfect apyrexia, the tremor less, bowels freely acted on.—Nihil.

23*d*.—A good night, tremor still continues, says it is owing to excessive indulgence in tobacco which he chews; two stools, they contain some undigested food.

Ol. ricini 1 oz. statim.—Spoon diet.

Vespere, fever returned at 11 o'clock, a well marked paroxysm; is now sweating, bowels moved by oil, a fur on the tongue which is moist, at noon he took 1 dr. of the phosphate of lime with sulphur.

Repet pulvis statim et h. s.

24*th*.—Continued to perspire all night, was restless, did not sleep. This morning feels giddy and suffers slightly from nausea and thirst, skin warm and moist, tongue clean, one stool feculence of pretty natural appearance.

Repet pulvis ter in die.

25*th*.—No return of fever, bowels free.—Repeat pulvis.

Vespere, fever returned at noon when he felt chilly, afterwards skin became hot and dry, with slight headache and pains in the loins, tongue remained nearly clean and moist, pulse 120 small, no stool.—Continue.

Temporibus Hirudines xiv.

26*th*.—About ½ past 5 o'clock began to perspire, he did not sleep well was restless but not from pain. There is perfect apyrexia this morning, one stool, tongue clean and moist.

Repet pulvis ter in die.
Nitro-muriatic drink 24 ounces.

27*th*.—No. return of fever.—Repeat.

Vespere, fever since noon, it began with a chill followed by heat and dryness of surface, some headache, and a great feeling of aching all over the body, vertigo. Begins to perspire, one stool feculent, tongue furred, moist.

Rec. Calomel, pulvis antim. Comp.
Each 5 grs. mis, fiat pulv. h. s.
Hot foot bath.

28*th*.—A good night, perfect apyrexia.

Mistr quinæ. 2 ounces omni hora.
ad 4 tam vicem.

29*th*.—No return of fever, but he could not sleep.—Repeat.

30*th*.—Slept well. Convalesces.—Nil.—Low diet.

1*st October*.—Low diet.

3*rd*.—Well.—Discharged.

Febris Quotidiana Intermittens.

James Malony, Gunner, aged 30, A Company.
Resident in India 8 years.

Admitted at noon on the 22d November, 1850, with the usual symptoms of fever which he says he got yesterday after 10 o'clock P. M. a chilliness followed by heat and sweating. In the morning he was well but fever returned about half an hour before admission. Tongue foul, bowels slow.

Rec. antim. potass tartras 1 gr.
Pulv. ipecacuana, 1 scru. mis, fiat.
Pulvis statim.
Rec. calomel, 6 grs.
Pulv. jalapæ comp. drs. 1 mft. pulvis.
after three hours.—Spoon diet.

Vespere, medicine has not acted.

Mist. salinæ simpl. 2 ounces.
Omni hora ad effectum.

23*d*.—After the 3d dose free purging and sweating, a good night, this morning apyrexia.—Nil.

Vespere, no return of fever.

24th.—At 10 P. M. chilliness followed by the usual symptoms of fever which remains, could not sleep but rolled about on his cot all night, pain in the loins, vertigo, slight headache, pulse 90; along with the heat of skin there is partial sweating, much thirst, tongue whitish moist, one pretty natural stool.

Pulv. phosphate of lime with sulphur.
2 drs. ter in die.

25*th*.—No return of fever, bowels slow.

Repet pulvis.

1½ drs. of nitro muriatic acid to 24 ounces of water as a drink, the whole to be taken in the course of the day.

26*th*.—Chilliness returned at 6 P. M., afterwards slight fever, some headache, one stool yesterday, to-day apyrexia, became cool at 4 A. M., tongue slightly white, it is moist.

Repeat as yesterday.

27*th*.—No return of fever, bowels open.—Repeat.

Vespere, fever returned at ½ past 11 o'clock, it began by a chill which was followed by heat and dryness of surface, some headache and thirst. These symptoms are now present and he complains of great debility and vertigo, tongue white and moist.

Rec. calomel, pulv. antim. comp.
Each 5 grs. mis fiat pilulæ h. s. s.
Hot foot bath.

28*th*.—Began to perspire at 7 o'clock after the foot bath. This morning perfect apyrexia, tongue cleaner, bowels open.

Mistr. quinæ 2 ounces, omni hora ad 4 tam. vicem.

29*th*.—No return of fever.

Repet mistr. quinæ ut heri.
Low Diet.

30*th*.—Convalescent.—Nil.

1*st December*.—Nil.

3*d*.—Low Diet.

4*th*.—Well.—Discharged.

REMARKS.

All these cases of intermittent fever (European and Native) were of the most uncomplicated kind, and without exception such as bark or quinine, as usually given, would at once have cured.

Seeing 6 Europeans and 2 native cases, 8 out of 15, got well under the use of Mr. Blacklock's powder, a stranger to the fever of this place will be apt to conclude that the results are very much in favor of the remedy. So many slight cases of ague however recover at this station without any treatment or after one emetic or purgative; and the failure of the remedy in the remaining 7 cases was so signal and contrasted so strongly with the effect of the bark or quinine which had eventually to be employed to prevent the return of the paroxysms that I suspect Mr. B.'s powder possesses very feeble antiperiodic powers. Many slight cases of intermittent fever will, I doubt not, recover under its use, and as it is a cheap remedy it deserves a further trial especially in the cases of natives to whom Mr. B. has given it most extensively and successfully.

The fever powder was slightly laxative and diaphoretic.

Each ounce of the quinine mixture contained 3 grains of quinine. The simple saline mixture was a weak solution of salts and tartar emetic.

J. ANDERSON, M. D.
Assistant Surgeon,
3d Battn. Artillery.

KAMPTEE,
12*th December*, 1850.

CASES TREATED BY SURGEON JOHN DREVER.

Febris Quotidiana Intermittens.

Private, Shaikh Emaum, D Troop, No. 382, aged 33 years, service 17 years.

Admitted 14th August 1850. Complaining of having suffered from fever since 12 A. M. yesterday, which left him at 12 P. M., with free perspiration, no previous rigors. It came on again at 2 A. M., skin hot, pulse quick, bowels confined, no thirst, no headache, tongue coated with red margins.

Can assign no cause.

Rec. phosph. of lime with sulphur 2 drs. ter die.

Vespere, 5 P. M., slight remission at 2 P. M., of short duration, one dejection, skin now hot and dry.

Repeat 2 doses of phosph. during the night.

15*th*.—Fever continued last night without remission, but had free perspiration, no local pains, skin warm, pulse small and quick, no thirst, two dejections, did not rest well.

Continue phosphate ter die.

16*th*.—The fever subsided last night, has had no return, skin cool and moist, tongue cleaning, no headache, no thirst, bowels opened, urine free and high colored.

Continue phosphate.

17*th*.—Free from fever, complains of general weakness, skin cool, in other respects the same, took one dose of phosphate of lime with sulphur last night.

Continue med.

18*th*.—Well.—Discharged.

Febris Quotidiana Intermittens.

Private, Shaikh Homed, C Troop, No. 675, aged 25 years, service 7½ years.

Admitted 15th August 1850, 5 P. M. Complaining of general febrile symptoms, skin hot, pulse frequent, tongue coated, bowels regular, no thirst or headache, muscular pains all over the body; states that the fever came on 3 days ago with slight remission at times, perspiring then freely, no previous rigors; no cause for his complaint.

Rec. phosphate of lime with sulphur 2 drs. ter die.

16*th*.—Fever continued last night and left him at 5 A. M., was very restless, skin now cool and perspiring, pulse weak, tongue coated with red margins, two dejections, urine free and high colored.

Continue phosphate.

17*th*.—Fever returned last night, was restless, skin now hot, pulse frequent, tongue the same, one dejection, urine free.

Continue phosphate.

18*th*.—Fever left him yesterday morning and is free from it since, bowels opened.

Continue phosphate.

19*th*.—Return of fever at 10 A. M. yesterday, and it continued till 4 A. M. this morning, skin still rather warm, tongue white, pulse small, one dejection, urine free.

Continue phosphate.

20*th*.—Heat of skin subsided during the day, and slight return of it last night, at present functions natural.

Continue phosphate.

21*st*.—No fever yesterday, feels better, two dejections.

Continue phosphate.

22*d*.—No fever, well.—Discharged.

Febris Quotidiana Intermittens.

Private, Kissen Singh, B Troop, No. 380, aged 41 years, service 17 years.

Admitted 22d August 1850, 5 P. M. Complaining of having suffered from febrile symptoms for the last 3 days pre-

ceded by rigors with morning remissions succeeded by free perspiration, skin hot and dry, tongue coated in centre with red margin, pulse quick, headache, pain across the loins and limbs, bowels irregular, no thirst, urine free.

Rec. phosph. of lime with sulph. 2 drs. ter die.

23d.—Fever continued till 5 A. M., skin now cool, tongue the same, pulse soft, two dejections, urine free, pain of head better, did not rest well.

Continue phosphate.

Vespere, 5½ P. M. slight return of fever at 4 P. M., pulse 80 small, skin now perspiring, bowels confined.

Repeat 2 doses of phosph. during the night.

24th.—Return of fever at 1 and left at 3 A. M. without previous rigors, but perspired slightly, was very restless, pains of limbs the same, skin moist, pulse weak, tongue brown and dry, slight pain of head and heavy, no thirst, very weak, three dejections, urine free.

Continue phosphate.

25th.—Return of fever at 10 A. M. and continued till 5 A. M., no rigors, slight perspiration, passed a restless night, skin now perspiring, pulse soft, tongue brown and dry, no thirst, head still heavy and feels giddy but no pain, pains of limbs not better, very weak, one dejection, urine free and scalds but not high colored.

Continue phosphate.

26th.—No return of fever since last report, slept a little, skin cool, pulse small, tongue foul and moist, giddiness the same, does not feel any heaviness of the head, pain of limbs less, one dejection, urine the same, is anxious to have an emetic.

Rec. pulv. ipecac. 20 grs.
Ant. pot. tart. 1 gr. M. st.
Continue phosphate.

28th.—Has continued free from fever for the last 4 days,

feels still giddy, and has heaviness of head, bowels opened, but not freely, in other respects well.

Omit phosphate.
Rec. pulv. jalap com. 1 dr. st.

29th.—Bowels have been freely acted on, and functions seems natural.—Discharged.

The above patient had a return of fever some time afterwards, was treated with an emetic, purgatives and quinine, and discharged well in seven days, no relapse since, now nearly three months.

Febris Quotidiana Intermittens.

Private Shaikh Homed, E Troop, No. 315, aged 34 years, service 9 years.

Admitted 24th August 1850, 6 A. M. Complaining of having suffered from fever for the last three days, generally with evening accessions and morning remissions, the fever preceded by rigors, which lasts nearly two hours followed by heat of skin and succeeded by perspiration. On his admission this morning the following symptoms were present, skin hot and dry, pulse hard and frequent, tongue brown in centre, bowels irregular, was restless, states that the fever came on at 10 P. M. last night and continues still, urine high colored, no thirst, appetite indifferent.

Rec. phosph. of lime with sulph. 2 drs. ter die.

25th.—Fever left him at 11 A. M. with free perspiration, and returned last night at 10 preceded by rigors, remained till 12½, passed a restless night, skin now cool, pulse small, tongue coated with brown lines in centre with red margin and moist, no thirst or headache, complaining of pain of back and limbs, appetite bad, one dejection, urine scanty and high colored.

Continue phosph.

Vespere, return of fever at 12 A. M., skin at present per-

spiring, has a tendency to nausea, slight giddiness, bowels opened.

Rec. calomel 8 grs.
Pulv. jalap comp. 2 scru. M. st.

26*th.*—No return of fever, giddiness and nausea the same, skin cool and moist, pulse soft, tongue brown, pains of body the same, did not rest well, medicine operated well.

Continue phosph.

Vespere, return of fever from 10 till 12 A. M. skin now cool, pulse soft, tongue white, pain of body better.

Repeat one more dose of phosph.

27*th.*—No return of fever, is weak, in other respects well.

Continue medicine.

Vespere, 6 P. M., return of fever from 10 till 12 A. M. without rigors, perspired a little, no headache or giddiness, skin cool, pulse soft, tongue clean, 3 dejections, urine free, appetite indifferent.

Repeat 2 doses of phosph.

28*th.*—Free from fever since last report, slept well, skin cool and moist, tongue clean, bowels opened.

Continue phosphate.

29*th.*—Free from fever for the last two days, feels weak, slept well, appetite good, urine free, three dejections, skin and pulse natural.

Omit med.

30*th.*—Improving.

Nihil.

31*st.*—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Shaikh Madar, D Troop, No. 607, aged 26 years, service 8 years.

Admitted 25th August 1850, 3½ P. M. Complaining of having had fever for the last six days attended with previous rigors which lasted half an hour, then the heat of skin followed generally with morning remissions succeeded by slight perspiration; the fever is said to have come on just now with previous rigors, skin hot and dry, pulse hard and frequent, tongue brown in centre with red margin, bowels regular, no thirst or headache, pains of back and limbs, feels giddy.

Rec. phosphate of lime with sulphur 2 drs. ter die.

26*th.*—Fever left him at 2 A. M. with slight perspiration, skin at present rather warm, pulse small, tongue brown, bowels opened, giddiness the same, pain of body less.

Continue phosphate.

Vespere, 6 P. M., warmth of skin subsided at 10 o'clock, and returned at 2½ P. M., skin warm, pulse frequent, tongue foul, two dejections, giddiness the same, pain less.

Repeat 1 dose of phosphate.

27*th.*—Fever continued till 4 A. M., did not rest well, skin perspiring, pulse soft, tongue foul, one dejection, complains of heaviness and giddiness of head.

Continue phosphate.

Vespere, 6 P. M. slight return of fever at 11 A. M. and remained till 4 P. M., skin now cool, pulse soft, tongue brown, four dejections, heaviness the same, is weak.

Repeat two doses of phosphate.

28*th.*—No return of fever, slept well, skin cool and moist, in other respects better, bowels opened.

Continue phosphate.

29*th.*—Slight return of fever at 12 P. M. which remained for half an hour, without rigors, perspired freely, slept well, slight pain of body, much thirst, skin cool, tongue very foul with red margin, pulse soft, one dejection.

Continue phosphate.

30*th*.—No return of fever, feels better, bowels opened.
Continue.

1*st September*.—Improving, bowels opened.
Omit med.

2*d*.—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Jemadar Noorodeen, D Troop, No. 64, aged 47 years, service 32 years.

Admitted 25th August 1850, 5 A. M. Complaining of having suffered from fever since 5 P. M. yesterday, preceded by rigors which lasted half an hour, and the remission is said to have taken place at 12 P. M., but returned at 4 A. M. with the usual rigors and continues still; skin hot and dry, pulse hard and frequent, tongue brown with red margin, heaviness and pain of head, muscular pains all over the body, bowels irregular, slight thirst, urine scanty and high colored, no appetite.

Rec. phosphate of lime with sulphur 2 drs. ter die.

26*th*.—Fever left him at 12 P. M. with free perspiration, slept well, skin rather warm, pulse frequent, tongue the same, headache better, pain of body much better, one dejection, urine and thirst the same.

Continue phosphate.

27*th*.—Fever returned last night, was very restless, but is now free from it, no headache, two dejections, pain of body the same, had two more doses of phosphate last night.

Continue phosphate.

28*th*.—Fever returned last night, and is now free from it, skin cool, pulse soft, tongue cleaning, three dejections, urine the same.

Continue phosphate.

Vespere, 6 P. M., fever returned at 12, but is now perspiring, tongue and pulse the same, two dejections.

Continue phosphate.

29*th*.—Was free from fever since last report, but did not rest well, skin cool and moist, pulse soft, tongue clean, three dejections, urine free.

Continue phosphate.

30*th*.—No return of fever, slept well, functions seem natural.

Continue phosphate.

31*st*.—Improving, bowels opened.

Omit med.

2*d September*.—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Cajah Mahomed, C Troop, No. 794, aged 23 years, service 12 years.

Admitted 25th August 1850, 5½ P. M. Complaining of having suffered from fever for the last two days, preceded by slight rigors and followed by heat of skin ending in the mornings by free perspiration; skin at present hot and dry, pulse quick, tongue white with red margin, slight pain of limbs, no headache, much thirst, bowels regular, appetite bad, urine scanty and high colored and scalds; the febrile symptoms said to have come on at 5 P. M.

Rec. phosphate of lime with sulphur 2 drs. ter die.

26*th*.—Fever continued till 8 P. M. and returned again at 10 P. M. without rigors and lasted till 4 A. M. did not perspire, skin warm, pulse frequent, tongue very foul and red at the margin, less thirst, urine the same, bowels confined.

Rec. haust. purgans 4 ozs. st.
Continue phosphate.

Vespere, 6 P. M., fever continued till 2½ P. M. though per-

spired freely, but skin still warm, pulse quick, tongue foul, purgative operated well.

Repeat one dose of phosphate.

27*th*.—Was free from warmth of skin till 11 P. M., when it returned and lasted till 5 A. M., skin still perspiring, pulse soft, tongue foul, two dejections.

Continue remedies.

28*th*.—Slight return of fever yesterday of short duration and did not return since, skin cool and moist, pulse soft, tongue cleaning, two dejections.

Continue phosphate.

29*th*.—No return of fever since last report, skin cool, pulse small, tongue clean, urine free, complains of giddiness, no headache, and is weak.

Continue phosphate.

30*th*.—No return of fever, bowels opened.

Continue phosphate.

1*st September*.—No fever, bowels opened.

Continue med.

2*d*.—Well.—Discharged.

This patient had a return of febris quotidiana intermittens 16 days afterwards, was treated with an emetic, purgative and quinine and discharged well in four days, no relapse since, now nearly three months.

Febris Quotidiana Intermittens.

Private Adbul Cawder, E Troop, No. 847, aged 22 years, service 4 years.

Admitted 25th August 1850, 5½ P. M. Complaining of having suffered from fever for the last two days with previous rigors and two remissions succeeded by slight perspiration, it has returned at 2 P. M. to-day with rigors, skin now warm and dry, pulse frequent, tongue foul with red margin,

no headache or pain of limbs, bowels regular, urine high colored, appetite good, much thirst.

Rec. phosphate of lime with sulphur 2 drs. ter die.

26*th*.—Fever left him at 2 A. M. with free perspiration, he is free from it now, skin cool and moist, pulse soft, tongue foul, was restless, bowels confined, urine the same.

Continue phosphate.

Vespere, slight heat of skin, pulse small, tongue foul, bowels opened.—Continue.

27*th*.—Heat of skin subsided during the evening and a slight return again from 10 till 12 P. M., skin cool, tongue the same, pulse soft, no stool, slept a little, urine high colored.

Continue phosphate.

Vespere, 6 P. M., return of fever from 10 till 12 A. M., preceded by rigors did not perspire, rejects food, bowels opened.

Repeat two doses of phosphate.

28*th*.—Free from febrile uneasiness, which has not returned since last report, was restless, skin cool, pulse small, complains of giddiness, no headache, bowels opened, is weaker, urine high colored.

Continue phosphate.

29*th*.—No return of fever, perspired freely last night, slept well, skin cool and moist, pulse small, tongue clean, one dejection, feels weak, appetite better, urine free and natural.

Continue phosphate.

30*th*.—No fever, bowels opened.

Omit med.

31*st*.—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Shaik Budrodeen, D Troop, No. 803, aged 30 years, service 13 years.

Admitted 26th August 1850, 5½ A. M. Complaining of having suffered from fever for the last three days, preceded by rigors with morning exacerbations, and afternoon remissions followed by perspiration; it returned at 2 A. M. with slight rigors, which lasted half an hour, then the heat of skin followed, skin now warm but perspiring, pulse hard and frequent, tongue brown in centre with red margin, muscular pain of body, no headache, feels giddy, bowels regular, appetite bad, urine free, much thirst, states that he has not been exposed, and can assign no cause.

Rec. phosphate of lime with sulphur 2 drs. ter die.

Vespere, 6 P. M., slight warmth continued, but has began to perspire now, pulse quick, tongue very brown, two dejections, in other respects the same.

Repeat one more dose of phosphate.

27*th*.—Warmth of skin continued, did not rest well, skin perspiring, pulse soft, tongue brown, feels giddy, bowels opened.

Continue phosphate.

28*th*.—No return of fever, skin cool and moist, pulse weak, tongue cleaning, giddiness better, bowels opened.

Continue phosphate ter die.

Vespere, 6 P. M., skin hot, pulse quick 94, tongue white, two dejections.

Repeat two doses of phosphate.

29*th*.—Heat of skin continued till 8 P. M., says he slept well, skin cool, pulse soft, tongue clean, two dejections, no thirst.

Continue phosphate.

30*th*.—No return of fever, bowels opened.

Omit med.

31*st*.—No fever, functions natural.—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Karojee Row, A Troop, No. 817, aged 24 years, service 5 years.

Admitted 26th August 1850, 4 A. M. Complaining of having suffered from fever, since 3 A. M. this morning, preceded by severe rigors which lasted an hour, then the heat of skin came on, skin now hot and dry, pulse hard, tongue very brown in centre with red margin; feels giddy with heaviness of head, of which there is no pain, muscular pains of body, slight redness of conjunctiva, much thirst, appetite bad, bowels regular, urine high colored; states that he has been subject to fever since his arrival here, and never been exposed.—Can assign no cause.

Rec. phosphate of lime with sulphur 2 drs. ter die.

27*th*.—Fever continued during the night, was very restless, skin now perspiring, pulse soft, tongue brown, two dejections, feels giddy, much thirst, urine free.

Continue phosphate.

Vespere, 6 P. M., slight warmth continued till 4 P. M., did not rest well, skin perspiring, pulse quick, tongue brown, giddiness and heaviness the same, no pain of head, four dejections, appetite bad, urine free.

Repeat 2 doses of phosphate.

28*th*.—Free from fever, was restless, skin cool and moist, tongue cleaning, pulse soft, bowels opened, feels weakness and giddy, also heaviness of the head.

Continue phosphate.

Vespere, return of fever at 5 P. M., and warmth of body still continues, very restless, tongue foul, bowels opened.

Repeat 2 doses of phosphate.

29*th*.—Fever left him at 7½ P. M., did not rest well, skin now cool and moist, pulse soft, tongue foul with red margin still giddy, no appetite, two dejections, urine free.

Continue phosphate.

Vespere, 6 P. M., slight return of fever this afternoon, is now free from it, bowels opened.

Repeat 2 doses of phosphate.

30*th*.—Return of fever at 3 A. M., skin still warm and dry, bowels opened 4 times.

Continue phosphate.

Vespere, 6 P. M., after morning report the fever subsided, but returned preceded by rigors at 10, skin still hot and dry, pulse quick, bowels opened 3 times.

Repeat phosphate 2 doses.

31*st*.—Skin at present cool and moist, tongue clean, bowels opened, urine free and natural, thirst urgent.

Continue phosphate.
Rec. imperial drink.

1*st September*.—No return of fever, bowels opened, tongue clean.

Continue phosphate.

2*d*.—No fever, bowels opened.

Omit med.

3*d*.—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Mahomed Rajah, B Troop No. 463, aged 33 years, service 13 years.

Admitted 28th August 1850, 5 P. M. Complaining of having suffered from fever from 12 o'clock last night, preceded by slight rigors and left him slightly at 2 A. M., succeeded by free perspiration, but skin still rather warm, pulse soft, tongue white, slight pain all over the body, bowels regular, urine high colored, appetite bad; has been exposed to night wind, while on guard duty last night.

Rec. phosph. of lime with sulphr. 2 drs. ter die.

29*th*.—Slight heat of skin still remains, although he perspired freely, rested well, pulse quick, tongue clean, one dejection, pain of body less, urine and appetite the same.

Continue phosphate.

30*th*.—Slight heat of skin yesterday about 2 P. M., now cool, tongue clean, bowels opened.

Continue phosphate.

31*st*.—Is free from fever, tongue natural.

Continue phosphate.

1*st September*.—Well.—Discharged.

This patient had a return of fever 2 months afterwards, was treated with an emetic, purgatives and quinine, and was discharged well in 5 days, no relapse since.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Mahomed Devon, E Troop, No. 533, aged 28 years, service 11 years.

Admitted 28th August 1850, 5 P. M. Complaining of having suffered from fever since 12 A. M. preceded by slight rigors, skin perspiring, pulse quick, tongue brown with red margins, bowels regular, giddy and feels thirsty, no pain any where; can assign no cause.

Rec. phosph. of lime with sulphur 2 drs. ter die.

29*th*.—No return of fever, slept well, skin cool, pulse soft, tongue the same, states he feels well, 2 dejections.

Continue phosphate.

30*th*.—No return of fever, but complains of giddiness and nausea, tongue foul, bowels opened.

Omit phosphate
Rec. pulv. ipecac. 20 grs.
Ant. pot. tart. 1 gr. st.

31*st.*—Emetic operated well, and giddiness relieved; skin, pulse, tongue, natural.

Nihil.

1*st September.*—Well.—Discharged.

FEBRIS QUOTIDIANA INTERMITTENS.

Private Ebrahim Khan, D Troop, No. 823, aged 22 years, service 5 years.

Admitted at 7 A. M. 31st August 1850. Complaining of having had fever since 2 o'clock last night preceded by rigors, at present skin hot and dry, pulse excited, tongue foul with red margins, sallowness of conjuctiva; one dejection yesterday, no headache or giddiness, but has pains of limbs, much thirst, urine high colored, no appetite; can assign no cause.

Rec. phosph. of lime with sulph. 2 drs. ter die.

Vespere, 6 P. M. continued feverish all day now cool, tongue cleaning, bowels opened, urine free.

Continue remed.

1*st September.*—No fever, bowels confined, tongue cleaning.

Rec. pulv. jalap com. 1 dr. st.

Continue phosphate.

2*d.*—No fever, bowels opened.

Continue phosphate.

3*d.*—Improving, bowels opened.

Omit medicine.

4*th.*—Discharged.

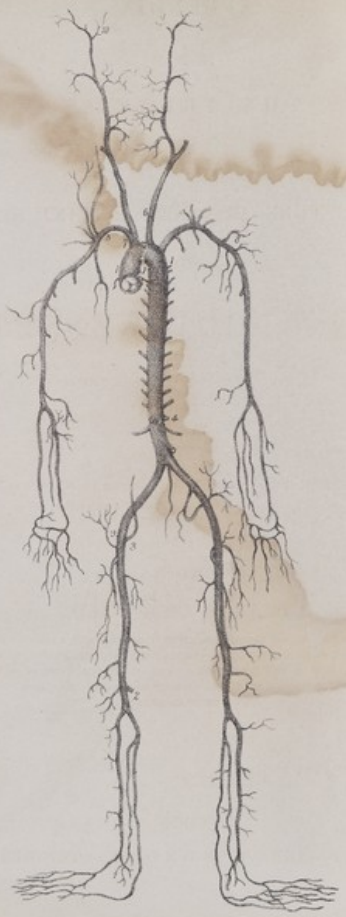
KAMPTEE, 31*st December*, 1850. } JOHN DREVER, *Surgeon, 4th L. C.*

Extract from the Annual Medical Report of Assistant Surgeon G. W. WALTER, *Tavoy, dated* 1*st April*, 1851.

In the treatment of the intermittent having an insufficient supply of cinchona, I was frequently compelled to trust entirely to the combination of phosphate of lime and sulphur, as recommended by Assistant Surgeon Blacklock, nor did I see any reason to regret this necessity, for at this station it has fully proved itself a most efficient remedy, and in the majority of the agues by which the sepoys have been affected, it has cut short the disease, in as short a time as any remedy could have done, and in a large number of the natives suffering from the same disease, it has been quite as efficacious; in a few cases after persisting in its use for some time without effect, I have substituted bark and quinine, but these cases have generally proved very obstinate even with the latter remedies, and possibly might have yielded in the same time, had the former combination been persisted in.

M. E. S. PRESS, TRICHY, MADRAS—S. P. C. COUNCIL PRINTER.

PLATE I



APPENDIX

TO

THE TREATISE

ON THE

STRUCTURE, DISEASES, AND INJURIES

OF

THE BLOOD-VESSELS.

WITH STATISTICAL DEDUCTIONS.

BEING

THE ESSAY TO WHICH THE JACKSONIAN PRIZE FOR THE YEAR 1844 WAS AWARDED BY THE ROYAL COLLEGE OF SURGEONS OF ENGLAND.

BY

EDWARDS CRISP, M.D.

PHYSICIAN TO THE METROPOLITAN DISPENSARY; MEMBER AND LATE ONE OF THE COUNCIL OF THE PATHOLOGICAL AND MEDICAL SOCIETIES OF LONDON; MEMBER AND LATE ONE OF THE PRESIDENTS OF THE PHYSICAL SOCIETY OF GUY'S HOSPITAL; AND ONE OF THE VICE-PRESIDENTS OF THE SOUTH LONDON MEDICAL SOCIETY.

LONDON:

PUBLISHED FOR THE AUTHOR BY H.TEAPE & SON, TOWER HILL.

1851.

STRUCTURE OF ARTERIES.

I have but little to add to the account of the structure of the arteries and veins. Subsequent researches have not enabled me to discover two of the coats described by Henle (the epithelial and granular); and many, whose microscopical investigations are better worthy of note, are in the same state of darkness respecting them. As regards the existence or non-existence of the epithelium, I conceive that it can be a matter of no moment; but the question of the muscularity of the arteries is one of *practical* importance: and as my desire in this Appendix will be to keep as much as possible to *practical* points, I shall endeavour to enter more fully into the subject.

In addition to my experiments and remarks on the yellow or elastic coat of the arteries, I may observe that this structure bears a greater resemblance to India rubber than to any other substance: when dried it cannot be elongated; but if folded in a cylindrical form it returns, if unconfined, to its former shape. In the dry state it is very brittle and transparent. On holding it to the light the fibres may be seen passing in a circular direction. After being kept dry for many years, and then immersed in water for a short time, it will resume its natural elasticity. The artery I am experimenting upon has been in my museum for six years.

The cellular coat, when extended and dried, resembles in appearance the web of some species of spider, or the cacoon of the silkworm. It is extremely tough, and from its reticulated and felt-like tissue, it is capable of great extension; but it has not, like the elastic coat, when distended beyond a certain point, the power of contraction. It is in this coat I believe (as many have before supposed) that the tonicity of an artery is seated. I have nothing to add here to my former description of the serous or subserous tunics, nor to the account of the structure of the veins; but in the pathological part of the Appendix I shall have occasion to allude to the structure * of the serous membrane.

If I were disposed to quote the various opinions upon the arteries which have been broached, even during the short interval that has elapsed since my former description of these structures was written, I could add largely to this treatise, but it would lead to no practical good. Mr. Guthrie, in a lecture at the Medical Society of London (Lancet, Feb., 1851), which should have been called Mr. Quekett's Letsomian Lecture, has described, like Henle, six arterial tunics, although in his work on the Diseases and Injuries of the Arteries, 1831, he enumerates only *three*. In his subsequent treatise, in 1846, he keeps to the same number; and he describes the cellular coat as *especially elastic and retractile* (page 8). I mention the circumstance because I consider this point to be one of great *practical* importance. I believe I was the first to describe the cellular coat as nearly *inelastic*; and it is the want of retractility, as I have stated (pages 7 and 293), in this tunic, that forms the greatest safeguard when an artery is torn or divided. The serous and yellow coats retract, the cellular (in torn arteries) is elongated, and by this means forms a kind of funnel-shaped covering to the mouth of the vessel. The arrest of hæmorrhage by torsion, I believe, is due to the same cause. Mr. B. Cooper, in his Surgical Lectures, recently delivered at Guy's Hospital, and published

* I can now refer the reader, in the College Museum, to two of the 21 preparations which accompanied the Essay, and which show the structure of the arteries. They are between specimens 942 and 943, but without name or number. See letter to the Council, vol. 1, Appendix, p. 12, of the "Examiner."

in the "Medical Gazette, 1849," (p. 881), speaks of this coat as the "cellular or elastic coat;" and, he says, "if an artery be much stretched, or tightly compressed, by ligature, its middle and internal coats give way—the external coat, from its superior elasticity, may again recover its normal condition." I scarcely need comment on the absurdity of this statement.

Mr. Wharton Jones, who has lately gained the Astley Cooper Prize of £300, on the state of the blood and blood-vessels in inflammation, describes the arteries of the frog's foot (upon which his experiments were made) as composed of three coats, and plates are given to show the middle or muscular coat in a state of contraction. Mr. Jones, in speaking of one of his experiments, says, "From the cessation of the flow of blood in the vessel, and the transparency of its coats, the artery may for the time not be distinguished from the surrounding structures." But it is difficult to understand, I think, how a microscopist can accurately distinguish the structure of the coats of arteries in the tubes of which the average diameter does not exceed 1-1000 of an inch.

Mr. Quekett, whose patient researches and laborious investigations deservedly give weight to his opinions, believes also in the muscularity of the arteries; but I think that the majority of physiologists are opposed to Mr. Quekett upon this point. When in Edinburgh, in 1848, Dr. Hughes Bennett was kind enough to invite me to a lecture on the Structure of the Arteries, and he, like Mr. Quekett, described six tunics; but he admitted that he had only seen four of them. Dr. Bennett concluded his lecture by the astounding assertion, "that Hunter's doctrine of the muscularity of the arteries, and Cullen's notion of spasm of the extreme vessels, were correct;" but to my eyes, the very evidence that Dr. Bennett's microscopes were supposed to afford of the muscularity of the arteries was conclusive of their non-muscularity. When obstruction took place in the capillaries of the frog's foot, the *vis a tergo* being the same, the vessels appeared to be more active; but it was the action of the heart (I believe), and not the muscular contraction of the capillaries. The tied and throbbing arteries of a stump, seen after amputation, are surely not self-acting; the blood is propelled against their extremities, and hence the reaction and throbbing. Let us test the doctrine of the muscularity of the arteries by common sense, an ingredient, I think, that is often wanting in microscopical deductions, both physiological and pathological.

We are told by some microscopists that the involuntary muscles of the intestines, and that of the arteries, present the same appearance under the microscope. I find, however, that the smallest fibre of this supposed muscular coat of the artery is highly elastic, and that it will retain its elasticity for many months if kept moist, but the muscular coat of the intestine scarcely possesses any elastic property; am I then justified in subscribing to this microscopical dogma of identity of structure? Again observe the peristaltic movements of the involuntary fibres of the intestines, and what analogy is there between their action and the pulsatory movement of the arteries? But endeavour to bring arterial muscularity to bear on the circulation of the blood, in any way you please, and you stop the current. Muscularity and elasticity are incompatible; they could not, I believe, act together in a cylindrical tube. It is true that the *ligamentum nuchæ*, which has a structure identical with the yellow coat of the arteries, is entirely passive, and influenced solely by the muscles; but this combination of action surely bears no resemblance to the supposed musculo-elasticity of the arteries.

Another insurmountable difficulty to the advocates of the muscularity of the arteries, is the absence of fibrine in the supposed muscular coat, and the want of contraction on the application of galvanism. Contraction is said to have been produced on the smaller arteries, by the use of this agent, by Van den Bosch, and Rossi: Müller, Weber, and others have tried it on the larger tubes without effect; but the Webers have since succeeded in reducing the mesenteric arteries of the frog to one-third of their diameter (Müller's Archiv, 1847, No. 2). But I need not remind the reader that *all* experiments made upon such minute vessels must be inconclusive, and that *contractility*, does not prove *muscularity*.

What aid does pathology afford to the advocates of muscularity? Blushing say they (which is a momentary inflammation of the cheek and neck), must arise from increased action of the vessels of the part, and, as far as I know, this argument has never been properly combated. But did any person I ask, ever blush without the heart's action being instantly increased? Again, the small vessels and capillaries of the cheek differ from those in every other part of the body. In some persons they are always distended, in others, no red blood is perceptible in them; and after a certain age, when they lose their elasticity, blushing is not seen. It is also probable, as I shall have occasion to mention hereafter, that the *tone* of these and all other vessels depends upon the supply of nervous fluid to the outer or cellular coat affecting their tonicity, and consequently their caliber. Inflammation, the great disorganizer of structure according to the language of the schools, arises from increased action of the vessels of the part, but I believe, (as I stated at the Medical Society of London, ten years ago), that inflammation is essentially a process of stagnation, a stoppage of the blood corpuscles in some of the capillaries, and this supposed increased muscular action of the vessels arises from the same cause that produces the throbbing of the arteries of the stump. The force of the heart is the same, but the blood does not find its way through the same channels, and the opinion of Hunter, that more blood passes through an inflamed part, I believe to be untenable. *The surrounding vessels are more distended, and probably convey a larger amount of blood; not so the inflamed capillaries.*

As regards the supply of nerves and blood vessels to the arterial tunics, these are questions I think that will be more advantageously discussed, when the pathology of these structures is further investigated; and as I proceed with the structural changes in the blood vessels, I shall endeavour to illustrate their physiology.

MORBID CONDITIONS OF ARTERIES.

In addition to the observations I have already made respecting the morbid conditions of arteries (page 8), there is one subject that especially requires further notice, viz.: what is called fatty degeneration, a change that is supposed by many to be essentially connected with the atheromatous and bony deposits. I have, however, (page 71), come to the conclusion that this oily and fatty condition in old age, rather serves to prevent the preternatural dryness of the arterial tunics; an opinion formed by deductions from a large number of cases of aneurism, which clearly show, that as the fatty or earthy deposits increase, so does the tendency to the formation of aneurism diminish. This doctrine will be considered heterodox and untenable by those who look to fatty degeneration as the cause of many fatal pathological changes. But let us enquire farther into the subject, and we shall find, I think,

that although a fatty condition of the liver and other organs is very common, it is generally the result of deranged structure and impaired function of the lungs and other parts, when occurring at an early period of life. But this condition must not be confounded with what is called atheromatous, or fatty deposit in the arteries. The one has little or no connexion with the other. In old age there is a *general* deposition of fat—in the cases alluded to it is only *partial*. All we know of fatty deposits, both in healthy and diseased structures, tends to confirm the opinion, that these depositions are the result of a slow or impeded circulation; accompanied *generally* with some alteration in the constituent parts of the blood. The animal that is kept at rest, with its circulation in a quiet state, fattens more quickly than the one that takes exercise. The fatty degeneration of the muscular structure of the heart I believe is almost invariably produced by ossification of the coronary arteries. The *arcus senilis* by the partial closure and drying up of the capillary vessels. I have before stated, no example can be adduced of the conversion of fat into ossific matter, but abundant evidence is afforded of the former deposit being the result of a bony and inelastic condition of the arterial tubes. It will naturally be said, that we have positive evidence that fatty degeneration is the cause of ruptured heart. But this lesion is very rare, and probably in 500,000 fatty hearts, it would not be met with in more than one, and the same remark will apply to fatty arteries. These are exceptions, not rules.

But what reasonable proof can be adduced that these deposits, whether fatty, bony, or atheromatous, are often the means of prolonging life, and of keeping up that balance in the animal machine so that one wheel may keep pace with another? Human bodies, like all living structures, are formed to last for a definite period; and it is a law of nature that certain changes, tending to decay and death, should take place in these bodies, which, like inanimate matter, are influenced to a great extent by external agents that hasten their decomposition and destruction. It is, I think, taking a very superficial view of this question to regard the arteries alone; and there is not, probably, a greater error committed by pathologists than that of overlooking the fact that, what is often called *disease*, is a salutary disorganization to regulate functional balance, and to maintain that equality of power so essential to the proper performance of the various functions of the body. The elastic artery would ill accord with the worn-out heart—the thin and flabby ventricle with the diseased valve—the strong and healthy stomach with the sluggish and diseased liver—acuteness of vision with dulness of intellect—the ossified artery with the dry and oilless muscle. It is not improbable, moreover, that this increase of fat and oil in the bodies of the aged serves to keep up the temperature, and by that means also, to prolong life.

I have recently had an opportunity of examining the bodies of two persons (in the same week), that afforded a contrast that is seldom met with. The one with Mr. Webber, of Connaught Square, the other with Mr. C. Taylor, of Camberwell. The first was a young gentleman, æt. 19, who was found dead in his bed. He had been for several years labouring under palpitation of the heart; a quick irregular pulse, generally too rapid to count; purring tremor, and increased impulse, so that his shirt-collars were moved by the heart's propulsion. These symptoms were accompanied with dyspepsia. Mr. Webber and others thought there was hypertrophy of the walls of the heart, whilst some who saw him supposed the derangement only functional. The peri-

cardium contained about 2 oz. of serum. The parietes of the heart were generally thickened; and this organ probably weighed about 16 oz. The left ventricle was so dilated that its cavity would hold three hen's eggs; the curtains of the mitral valve, and the carnæ columnæ, greatly elongated and enlarged. But the valve, in consequence of the increased size of the auricular-ventricular cavities, only partly performed its office, and the column of blood lost its natural support; and from this disproportion of parts arose the cardiac disturbance, and the fatal termination. There was no fat in the muscular structure of the heart; the arteries and all the organs examined were perfectly healthy. There was not a trace of *disease* found; and the brain, which unfortunately was not examined, is scarcely likely to have been in a diseased state.

The other case was that of an old lady, æt. 88, whom I saw with Mr. Taylor, a few days before the autopsy. She was unmarried, very tall, had generally enjoyed good health, and was descended from long-lived parents. Mr. Taylor was called to her in November last, when she had a large deep ulcer on the left leg, which had existed for several years. Early in January the lower part of the wound began to slough, and gangrene of the foot ensued. A line of demarcation was formed; and in February, spontaneous amputation of the foot, at the ancle-joint, took place. There were also gangrenous sloughs over the sacrum, and on the knee of the affected side. She died on the 5th of March from exhaustion. It is remarkable that during this extensive sloughing of the soft parts, and separation of the bones of the foot, that she never complained of pain. It must be observed, however, that the case was not one of dry gangrene and arteritis.

The external surface of the body (seen at the autopsy 12 hours after death) presented nothing remarkable. The integuments were flabby, and covered a layer of fat. This substance appeared to pervade nearly every tissue of the body. The arteries of the lower extremities were imbedded in it; and their coats, as well as those of the arteries of the chest and abdomen, were saturated with an oily fluid. The heart weighed about 9 oz.; the sides of the left ventricle were slightly increased in bulk; and the muscular structure generally fatty. Both coronary arteries were ossified. The semilunar aortic valves were free from deposit; but they were thick, dry, and parchment-like, and stood out, so that their convex edges approximated. The thoracic aorta contained a few patches only of bony deposit; in the abdominal aorta, especially near its bifurcation, these were more abundant, and several bony plates were present in the external and internal iliacs. The femoral artery was nearly free from deposit, as were also the anterior and posterior tibial; but in the former artery, when dry, ossific granules were seen. The pulmonary artery, the venæ cavæ, as well as the femoral and saphena veins, presented no trace of deposit, and none could be felt (through the skin) in the radial arteries. The valves of the saphena and femoral veins were dry, and adherent to the lining membrane; they had entirely lost their office. Along the course of the posterior tibial arteries were several firm bodies, about the size of large peas, with two curled tapering extremities. These were composed of blood corpuscles, and lymph; and they were enclosed in distinct fibrous cysts. I carefully examined the tarsal, and internal and external plantar arteries of the gangrenous foot; their coats, like the surrounding structures, were of a dark colour, but I could discover no disease in them.

Of the two individuals above-mentioned, the arteries of the former resembled the young and elastic sapling; whilst those of the latter may be compared with the dry and withered branches of the sear-oak. Life's time-piece—the heart—in the youth,* perhaps, was congenitally mal-formed; or the harmony of its structure deranged by mental or bodily efforts, undertaken before the organ had attained its full development and strength. The old lady, on the other hand, with a constitution originally good, and in her sphere of life less exposed to intellectual and physical evils, lived to a ripe old age; and, without speaking irreverently, rotted long before the vital principle had left her body. In the one instance the thread of life was early snapped by a want of equipoise in the heart's mechanism; in the other, nature's balance was kept up in all the organs of the body. There was no disproportion of power; and the rusty and worn-out machinery was well lubricated with oil and fat. Clog-wheels and safety-valves existed in the arteries to diminish their elasticity and force, so as to render their power commensurate with the supply of blood required by the secretory apparatus. These two cases, if *well studied*, would afford *practical* matter for a larger volume than has yet been written on the physiology and pathology of the blood vessels.

I am far from asserting that a fatty condition of the heart may not be a more frequent cause of death than has hitherto been supposed; indeed, we have positive proof that in the vast majority of cases of rupture of this organ, that there is fatty degeneration of the muscular fibre. When I showed the specimen of rupture of the left ventricle (*Examiner*, vol. I., p. 160), at the London Pathological Society, November, 1846, I stated (*Transactions*, p. 62), "that from the examination of a few specimens I had made, of spontaneous rupture of the heart, I thought that fatty degeneration was generally present in these cases." Dr. R. Quain, in his interesting paper on "Fatty Diseases of the Heart" (*Med. Chi. Trans.*, 1850), has gone fully into the subject of this degeneration, which, no doubt, in many cases, leads to premature dissolution. But the question I ask is, not whether this fatty degeneration in the heart and arteries is the immediate cause of death in some cases, but whether it does not *generally*, in the aged, tend to prolong life, by adapting the frequency and force of the heart to the requirements of the system? In the case above alluded to, the gentleman was 73 years of age, and had enjoyed good health, but he died of rupture of the heart from fatty degeneration. Who can say that this senile change did not prolong life? that a vessel of the brain might not have given way at an early period, if the heart had been strong and vigorous? In the case before-mentioned (p. 71), of rupture of the heart from fatty degeneration, the patient was 90 years of age, and a table-spoonful of limpid oil flowed from the vena cava.

* Since the above was written I have learnt from Mr. Webber a circumstance that adds much to the interest of this case. When about four years of age, the child's nurse held him over a well, and threatened to throw him in. After this he had, for a long time, such a dread of water that, when travelling with his father and mother in a carriage, he would get out and cross the foot-bridge rather than pass through the stream in the carriage. This fright is supposed by his friends to have occasioned the heart affection.

The history of the examination of Old Parr (*Phil. Trans.*, vol. 3), who lived to the age of 152, is a very interesting one. It is doubtful whether Harvey was present at the examination of the body, although the account is said to have been written by him. The arteries * are not mentioned, but the heart was great, thick, fibrous, and fat; the kidneys covered with fat, and the cartilages of the sternum, flexile and soft. Unfortunately the body of Henry Jenkins, who died at the advanced age of 169, was not examined. In Dr. Keil's account (*Phil. Trans.*, 1704, p. 2247) of the dissection of John Bayles, the button-maker, of Northampton, who died at the age of 130, the heart is said to have been large, thick, and fat; the diameter of the thoracic aorta, near the carotids, was two inches. The abdominal aorta, and the iliac arteries, were for the most part *cartilaginous*. There was also a small ossification of the dura mater. Dr. Keil attributes his death to thinness of the stomach and hardness of the aorta. These arteries, probably, when dry, would have formed bony tubes.

I have lately been occupied in the examination of the radial arteries of old people; and as these branches are probably less subject to ossification than any of the arteries of the limbs, we may infer that when the ossific deposit is present in them, that it exists to a greater extent in the larger arteries. In speaking of ossification of the radial arteries, it must be understood that I have never found these tubes converted into *cylinders* of bone in the living subject, although when dried, after death, they would often present this appearance. The deposit is in rings, or patches, which are situated at a greater or less distance from each other. Sometimes only one artery is ossified; and the right, I believe, is more frequently affected than the left.

Through the kindness of Mr. Lowne, Surgeon to the Walworth Poor-house, I have examined the radial arteries of eight men and ten women. The ages of the former amounted to 70, 73, 75, 78, 80, 84, 84, 90; of the latter to 75, 75, 75, 78, 79, 79, 83, 84, 85, 88. In the eight men the radial arteries were bony in three, thickened in two, and apparently healthy in three. Amongst the ten women two had slight ossific deposit, in two both arteries were thickened, in one the right only, and in six the arteries appeared to be healthy. Two of these eighteen persons had these arteries tortuous.

My friend, Dr. Bainbridge, who is Surgeon to the large Poor-house of St. Martin's in the Fields, has permitted me to examine the aged paupers in that establishment. Of sixteen men, whose ages were 70, 71, 72, 74, 76, 77, 78, 78, 78, 79, 80, 80, 83, 84, 86, 87. There was ossific deposit in three: these arteries were more or less thickened in eight; apparently healthy in five, and in three they were tortuous.

* I believe I have seen a statement somewhere, that the arteries of Parr were converted into bone; but as these vessels were not noticed at the autopsy, it is not unlikely that I have confounded the case of Parr with that of some other person of advanced age. If the radial arteries of Parr were bony, it is most likely that the fact would have been noticed by some of those who felt his pulse, as he must have been an object of great curiosity to members of the medical profession. I have in my possession a copy of *Hodgson on the Arteries*, which belonged to the late Mr. Langstaff. It contains several of his manuscript notes. He says, "I have seen the radial arteries in two subjects converted into bony tubes."

The ages of the thirty-two women examined were 70, 72, 73, 73, 74, 74, 75, 75, 75, 76, 76, 76, 77, 77, 77, 77, 77, 78, 79, 80, 80, 80, 80, 80, 82, 84, 85, 86, 87, 88, 89, 90. Three of these had ossific deposit in the radial arteries to a slight extent; in fourteen the tubes were more or less thickened, and in the remaining fifteen, they appeared to be free from disease. In three of the above the arteries were tortuous.

Dr. Beith, one of the Surgeons of Greenwich Hospital, allowed me the privilege of examining the old sailors in that institution, and I probably felt the radial arteries of more than a hundred men beyond seventy years of age, and their conditions varied as in the above instances. I select a few of the cases, C. R. æt. 86, both arteries bony. C. A. æt. 80, both arteries bony: has been subject to gout, and has taken mercury several times. W. A. æt. 89, both arteries ossified and tortuous. W. D. æt. 72, a little deposit on both arteries. D. G. æt. 86, the arteries in the same state as the last. R. G. æt. 72, both arteries bony. In four others both the tubes were a little ossified, and in two, the deposit was confined to the right artery. In one man, æt. 94, both the arteries were sound, and in another, who was 93 years of age, the left was bony. W. Strickley, æt. 103, had the arteries free from deposit.

As regards the *arcus senilis*, it was more or less developed in all; but I am unable to make out that its size in old people is an indication of debility or want of power in the circulating organ. A physician, who has now attained his 82nd year, who is well known to the profession, and is one of the healthiest men of his age I have ever met with, has the arcus developed to a great extent. Its early appearance in the young, as supposed by Mr. Canton, and Drs. Williams and Quain, may be indicative of a fatty state of heart, but at present, I think the evidence is inconclusive.

I could add largely to the Table of 102 inspections, (p. 11,) I have recorded of the arteries of the chest and abdomen, but this addition I find would serve no useful purpose. I see no reason to alter the opinion I have before expressed respecting the origin of arterial deposits, (p. 67,) viz., that they are generally occasioned by congestion or inflammation of the capillary vessels or channels that convey the serous part of the blood, and that they are seated under the inner coat of the artery, and not upon it, as Rokitansky and others have asserted. That these deposits are more frequent in persons of advanced age, and in those who have the cutaneous transpiration diminished or suppressed. That individuals of scrofulous or gouty diathesis, and those who labour under granular disease of the kidney, are *probably* more liable to them, and that the bony depositions are more commonly met with in the strong and vigorous who attain a great age.

As regards the effect of mercury in the production of arterial disease, I may mention in addition to the evidence adduced at page 116, that I have now a man under my care at the Dispensary, æt. 51, who labours under partial paralysis of both hands, and mercurial trembling: his habits have been intemperate: at the age of fourteen he was apprenticed to a looking-glass gilder, and after a few years he got into his present state, and has remained so ever since: both radial arteries, judging by the finger, are free from disease, but there is a rough sound over the aortic valves. In my investigations respecting the condition

of the radial arteries of old people, above-mentioned, I endeavoured to ascertain whether they took mercury at any period of their lives: many were unable to answer the question, but several who had reached a great age had been salivated by this mineral, although these formed, especially amongst the women, a small proportion. I think we possess no evidence at present to show, that arterial deposits owe their origin to the administration of mercury; but it is more than probable that alchohol, both by its influence upon the blood, as well as its tendency to increase mental and bodily effort, is the most powerful agent, in cold and temperate climates, in the production of these changes in the arteries. As I have observed before, it is important to distinguish between the atheromatous deposits found sometimes in early life, and the bony inelastic, and fatty impregnation of the arterial tubes generally met with in old people. I may also repeat, that the atheromatous and ossific patches are not essentially fatty, for, as I have remarked, (p. 79,) the cholesterine and fat are much less abundant than the other constituents.

There is one condition of the radial arteries that I believe has not been alluded to by any previous writer, viz., the tortuous and zigzag state of the tube. The coats of the artery in all the cases I have seen have been thickened, and most of the patients have laboured under hypertrophy of the left cardiac ventricle. The meandering stream often seen flowing from angle to angle, through the meadow, is a good representation of this change in the blood-current, and, on watching the artery, the impulse is chiefly perceptible at the curve. I will mention briefly two or three cases among several that I have lately met with at the Metropolitan Dispensary. M. B. æt. 62, has both the radial arteries curved in the manner described: she has had several attacks of inflammation, for which she has taken mercury to salivation: has also suffered from great mental anxiety. Palpitation of the heart has existed for several years, and the ventricular impulse is now greater than natural, but there is no abnormal sound.—Mrs. F. æt. 59, has the same condition of artery, and it exists to a much greater extent in an old lady 81 years of age. Both the last patients are suffering from bronchitis, and in both, the impulse of the heart is increased, and the first sound prolonged.

One of the best collections I have seen of diseased arteries is in the Chatham Museum; and those who are interested in these lesions will find numerous specimens. The London Hunterian Museum also contains some curious preparations of this kind. Those numbered 1597, 1607, 1608, 1611, 1612, 1613, 1614, 1617, 1618, 1619, 1625, 1627, 1628, 1629, are good examples of these changes, but unfortunately the histories of these cases are but imperfectly recorded. The Museums of the Colleges of Surgeons of Dublin and Edinburgh are also rich in these preparations. In the latter museum the specimens 1184, 1185, 1180, 1181, 1174, are well worthy of notice. In preparation 1174, the ossific deposit appears to me (as in case 41, p. 14, and plate 4, f. 9) to have arrested the progress of dilatation of the arterial walls.

Ossification of the Pericardium.

At page 73 I have alluded to ossification of other parts, for the purpose of showing the analogy between the isolated deposits of bone

in the arteries, and in other organs. Occasionally a portion of the abdominal aorta only is found ossified, the thoracic being free from disease, and *vice versa*. Before I quit this subject I am induced to mention the following example of ossification of the pericardium, which I saw with Mr. Skegg, of St. Martin's Lane, in July, 1850, because I believe, considering the age of the patient, it is *unique*. Mr. Skegg has been kind enough to furnish me with the subjoined outlines of the case.

"E. B., æt. 11 years, in February, 1844, became the subject of a severe attack of acute rheumatism. A fortnight from the commencement of the attack, pericarditis supervened, which was treated by leeching, blistering, mercury to ptyalism, colchicum, &c. The following May the medical attendance ceased. Auscultation evidenced fibrinous deposit in the pericardial cavity; turbulent cardiac action, with dyspnœa on increased exertion. In all other respects he enjoyed tolerable health, until November, 1845, when pain in the region of the heart required antiphlogistic treatment and blistering for a short time. September, 1846: he became the subject of a severe attack of hæmoptysis, which, being subdued, was followed by ascites, which was with much difficulty removed by diuretics. In June, 1847, ascites re-appeared, but was again removed by diuretics. In September, 1848, he once more became the subject of dropsy, which lasted several weeks. Again, at the close of 1849, ascites re-appeared, accompanied by a severe attack of hæmoptysis, which for several days threatened his existence, but was ultimately stayed by the gallic acid and lead. His sufferings during the last three years had been very severe; from this date (November, 1849) they were dreadfully so, frequently passing several successive nights in a chair. Great abdominal distension, attended at times with severe pain in the epigastric region, and œdema of the lower extremities, and infiltration of the scrotum, enlarging it to the size of an infant's head. Punctures with a needle afforded some relief. A more severe attack of hæmoptysis took place in July, which lasted ten days. It now became evident that the manifold and long-continued sufferings of the patient were drawing to a close. The dyspnœa became painfully urgent; the ascites was no longer amenable to medicines; every known diuretic failed to act on the kidneys; the violent action of the heart, to use his own words, 'appeared as if it would burst through his chest,' prevented his sleeping. Death was earnestly prayed for by the sufferer, who died rather suddenly on the 11th of September, 1850. Repeated auscultation led to the conclusion that pericardial adhesion existed, with hypertrophy of the heart. Some disease of the valves was suspected. During the whole of his illness the pulse maintained a strong, full, and rapid beat, but perfectly regular, which lessened the suspicion of valvular disease. The carotids and cerebral arteries beat violently; and this palpitation was the cause of much distress to him. Bleeding from the arm was more than once tried; but the patient, declaring himself worse from the operation, it was not allowed to be repeated. A dry cough repeatedly harrassed him, more especially towards the close of his existence. Notwithstanding his sufferings he grew in stature, being at the time of his death six feet high.

Autopsy. By Messrs. Canton & Skegg, 36 hours p. m. The

pectoral and abdominal muscles well developed, a moderate quantity of adipose substance covering them. On attempting to raise the sternum, after dividing the costal cartilages, and dissecting off the attachment of the diaphragm, the separation was found to be impracticable, being firmly bound down by a thick fibrinous membrane, which required dissection from the entire posterior surface of the sternum. The fibrinous membrane was firmly attached to the pericardium, and the thoracic fascia covering the large vessels, suggested the idea of an hypertrophied thymus gland.

Lungs. The anterior margins adherent to the pericardium and their outer surface so firmly adherent to the pleura costalis, as to defy other means than the scalpel for their separation. The thoracic fascia pericardial covering of the great vessels amazingly thickened. Recent lymph on and between the folds of the intestines. The *omental margins* curiously turned up. The liver hypertrophied, apparently from increase of Glisson's capsule: its anatomical characters as to shape, destroyed; no acute margins; rhomboid in form, and firmly adherent to the diaphragm.

Kidneys rather larger than normal, but free from disease. *Spleen* much enlarged, and firmly adherent. *Bladder* extending nearly to the umbilicus; adherent to peritoneum, and curiously elongated in consequence of the adhesions."

I was unable to attend the post-mortem examination, in consequence of absence from town, but I was present at the Pathological Society, when Mr. Canton exhibited the heart and pericardium, and through his kindness, I have since been enabled to make a more careful inspection of the parts.

The heart weighs 14 ozs. The left ventricle dilated, and its walls much thickened; the curtains of the mitral valve also thickened. The pericardium universally adherent to the heart, and that portion of it over the mitral and tricuspid valves is converted into a thick bony ring, which entirely surrounds the heart; it is $10\frac{1}{2}$ inches long, and varies in breadth from one to two inches; it is firmly united to the cardiac pericardium, by means of false membrane; the aorta of a natural size, and free from deposit. Other parts normal.

When I saw this patient with Mr. Skegg, I supposed with him, that there was great hypertrophy of the left ventricle of the heart, and disease of the aortic valves, the result of previous inflammation and of inordinate action. The *bruit de rape* was heard over the upper part of the heart, but no purring-tremor was present. The file-sound was probably occasioned by the bony deposit.

The peculiarity of the above case is the age of the patient, and I am not acquainted with any other instance of ossification of this membrane at so early a period of life. Morgagni in his 22nd Letter, c. 10, relates the case of a man æt. 30, who, after fever, had oppression at the chest, with general œdema. The pericardium was universally adherent to the heart, and in some parts it was cartilaginous. Seiler, in his *Anatomiæ corporis humani senilis specimen*, 1749, p. 92, refers to cases of ossification of the pericardium in old people, mentioned by Haller, J. G. Walter, and Bonetus. Otto, in his Compendium of Pathological Anatomy, (South's Translation, p. 257,) alludes to instances related by Sénac, Saviard, Aurivillius, Pasta, Prost, and Rayer. I have seen a good specimen in the Museum of the late Mr. Langstaff, but no history was attached to it; it is now in the Hunterian Musem, p. 1516. There is another in the St. George's Hospital Museum. In the Museum of the College of Surgeons at Edinburgh, prep: 1005, shows ossification of the pericardium over the right ventricle. In the Chatham Museum there are two specimens, (62, 63,) in one the ossific matter is three inches in length, and a quarter of an inch in thickness; in the other, it nearly surrounds both auricles. *M. Verneuil, Gazette Medicale, de Paris,* 1850, *p.* 557, relates a case of ossification of the endocardium, situated over the border of the right auricle. There was also rupture of the auricle. The woman, æt. 70, had had acute pericarditis at a former period. In the Glasgow Hunterian Museum, preparation 66, shows a large ossification in the substance of the heart round the mouth of the left ventricle, from a fat woman, in whom the arteries of the uterus and brain were also ossified.

Arteritis.

There is scarcely a subject connected with pathology that is less understood or studied than arteritis, and I am compelled to make the same remark as I did when treating of this disease before; that the morbid conditions of arteries are but little attended to, and in the great majority of post mortem examinations these vessels are not inspected: the upper part of the aorta is sometimes examined, but the descending thoracic and abdominal portions, as well as the larger arteries of the limbs, are rarely looked at. I could adduce many examples of gangrene of the extremities where the arteries have been superficially examined near to the gangrenous part, but the tubes some way above the mortification, where the plug is often seated, were unnoticed. In the living body what valuable assistance may we derive from the careful examination of the arterial and venous trunks? How many thousands of legs and arms have been amputated, and the surgeon has not taken the trouble, nor considered the importance of examining the femoral or brachial arteries before the operation? What a useful lesson does the case of gangrene of the leg, recorded in the first volume of the Examiner, (p. 152,) teach us. Both femoral arteries are plugged: a surgeon cuts off the leg; and the patient dies in three days with a sphacelated stump; the almost certain result of amputation performed under such circumstances. I am not acquainted with a surgical writer who has especially inculcated the necessity of a careful examination of the arteries before amputation of the limbs, and I believe that I have seen fifty legs removed without this inspection having been made in a single instance. Again, in all affections of the brain, how important is it to investigate the state of the carotids; to observe their force; to ascertain whether their power is equal, and whether the pain in the head, or the singing noise in the ears, is removed by the compression of one or both these vessels. In aneurism of the arch of the aorta how necessary is it to note the state of the vessels of the neck, and the condition of the arteries of the arm; if there is a want of correspondence between them, a mechanical obstruction may be suspected, for, *I believe there is no well-authenticated case on record, of the local absence of pulsation in an artery, without a mechanical cause.*

I have been induced to hazard the above remarks, believing that this mode of diagnosis is one that has hitherto been much neglected.

As my object in this, and in the subsequent chapters of the Appendix, will be to include only such matter as I think practically useful, I need not again enter into the causes, diagnosis, and treatment of arteritis, especially as I have but little to add to the opinions already expressed.

Aortitis.—As regards the diagnosis of this disease, I confess myself as much in the dark as ever respecting it; nor do I belive that there are any positive signs by which it can be distinguished. Gendrin, in the *Gazette Médicale des Hopitaux*, Paris, 1850, has published some interesting papers on arteritis, which he divides into three forms:—1. That in which all the tunics are more or less affected; 2. The valvular; 3. The aneurismal. In the acute arteritis affecting the larger vessels, especially the aorta, he describes a tumultuous action of the heart; a small pulse; and a disposition to syncope, as the most prominent symptoms. He relates the case of a man under his care, who died of aortitis; fibrinous concretions adhered to the semi-lunar valves, and to the lining membrane of the aorta.

Professor Virchow (*L'Union Médicale*, Paris, 1850) has instituted several experiments on the arteries of dogs, by irritating them in various ways; and in no instance did he produce exudation of lymph from the serous membrane of the artery. He concludes that the fibrinous deposits are from the blood within the vessel, and that they are often formed in or near the heart; then carried into the smaller trunks, where they plug up the tubes, and by arresting the circulation produce gangrene. I am not prepared to deny that lymph may not sometimes be transposed in the manner described; but I suspect the circumstance is of rare occurrence, especially in the larger arteries; and nature's laws are too perfect to allow of such chances of obstruction to the blood current. What a wise and beautiful provision is it that pus, which courses through the veins with deadly and poisonous effect, is not formed in the arteries. How common are deposits of fibrin about the heart in acute rheumatism, in young subjects; but how rarely are these patients affected with gangrene. In the fourteen cases of gangrene of the extremities that I have related (p. 66), the coats of the arteries were often intensely red; the clot sometimes firmly adherent to the sides of the vessel, and never wedged in, as described by Professor Virchow. As regards the exudation of lymph, we have indisputable evidence, after the application of a ligature, or after violence done to the tube, of the deposit of fibrin, thickening of the middle and cellular coats; and the conversion of the artery into a ligamentous cord. These phenomena could not be effected without the presence of inflammation; and there is no reason, that I can discover, why this process should not occur spontaneously in arteries as well as in other structures. Whether lymph is *first* poured out from the serous membrane of the artery, is not, I think, a question of importance, so that we know that inflammation produces fibrinous coagula, and obstruction or obliteration of the tube.

The foregoing remarks it will be perceived are not irrelevant to the disease in question, viz., *aortitis*, about which so much difference of opinion prevails. The symptoms must depend in a great measure on the seat of the inflammation; its extent and acuteness of character. If the inflammatory action is confined to the aortic valves and endocardium, the rapidity of circulation orthopnœa, and disposition to syncope will probably be greater than when the lower part of the vessel is affected: the presence of previous disease, such as, atheromatous and bony deposit, may also add to the severity of the symptoms. The composition of the blood, too, may exercise some influence on the nature of the inflammation, and in those cases where the aorta and the larger arteries are generally inflamed and reddened, it is probable that the fibrin is less in quantity, and the plasticity of the blood diminished; much, however, connected with this affection, as I have said before, is conjectural and speculative.

I have not met with a well-marked example of acute inflammation of the aorta since I last wrote upon this subject, although the subjoined cases, by some may be considered to come under this head, and several instances of supposed aortitis have been published, whose authors have not had better evidence of the existence of the disease than these afford. Mr. B. Evans, of Trinity-square, allowed me the opportunity of seeing the following case in December, 1848. Mr. A. æt. 46, a warehouseman, had generally enjoyed good health until November, 1847, when in ascending some steps he found his breath short and quick, and after this he was affected frequently with palpitation of the heart and dyspnœa; he consulted Mr. Evans, and when I saw him with this gentleman, he had loud bellows' sound over the base of the heart and strong ventricular impulse. The pulse was intermitting at every twentieth beat. These symptoms continued, with occasional mitigation, until January, 1850, when he consulted a physician, who gave him very large doses of Iodide of Potassium, Sulphuric Ether, and Prussic Acid: he called upon Mr. Evans three weeks before his death, and told him "that the medicine had had such an extraordinary effect that he considered himself quite well." The same sound, however, was present over the region of the heart. A fortnight after his visit, Mr. Evans was called to him in great haste, in the night, and found him apparently dying. The difficulty of breathing, and orthopnœa, were extreme. Blood was taken from his arm, to relieve the oppressed lungs—ether administered at intervals; and to the surprise of Mr. Evans he rallied, and the next day he appeared much better. A week after this attack he died rather suddenly. I examined the body, with Mr. Evans, on the following day (February 18th).

The muscular development greater than usual. On opening the chest the cellular tissue around the root of the aorta was seen distended with air. The heart was very large—its weight 18 oz. The left ventricle much thickened and dilated; it would contain a small-sized fist. One of the *carnæ columnæ* as big as the little finger. A thick bony plate was seated between two of the aortic valves; and it diminished the length of one of them to four lines, so that regurgitation must have taken place into the ventricle. The ascending aorta was dilated, and the lining membrane of this vessel, throughout its whole extent, was of an intense red colour, and bony and cartilaginous

patches were deposited under the serous membrane of various sizes. The blood was fluid. I exhibited the heart and aorta at the Medical Society, of London, a few hours after they were taken from the body; and if redness, which could not be washed off, is to be considered as an indication of aortitis, a better specimen could not have been met with; but after the aorta had been in water for five days, the redness had entirely disappeared.

The other case was that of a member of our profession whom I saw several times last year; he had suffered from disease of the heart for a long time; (the case is briefly alluded to in my paper on blood-letting, page 199), he had loud bellows' sound over the base of the heart, and difficulty of breathing on exertion, or on ascending hilly ground. Before his death he had severe pleuro-pneumonia and intense pain in the epigastric region so as to lead me to suspect and hope that it might be occasioned by the passage of a gall-stone. The gentleman who performed the autopsy wrote me word, that there was general and intense pleuro-pneumonia on the left side: a small abscess also existed between the lobes of the liver, and this had discharged its contents into the peritoneal cavity. This gentleman adds, "that the serous coat of the aorta was highly inflamed, and that death had been occasioned by inflammation of the lining membrane of the heart, arteries, and veins."

I record the above cases, not as examples of aortitis, but rather as warnings to those who are apt to confound redness of the aorta, arising from a peculiar condition of the blood, with arteritis.

As regards the treatment of this disease I have nothing to add to my former statement, (page 30), except again to express my ignorance of any mode by which the affection can be diagnosed: but whether the symptoms mentioned depend upon inflammation of the lining membrane of the aorta, of the semilunar valves, or of the endocardium, I believe that the treatment recommended will be quite as applicable.

Chronic Aortitis.—In speaking of the morbid conditions of arteries, I have alluded to the cartilaginous and bony state of the aorta, combined with redness of the internal coat, which is sometimes met with in subjects under fifty years of age. Table of cases—9, 10, 41, 59, 60, 62, 86, 92. It is, I think, probable that some of the throbbing pains which are so common along the course of the left side of the spine, owe their origin to this chronic form of aortitis which differs from the cretaceous and bony infiltrations, the result of old age. The formation of the latter deposits is probably unattended with pain. I could relate several cases where I have suspected pain in the back and increased action of the heart to depend upon chronic aortitis, but the narration of one will suffice for illustration.

Mrs. B., æt. 36, consulted me, March 26, 1851, in consequence of pain in the back along the upper and left side of the spinal column which she has had at intervals for the last two years; she has been subject to chronic rheumatism, and her occupation (the landlady of an inn) has compelled her to lead a very active life. The impulse of the heart is greater than usual, and its first sound prolonged; on applying the stethoscope over the back where she complains of pain and throbbing, no abnormal sound is heard. She has occasionally had numbness of the arm, and a disposition to syncope. On the application

of a belladonna plaister to the cardiac region, counter irritants along the spine, mild mercurials at bed-time, and small doses of colchicum for a short time, the symptoms improved. I was again requested to see her in August last, when she was suffering from the same symptoms. I mention this case as one that may, I think, depend upon a chronic inflammation of the aorta, although the evidence, I admit, is purely hypothetical. The pain in the back and in the cardiac region is not of the character of that which frequently attends liver and stomach derangements, nor do the symptoms warrant the belief that these organs are out of order. There is sometimes, in persons of this age, great change of structure in the coats of the aorta; but at present we have no proof that these lesions are accompanied by pain. The matter for future pathologists to decide is, whether many pains in the back, which are generally supposed to be rheumatic, or to arise from chilopoietic disorder, are not occasioned by chronic aortitis?

Acute Inflammation of the Arteries of the Extremities.

My Essay on this subject (*Lancet*, May, 1841) was the first that appeared in this country on the connexion of dry gangrene with acute arteritis. I take no credit to myself for the circumstance, as I only followed in the wake of Dupuytren, and other French surgeons, who had before investigated the pathology of this form of gangrene; but I again mention the fact for the reason I have stated in my introduction to this Appendix. My object will now be to allude to such practical matter as will tend to the farther elucidation of this subject. I refrain from relating more cases in detail, as I think the pathological evidence I have already adduced is sufficient.

I will only speak of one of my former cases (case 1, p. 37), that of the girl whose leg I amputated in 1835 for dry gangrene. Since the last report, October, 1845 (page 45), she has been in a good state of health, but she is now much stouter than before. The heart's impulse is rather increased, and the first sound a little prolonged; no abnormal *bruit* is heard in the cardiac region. No pulsation can be felt in the left brachial and radial arteries, and it is very feeble in the right. When last examined pulsation was also absent in the right popliteal and tarsal arteries. In cold weather she has great difficulty in keeping the extremities warm. She is able to take long walks, although the wooden leg, of course, is some hindrance to her doing so. I need scarcely comment again on this case, more than to repeat that the plugging of the arteries from arteritis becomes now more than probable. No well authenticated instance can be adduced of the occurrence of non-pulsation in an artery, without a mechanical cause; and there is no evidence to show that an artery possesses any power independent of the heart. The increased action of arteries so constantly spoken of, and taught in our schools, does not arise, I believe, from any contractile power in the artery.

I have not said that every case of *dry* gangrene is produced by arteritis; but I say that, in every case that I have examined, I have found evidence of its existence; and in the great majority of those cases in which the disease has been attributed to other causes, the arteries have not been *carefully* inspected. The morbid anatomy of the arteries and veins, as I have several times remarked, is more

neglected than any department of pathology; and I mention the two following cases, which I heard related at the Pathological Society of London, February 5, 1850, as illustrations. They are published in the Transactions for 1850, pp. 233, 251. I give them much abreviated. Dr. Bence Jones exhibited two masses of fibrine, taken from the knee-joint of a strong healthy-looking maid-servant, æt. 21, who died in St. George's Hospital, of acute rheumatism. Besides the joint affection there was pain in the chest, and a soft murmur over the base of the heart, towards the left clavicle. Symptoms of *delirium tremens* came on; laudanum was given; the pupils were contracted; a state of coma succeeded the excitement; and the patient died apparently from exhaustion. The condition of the brain was normal. The pericardium contained about two ounces of serum. There was a slight roughness on the surface of one auricle. The valves of the heart normal; and those of the arteries free from fibrinous deposit. The synovial membrane of both knee-joints was inflamed; and the joint contained two thickish masses of fibrine.

Mr. P. Hewett, on the same evening, exhibited the kidneys of a woman, æt. 53, who died in St. George's Hospital. She had comminuted fracture at the middle third of the leg, from a fall in the street. Gangrene of the foot and leg took place on the fourth day; amputation was performed, as no apparent disease (Mr. Hewett says) was present in the heart or main arteries of the limb. In forty-eight hours the stump began to slough, and death took place on the following evening. Dissection of the limb *removed*, showed the vessels were healthy and pervious, but the kidneys were found larger than natural; their cortical substance was coarse and congested, and the tubuli, when seen under the microscope, were most of them crammed with cells and granular matter. Mr. Hewett believed that the gangrene was occasioned by the state of the kidneys. I shall not stop to enquire whether gangrene is likely to be produced by such means? I allude to these cases because they appear to me to bear upon the question at issue, viz., the neglect of arterial inspection, even by some ardent cultivators of morbid anatomy. Mr. Hewett, in the course of the discussion, admitted that the upper part of the femoral and iliac arteries had not been examined; and Dr. Jones said no inspection of the aorta had been made in his patient. Now, in the one case aortitis may have existed; in the other, plugging of the iliac arteries? The curious example of dry gangrene in a child, only three years old, related by Mr. Solly, in the Medico-Chirurgical Transactions, 1840, is considered by many to have been unconnected with arteritis; and Mr. Solly remarks, "he is at a loss to account for the gangrene, there being little or no alteration in the arteries." But Mr. Bury, who first examined the child, says, "the femoral arteries were remarkably shrunk, very pale, and not patulous, on division with the knife."

I scarcely need revert to the error of Dr. Carswell and others, who supposed that this disease was confined to the old; nor need I comment on the inappropriateness of the term "*senile gangrene*." Dr. Hecker, of Stuttgard, published a small treatise, which had before escaped my notice, "On the Nosological and Therapeutical Researches on Gangrene, from Obstruction to the Circulation of the Blood." In a table of 69 cases collected by him, 27 only were females. In 67 of

the cases, where the age had been recorded, one occurred between the age of 1 and 10 years; six between 10 and 20; seven between 20 and 30; nine between 30 and 40; one between 40 and 50; twelve between 50 and 60; nineteen between 70 and 80; eight between 80 and 90; and one between 90 and 100. Of the above, 43 recovered, and 26 died.—*British and Foreign Medical Review, vol.* xiv. 1842.

I could quote cases in abundance, to illustrate the opinion I have before expressed respecting the nature of this disease, but I will only briefly mention a few of the most remarkable.—1. A girl, æt. 27, was admitted into the Leeds' Infirmary, May, 1845, under Mr. Teale, with dry gangrene of the leg and foot, of which she died. "The femoral artery, at its commencement, presented an annular contraction, as if it had been embraced by a ligature, and its cavity at this part was completely obliterated."—*Provincial Journal*, 1845.—2. In the remarkable case published in the same Journal, 1846, by Mr. Drayman, the boy was only ten years old, and although the hands, feet, nose, lips, and ears, were affected, he lived fourteen months. An examination of the body could not be obtained.—3. Mr. Cooper, at the Norwich Pathological Society, mentions the case of a girl, æt. 10, who, after severe pain in the foot, was affected with dry gangrene, for which the leg was successfully amputated. There was no pulsation in the femoral and popliteal arteries. Mr. Johnson, in commenting upon this case, attributed the gangrene to arteritis, and spoke of examples of this kind, in which he had found the antiphlogistic treatment successful.—*Provincial Journal*, 1848.—4. Mr. Bottomley's case, alluded to in the *Medical Examiner* (Vol. II., p. 152), is one of great interest. A man, æt. 53, in running down a hill, thought he felt something give way in his leg; pain and dry gangrene of the foot occurred soon after this; pulsation was absent in the popliteal artery, and it was very faint in the femoral. Spontaneous amputation, to a partial extent, took place, and the patient did well.—5. A man, æt. 38, died under the care of Mr. Clark, in St. Thomas's Hospital, February, 1850, of dry gangrene of the foot, which came on after rigors and erratic pains in the joints. "The lower part of the popliteal artery and the posterior tibial, for half an inch below the origin of the femoral, were filled with firm, contracted coagula, in all parts adherent to the lining membrane of the artery.—*Lancet*, 1850.—6. Dr. T. Thompson attended a woman, æt. 52, affected with phthisis, who died of gangrene of the hand and arm, which was preceded by pain in the limb. Some doubt existed as to the presence of pulsation in any of the arteries of the extremity soon after the discolouration commenced; but on the fith day pulsation was entirely absent an inch below the clavicle. Various coagula were found, but a roll of fibrine three inches long extended from the origin of the subclavian; it was closely adherent to the aorta at one point, and when torn from this attachment a portion remained fixed to the artery. *Tran. of the London Pathological Society* 1851, *p.* 85.—7. Dr. Aran narrates an example of dry gangrene of the leg in a man æt. 71, in which the external iliac artery was found obliterated by a hard fibrinous clot, adherent to the inner coat of the vessel. *Gazette des Hôpitaux* 1851, *p.* 432.—8. A girl æt. 19, who was under the care of Dr. Wilmot had intense and burning pain in the great toe succeeded by dry gangrene; she lost two of her toes,

and after this recovered. This case Dr. Wilmot considered one of arteritis, but in another case which he relates, the subject of it a pensioner fifty years of age, the disease is thought to have arisen from ossification of the arteries; but, judging from Dr. Wilmot's narrative, I am at a loss to discover any evidence of this? *Dublin Journal, vol.* 9, 1850.—9. Dr. Fuller read a case of spontaneous gangrene at the London Medico Chirurgical Society in 1847. A woman æt. 37, was seized with most acute pain in the right foot with excessive tenderness of the integuments. After a few days both lower extremities became gangrenous, and she died five weeks from the time of seizure. The arteries are reported to have been free from inflammatory action, but the lower part of the abdominal aorta, the arteries given off below the superior mesenteric, and the arteries of the affected limbs, together with their corresponding veins, were blocked up with firm fibrinous coagula, which were in a great measure discolored and slightly adherent to the internal coat of these vessels. The kidneys were granular, and the urine albuminous. *Lancet*, 1847.

Dr. A. De Martino *(Annali Universali di Medicina*, 1848, vol. xxx.) thinks "that dry gangrene does not arise from the obstruction of the large arteries alone, but that the blood in the capillary vessels of the part is in a state of stagnation and dryness, and this he attributes to innervation. In forming these conclusions he has been assisted by microscopical examinations." But it must be apparent to all, that if the main artery of a limb is obstructed from any cause, or if the *vis a tergo* is not sufficient to propel the blood, that stagnation must be the result. I believe, in every species of gangrene, the circulation is more or less obstructed, and as I have before said, when the main artery, *and the collateral branches* are closed, the effect is, the death of the parts below, as shown in the black, dry, and shrivelled state of the limb.—That the varieties of spontaneous or dry gangrene depend upon the condition of the heart, and the amount of local obstruction in the artery—that this increased action of the arteries of the part spoken of by Professor Syme, and many modern writers, depends upon the obstruction, and upon the reaction of the blood; and not upon any independent power of the artery.

In many cases of dry gangrene, the artery is said to have been free from inflammation even when the clot was adherent to the serous coat, but we know that the vessels of the cornea and other parts, which in their normal condition carry white blood, may, when inflamed, be distended with red corpuscles, and no trace of the congestion or inflammation be found after death. In other instances, we can only judge of the previous existence of inflammation by its effects.

Treatment.—I have nothing to add to the opinions already expressed at page 35, but I would endeavor especially to impress upon the practitioner, the importance of having recourse to *early* antiphlogistic treatment in this disease when the symptoms are of an active character, and to divest his mind of the fashionable dogmas of the day, "that bleeding is necessarily a debilitating process, and that dry gangrene depends upon a want of constitutional power." Mr. Syme in his *Pathology and Practice of Surgery*, 1848, advocates the same treatment he recommended in 1841, although he says, the highest surgical authority in the southern metropolis (Sir B. Brodie) is still

in favor of nourishment and stimulation. I terminate this part of my subject with a quotation from a clinical lecture, delivered by Mr. Teal at the Leeds Infirmary, 1845:—"The value of the treatment, recommended by M. Dupuytren, I have had repeated opportunities of verifying. Frequently have I witnessed, even in aged subjects, most evident and immediate relief of pain from the abstraction of a few ounces of blood, when opiates, stimulants, and tonics, had proved unavailing; and by the same treatment I have not unfrequently seen threatened gangrene averted, and existing gangrene arrested in its progress. But I need scarcely tell you, that in adopting this moderate antiphlogistic treatment, regard must be had to the condition of the patient. If the skin be of somewhat higher temperature than natural, the cheeks flushed, the pulse not deficient in force, although the patient be advanced in years, and apparently much shattered by the disease, the repeated abstraction of small quantities of blood may be employed with safety and advantage; but if the powers of life be flagging, as in the case which you have lately witnessed, we have no alternative but to endeavor, by the use of stimulants and cordials, to prolong life for a brief period."

Gangrene from disease of the Heart or Blood.

Although John Hunter said that blood would not coagulate in a healthy artery, it is not improbable that gangrene is sometimes produced by disease of the heart, combined with an abnormal condition of the circulating fluid; but whether the clot be caused by inflammation of the artery, or by disease of the heart and blood, the effect is nearly the same. In the latter cases however, the veins are generally obstructed by coagula, and the limb does not present the dry, shrivelled appearance, that it does in gangrene from arteritis, and the pain too is generally less severe.

Mr. Clark, of Devizes, in the Medical Gazette, 1837, relates the case of a woman, æt. 50, who, when in a profuse perspiration, placed her right heel on a damp brick floor, this was followed, after twelve or fourteen hours, by severe pain in the foot, darting upwards to the thigh, and attended with coldness of the limb. The pulse was extremely irregular in frequency. The limb, in two days, became of a leaden hue, and pulsation was absent in the posterior tibial, and popliteal arteries, and it was very slight in the femoral. The thigh, on the sixth day, was considerably enlarged, but without any symptoms of inflammation; decomposition advanced rapidly in the leg, and death took place eight days from the commencement of the attack. The heart was very large, the left auricle double its natural size, and the mitral valves cartilaginous at their edges. The femoral artery gradually tapered until its cavity became obliterated above the part where the sloughing commenced; the coats healthy. Although this is described as a case of gangrene from disease of the heart, I think it may fairly be asked whether the exposure to cold did not occasion arteritis, which rapidly extended upwards to the femoral artery? A case of gangrene of the extremities is recorded in the *Dublin Journal*, vol. x., 1850, by Dr. Power, which appeared to depend upon disease of the mitral valve. A collier, æt. 42, affected with disease of the heart, and also subject to rheumatism, after exposing his legs to cold, felt a numbness in the legs

and toes; in a few days both limbs were gangrenous (not dry gangrene.) The heart was much diseased, and may be seen in the Museum of the Dublin Richmond School of Medicine. The popliteal, and the anterior and posterior tibial arteries contained coagula, but they were not adherent to the vessel.—Professor Schutzenberger, *Gazette Médicale de Paris*, 1847, p. 612, narrates a case of arrest of the circulation from the same cause. "The feet were black and œdematous. The patient was of a rheumatic diathesis, and only 24 years of age. The mitral valve would scarcely admit the end of the fore finger; both iliac arteries, as well as the veins, contained coagula." I had an opportunity of seeing the following remarkable case in August last, with my friend Mr. Richardson, of Mortlake, who has kindly furnished me with the undermentioned history.—

"Mrs. B., æt. 26. This patient states that she first became ill in 1848. She is a dress maker by occupation; is tall, pale, and emaciated. Previous to her illness she had been subjected to bad treatment from her husband. When first indisposed in 1848, she complained most of lowness of spirits, with fits of feverish excitement, and frequent palpitations of the heart. One evening on getting into bed she felt a sensation of pain in the feet, and on looking at them she said, that they were swollen and red. For this she was put under medical treatment and she soon recovered. The only immediate cause for this attack which the patient can name is, that she had been using a foot-bath of hot water with mustard, on the evening previous to the appearance of the symptoms.

From the period above named, until the month of November, 1850, she continued debilitated and had some slight returns of the pain and swelling in the feet. After this date the symptoms were more frequent and severe. On the 28th of February, 1851, she first came under my care. I found her in a most deplorable condition; an enfeebled heart was acting with a rapidity such as I have rarely seen. The feet were swollen, and the tips of the toes were gangrenous; the pain seemed intolerable; the superficial veins of the legs were large, and the circulation through them seemed remarkably impeded. The heat of the surface of the body was low but uniform, and all the secretions were natural.

My opinions as to the cause of this gangrene were divided. The palpitations naturally led me to the heart, but what state of the heart could exist to lead to it? were there on the valves warty vegetations, and were portions of these being removed by the circulating currents, and being carried round in the course of the vessels, thus plugging up the minute arterial branches, or the capillary system? or was there a fibrinous concretion in the heart, undergoing the same process? At first I inclined to the latter opinion. I prescribed mercury with morphia, and to my surprise, as the system came under the influence of the mercurial, the symptoms entirely passed off, the pain ceased, the gangrene disappeared, nourishing food was borne, and recovery so far took place that the patient was enabled to resume her ordinary occupation, coldness of the feet only remaining.

I heard no more of this woman for three months, when I was one day again requested to visit her. I found her suffering from the palpitations as before, and expecting an attack at the feet. In this

she was right. As the frequency and power of the heart lessened, the pain and swelling of the feet returned, followed by blackness of more than half the feet with gangrene at the tips of all the toes, and enlargement of the superficial veins. The right hand was now affected in a similar manner, the fore-finger of her right hand being in a gangrenous state also. The present attack seemed to have been induced by a diarrhœa, from which she had been suffering some time, and which had much reduced her. On this occasion I prescribed profuse quantities of saline drinks, combined with light nutritious diet. Since this period, I have seen her suffer from two other similar attacks, and I have observed the symptoms closely: they always follow any cause which robs the blood of its fluidity, or produces much prostration. Rest also tends greatly to bring them on, and indeed sometimes when she feels premonitory symptoms, (numbness of the legs, &c.,) she can keep off the attack by taking a brisk walk, and having recourse to saline medicines, consisting of nitrate and citrate of potash. The gangrene is always superficial and always passes off dry, leaving a delicate skin beneath. During the severer periods of attack the cutaneous veins are always enlarged, and in the flexures of the joints may be felt in the veins, moveable soft bodies, the size of small peas, which can be pushed along in the course of the circulation. The heart presents no form of disease, as far as I can ascertain, and in this opinion I am backed by those of my friends, Drs. Willis, J. Arnott, and Crisp, all of whom have seen the case with me.

I set this case down now, as belonging to the same class as some of those described by Dr. Burrows in his Croonian Lectures. I believe that the gangrene is the result of an impeded circulation, from a soft coagulation of fibrine in the extreme venous trunks, such coagulation being itself induced by any cause that enfeebles the circulatory powers, or robs the blood of its fluid matters. I have formed this opinion on three grounds; 1st, That the gangrene is always brought on by diarrhœa, and is in exact proportion to the feebleness of the circulation. 2nd, On the enlargement of the veins, and the existence of the soft masses to which I have alluded. 3rd, On the beneficial results which always arise from the use of mercury and of saline solutions, and also from the good effects of motion."

The subject of fibrinous concretions in the blood-vessels is one that requires farther investigation; and it is not unlikely, as supposed by Mr. Richardson and others, that these concretions may sometimes lead to a fatal result. I am disposed to think, however, that in the above case, the partial stagnation of the blood arose from weakness of the heart and blood tubes; and that nearly all these polypous concretions in the heart, and its contiguous vessels, arose from a deficiency of the *vis-a-tergo*, and that they are generally formed a short time before death. The inflammatory deposits in the arteries and veins, as I have before remarked, are of a different character.

Absence of the Pulmonary Artery.

In addition to the case of absence of the Pulmonary Artery, related at page 92, I may mention another specimen I have in my museum, given to me by Mr. Skegg, of St. Martin's Lane, who attended the

child. It was affected with blue disease, and died of convulsions, at the age of ten weeks. These convulsions first appeared eight or ten days before death. Previous to this, the child increased in weight, appeared lively, and had no difficulty of breathing. The heart is of a rounded form, and has a flattened fig-like appearance. It consists of one large ventricle, from which the aorta arises at the anterior and upper part: this vessel is divided near to its origin, giving off branches, which probably supplied the lungs. The semilunar valves are in a normal state; and below these is an irregular opening formed by tubercular projections from the sides of the ventricles. The parietes of the ventricle are rather thicker than natural. Behind the aorta there are the remains of a vessel, which terminates in a cul-de-sac on the surface of the ventricle; and above this, the extremity of a larger artery, which has been divided. The auricle has a larger, and smaller auricular appendix, imperfectly divided by a septum, and two auriculo-ventricular valves. Four veins enter the auricle; one situated below, and on the right, is of larger size, and corresponds with the inferior cava; another above, and smaller than the last, occupies the situation of the superior cava; the other openings are small, and posterior to the aorta.

The above cases, and others alluded to (p. 90) are so seldom met with, that they are not of much practical importance, although the physician should be aware of the possibility of their occurrence. There are no signs by which these curious arrests of development can be diagnosed.

Abdominal Pulsation.

If the reader believes with me, that the tone of an artery depends entirely upon the state of its cellular coat, and that the nerves are only distributed to this tunic, the occurrence of these *local* pulsations will be more readily explained; it must be borne in mind that the pulsatory movement is always synchronous with the ventricular contraction, and that there is no independent action of the artery. I could make numerous additions to the cases recorded (p. 99) but these would be of little practical benefit. I have now two patients under my care at the Metropolitan Dispensary, who are affected with this malady, and if the abdominal aorta be examined in various diseases, it will be found that these abnormal pulsations are of more frequent occurrence than is generally believed. Mechanical obstruction, disorder or disease of a neighbouring organ, especially of the stomach, liver, or pancreas, will occasion them. Inflammation of the peritoneum, whether acute or sub-acute, is another cause, and they sometimes occur from the loss of large quantities of blood, although in most of these instances, the pulsation is general. I apprehend, that in all the above-mentioned causes, the nervous supply to the cellular coat, is so deranged, as to weaken and diminish the tone of the artery, and thereby allow of its greater distension at the part affected.

The diagnosis, as I have before remarked, is sometimes attended with difficulty; as, where the pulsation is very circumscribed, it may readily be confounded with aneurism. If, however, the age, sex, temperament, previous diseases, and occupations of the patient are taken into account, the disorder may generally be recognised. The freedom

from pain in most cases, the greater throbbing and bounding of the artery, and the absence of *bruit*, will *generally* enable the practitioner to form a correct opinion.

The object of the treatment will be directed to the disorder or disease of the organ which is supposed to occasion the derangement, indeed I have nothing to add to the suggestions I have before given.

I may mention, that in several cases of tape-worm, that I have treated, palpitation of the heart has been present at intervals, but I have never met with preternatural pulsation of the abdominal aorta in these patients.

Preliminary Remarks.

In the various *critiques* on this work in the Journals, 1847, although it generally, perhaps, received a more extended notice, and greater commendation than it deserved, I believe I scarcely had the credit of an original idea. The reviewer in the Medico-Chirurgical Journal, says, "The work, although not admitting of comparison with some of its predecessors on the list, *in originality, or novelty of views*, is nevertheless a very *creditable* production, and one of which the *general* practitioners have reason to be proud!" Now this is the *unkindest* cut of all. I could bear the accusation of not noticing Mr. Bellingham's work on pressure, for the cure of Aneurism, (which I had never seen or heard of, until after my book was written,) or of the jumbling together of the medical and surgical aneurisms, (although I had made a perfect classification myself in each chapter,) but to be told that the work was wanting in *originality* and *novelty*, when I supposed that I differed, in *most* of my deductions, from those who had previously written upon the subject, was a great blow to my vanity. I must entreat the reader's pardon for drawing the attention of this learned reviewer to some of the matter in this volume, which I believe to be* original and novel. 1st. The experiments on the extensibility of the arteries, (p. 5) and the physical difference between the supposed muscular coat, and the involuntary muscular fibre; the *one* being highly elastic, the *other* possessing scarcely any elasticity. 2. The non-elastic nature of the cellular coat, and its practical bearing on the injuries and diseases of arteries. 3. The various tables of the abnormal conditions of arteries, and the deductions from them. 4. The belief that fatty degeneration and atheromatous deposit, are much less concerned in the production of aneurism than is generally supposed; and the *fact*, obtained only by statistical evidence, that as these deposits increase, so does the tendency to aneurism diminish. 5. That the deposit of fat after a certain age, besides keeping up the heat of the body, serves by lubricating the coats of the arteries, to prevent their fracture. 6. That most aneurisms may be traced to a mechanical cause, and that the arteries most subject to aneurism are those most exposed to violence, and to the influence of the heart's impulse. 7. The rare occurrence of true aneurism,

* With the exception of Lisfranc's table of surgical aneurisms, (1834,) I know of no one who had attempted to elucidate the pathology and morbid anatomy of the arteries by the statistical method, before 1844, when my essay was written, and I did not then know of Lisfranc's table. I thought my plan was *entirely* original.

so-called. 8. The statistics of age, sex, occupations, and climate. 9. Especially the pathology of internal aneurisms, and the fact that rupture into the pericardium, which was considered by Hope to be "very rare," and of which Laennec never met with an example, and Hodgson only alludes to two, is probably the *most frequent termination of thoracic aneurism*. 10. The conclusions respecting dissecting aneurism. 11. The statistics of external aneurisms, the effect of operations, and the time of separation of the ligature. 12. The table of 45 cases of nævus, and the result of operations, &c.

Value of Statistics.

I could find many other suggestions and conclusions that I believe to be original, but I have surely said enough to shew that the reviewer's remark is scarcely just, allowing him, even, the benefit of the *comparison*.

It was scarcely necessary to apologize for this egotistical flourish, because I am *especially* anxious to direct the attention of the reader to some of these subjects; I do not presume to think that all my opinions are correct, for I am too well acquainted with medical fallacies to attempt to draw positive conclusions. Statistical evidence requires a great deal of sifting to remove the chaff and rubbish that often accompanies it, and in all numerical deductions, the first question should be, what are the *likely sources* of error? The climate, diet, habits of the people, mode of treatment, publication of rare and successful cases, and the frequent suppression of those that terminate fatally, are matters that should be *well* considered before general conclusions are drawn. The constitutional differences also between town and country people must be borne in mind; the locality of the hospital, and the class of diseases and accidents more generally admitted. But in all medical reasoning, the possible bias of the writer, and his deference to authority (so called,) should be especially remembered. The dogmas of men are handed down from generation to generation, and few will take the trouble to enquire into their truth or falsehood; but parrot-like, without thought or reflection, they re-echo the notes of their teachers. Let me give one example that will be familiar to every reader.

Morgagni, whose memory must be venerated by every lover of science, said, nearly 100 years ago, (Letter 17, c 18,) "that he and his friends had observed aortic aneurisms more frequently in postillions than amongst any other class of men, and he thought taking into account their occupation and habits, that the circumstance was not surprising." Morgagni was probably correct, as regards his own countrymen, for these *Vetturini* were, perhaps, the hardest working men in Italy. Observe, however, the importance of taking the habits and occupations of the people into consideration! In almost every surgical treatise on aneurism, and in many modern lectures on surgery, it is stated upon Morgagni's authority, that popliteal aneurism is more frequent among postillions. The table of 182 cases of popliteal aneurism in this appendix, shews that of the 133 whose occupations are named, one only was a postillion, and another an ostler who had been a postillion ten years before the appearance of the aneurism. But there are 26 sailors and bargemen, 17 soldiers, 9 porters, and 8 labourers; whilst in aneurism at the upper part of the femoral artery, out of 40 whose occupations are named, 12 were sailors and bargemen.

If the habits and occupations of these people are considered, an explanation is readily afforded of the cause of their greater tendency to aneurism of the lower limbs.

The above remarks, I hope, will not be thought irrelevant to the subject I am about to investigate, and although I have thought it necessary to allude to the numerous errors that may attend statistical deductions (and I, perhaps, have fallen into some of them myself,) I believe the numerical is the only method by which the many doubts and difficulties connected with our art, can be removed. Let me ask those who sneer at statistics, whether *all* their experience is not based upon statistical evidence? whether they have not been generally guided in their diagnoses and therapeutical indications, by the number of cases they have seen and treated? The greatest curse to medical science, has been the blind deference paid to authorities, who depended solely upon their *own* experience, and whose dogmatism, for the most part, is only equalled by their ignorance. I know one of these sage practitioners, who, when the correctness of his opinion was called in question, replied, "Sir, I have had forty years experience, and have not read Hippocrates for nothing." The works of Andral, Louis, Cruveilhier, and Rokitansky, were to him sealed books; he rejoiced more in *classical*, than in *pathological* knowledge. The reader will remember many such.

Aneurism.

For reasons stated in my former Treatise, as well as in the preliminary observations just made, I purpose in *this* Appendix to confine myself to British statistics, so that the work may serve as a reference to foreign writers who may be disposed to make comparisons between the prevalence of arterial diseases, modes of treatment, &c., in this country and their own; a practice, I think, too much neglected by most writers. In the German translation of this Essay, (Berlin, 1849,) the translator has finished his task, without *word* or *comment*, but if he had collected the statistics of the diseases and injuries of the blood vessels, and their treatment, in the German States, he would have added greatly to the interest and practical utility of the work. I throw this out as a hint to those who translate books without consulting or communicating with the authors.

When in Paris, (September, 1850,) I saw a man at the *Hôpital de la Charité*, whose femoral artery had been tied for popliteal aneurism; the wound was filled with *charpie*, and this treatment had been continued to the time I saw the patient, (the 17th day, p. o.) M. Velpeau told me that "he adopted this plan of treatment on account of glandular enlargements in the groin," but "he did not always employ it," he said, "after the ligature of an artery." Up to the present time, in France, two ligatures are sometimes used. I find in the *Boletin de Medicina, Cirurgia, y Farmacia of Madrid*, that Dr. Olivares, Surgeon to the General Hospital, in April, 1851, tied the femoral artery for popliteal aneurism, and he adopted the same practice, and judging from

an Essay on Aneurism by Dr. Salvolini, published at Venice in 1849, (*Gazette Med. de Strasbourg, Aug.*, 1851,) a like method is pursued in Italy. Dr. Bouer, a German physican, now residing in London, who is well conversant with the practice of Surgery in the German States, tells me that in Germany one ligature is generally used. In the United States, the mode of operating is, for the most part, similar to that practiced by the English surgeons.

The importance therefore of keeping the statistical inferences respecting the results of operations, &c., in different countries, separate, must be at once apparent.

I now continue the Table of Aneurisms, selecting all the published cases in the British Journals, *from the last date up to the present time, September*, 1852. It is not unlikely, owing to imperfections of some of the indices, that I may have omitted a few. I give the name of the Journal, as well as of the medical attendant, so that the cases can be referred to if necessary. The classification and statistics of the various aneurisms I shall notice *separately*, as in my former essay.

Lancet.—Cases by Messrs. Burchell, Farr, Canton, Walsh, Ellerton, Adams, Canton, Clarke, Partridge, Parrott, Briscoe, Lankester, Avery, Latty, Hilton, Ward, Lawrence, Allan, Swettenham, Reeves, Norgate, Addison, Little, Barlow, Clarke. *Medical Gazette.*—Hawkins, Hare, Hare, Renaud, Hancock, Turnbull, Clarke, Crompton, Fife, Heath, Fergusson, Paget. *Medical Times.*—Ayres, Corfe, Judd, Duke, Ambrose, Gordon, Gelston, Crompton, Adams, Gairdner, Thompson, Cotton, Smith, Wakley, Edwards, Dartnell, Forbes, Ward, Hutton, Birkett, Stephens. *London Journal of Medicine.*—Hare, Gairdner. *London Pathological Society's Transactions*—M'Intyre, Glendenning, Fuller, Day, Bird, Avery, Little, Nun, Hassal, Bently, Peacock, Moore, Shelley, Peacock, Thomson, Hewitt, Partridge, Jay, Hancock, Avery, Brinton, Obré, Vickers, Barlow, Fuller. *Medical Chirurgical Transactions.*—Wright, Bennett, Bellingham, Hewett. *Provincial Medical and Surgical Journal.*—Freer, Russell, Fletcher, Fletcher, Fletcher, Pemberton, Hunter, Wardell, Kingdon, Fletcher, Gorham, Fletcher, Fletcher, Ward, Humphrey, Gooch, Hallowes. *Dublin Journal.*—Cusack, Gilston, Lyons, Lyons, O'Conner, Hobart, Popham, Lyons, O'Ferrall, Stokes, Colles, Ross, Hargraves, O'Ferrall, Mayne, M'Dowell. *Dublin Medical Press.*—Togood, Wishart, Bellingham, Bellingham, Adams, Tuffnell, Ainsworth, Bellingham, Barrow, Kingdon, Madden, Butcher, Bellingham, Bellingham. *Edinburgh Monthly Journal.*—Syme, Syme, Pirrie, Douglas, Gairdner, Miller, Syme, Syme, Drummond, M'Kensie, Syme. *Private cases.*—Holt, Crisp.

It must be observed that in this, as in the other table, I have rejected the insertion of all *internal* aneurisms where the nature of the aneurism has not been verified by a post mortem examination, and I have only introduced two private cases, one of Mr. Holt's and another of my own, as they both present features of *practical* importance.

The last table includes 151 cases; 123 were males, and 28 females. 67 of the aneurisms were in the thoracic aorta, 12 abdominal aorta, 2 superior mesenteric, 6 femoral, 45 popliteal, 6 internal carotid and cerebral, 3 common and external carotid, 2 innominata, 2 subclavian, 6 axillary. As regards age—one was under 20. Between 20 and 30—23. Between 30 and 40—59. Between 40 and 50—36. Between 50 and 60—17. Over 60—7, and in 9 the ages are not named.

On adding these 151 aneurisms to the 551 in the former table, the following are the general statistics of the 702 cases.

Thoracic aorta, 241. Pulmonary, 2. Abdominal Aorta and its branches, 73. Common iliac, 2. External iliac, 9. Gluteal, 2. Femoral, 72. Popliteal, 182. Posterior tibial, 21. Innominata, 22. Carotid, 28. Cerebral, 13. Temporal, 1. Ophthalmic, 1. Subclavian, 25. Axillary, 24. Subscapular, 1. Brachial, 1.

I now proceed to treat briefly of the most important aneurisms, under different heads, and my deductions will be drawn chiefly from the above tables. I may premise, that of late all popliteal aneurisms cured by pressure, have been published, and that external aneurisms are generally made known, whilst many in the chest and abdomen are not examined after death; or if examined, they are not publicly recorded; moreover it is not unlikely that small aneurisms, when autopsies are imperfectly performed, sometimes escape detection. The reader must take these circumstances into account in forming his estimate of the value of the statistical inferences. The exact boundary, too, of an aneurism in the curvature of the aorta is *sometimes* difficult to determine: for example, it may be a matter of doubt whether it should be classed among those of the ascending, transverse or descending portions of this vessel, or whether two parts (as is often the case) are implicated? Another important consideration is the fact, that most aneurisms commence a long period before the death of the patient: Probably the average duration of thoracic aneurism, is about eight or ten years, although this is a point difficult to determine.

Nomenclature.

In speaking of the nomenclature of aneurism, (p. 109,) I have remarked, "I think there can be no doubt that Scarpa's opinion as to the non-existence of true aneurism was erroneous, as some specimens of true aneurism can be found; it is however, probable, that this form of aneurism is much less frequent than modern writers suppose. It is extremely difficult when the sac has attained a large size, to ascertain the exact condition of the coats, and in many small aneurisms in which the inner tunics were considered entire, I have, on a careful examination, found them imperfect." When I made these remarks, I had examined nearly 400 specimens of aneurism, and although I had never seen what is called *true* aneurism, I was *then* induced, in deference to the opinion of others, to speak guardedly upon the subject. Farther research, however, and the more careful examination of other specimens leads me to think that this form of aneurism, if it exist at all, is of *very* rare occurrence. Dr. Bellingham, in the Dublin Medical Press, has recently expressed a similar opinion.

The reader must understand that I speak of the *circumscribed* bulging of the coats of an artery—not of the *uniform* dilatation which

sometimes occurs, and which does not strictly come under the definition of aneurism.

The most applicable terms to distinguish the traumatic from the (falsely called) spontaneous aneurisms, I think are those I have already suggested, viz., the *endogenous* and *exogenous;* the former commencing in lesion of the inner or middle coats; the latter arising from injury to all the tunics. I admit, however, the inconvenience of departing from an old and generally received arrangement, however faulty it may be found.

Causes of Aneurism.

If the reader will turn to the sketch of the arterial tree at the commencement of this essay, he will readily find, as I have described at page 114, a mechanical cause for the occurrence of aneurism in certain arteries, and if he still further investigate the matter he will discover that the arteries most subject to atheromatous and bony deposit, are not always those most affected with aneurism.

Age and *sex*.—The statistics of sex I have already given, and the additional table, somewhat alters the inferences before drawn (p. 115) respecting the proportion of cases in females; the difference as regards the prevalence of aneurism in the two sexes not being quite so great as before remarked. The following is the analysis of the ages of the 702 cases. Under 10, 1; between 10 and 20, 6; between 20 and 30, 94; between 30 and 40, 247; between 40 and 50, 164; between 50 and 60, 82; between 60 and 70, 32; between 70 and 80, 8; between 80 and 90, 2; over 90, 1; age not stated, 55.

Occupations.—There is plenty of evidence in the table to show that laborious occupations tend much to produce aneurism, a disease that frequently occurs in the strong and muscular, who have before enjoyed good health. The greater number of men that I have seen affected with aneurism have been robust and muscular. Inordinate and irregular exertion, under the stimulus of alcohol, is one of the most fruitful sources of this complaint.

Blows and *falls*.—A vast number of aneurisms may be traced to these causes, and I believe (as before stated) that this disease may occur in a healthy artery. A strong muscular man under 25 years of age, (a time of life when the blood-vessels of the limbs are seldom diseased,) perhaps when walking, slips his foot into a hole, in the ground; he feels a sudden snap in the ham, and popliteal aneurism is the result. A blow, (as in the case of poor Mr. Liston,) is received upon the chest; the patient, although probably not conscious at the time, of any important inconvenience from the accident, begins sooner or later to complain of occasional pain, more or less severe, in the thoracic region; or if pain be not present, he may experience a feeling of oppression about the præcordia, or a throbbing sensation on exertion. But these symptoms do not generally induce his medical attendant to suspect the presence of aneurism, and unless an external tumor is formed, the occurrence of this fearful lesion is only revealed by a post mortem examination. Blows, falls, accidents, and exertions of various kinds, by fracturing the coats of the artery, or by weakening the integrity of its tissues, are, I believe, the chief *exciting* causes of aneurism.

Atheromatous and *fatty deposit*.—I have already expressed my belief that the latter deposit is one of the greatest preservatives against aneurism—a beautiful provision of nature for lubricating the arteries of the old, and preventing their fracture. That this small proportion of fat in the atheromatous matter arises, as in other parts, from an altered state of the blood, and a slow and arrested circulation in the white blood channels; and that it is, to a great extent, a consequence of the partial choking or obstruction of these canals, by the previous congestion, or inflammatory action.

There is one great source of error into which I think those who attribute so much importance to these deposits, as a cause of aneurism, have fallen, viz., the inference that the deposit takes place before the occurrence of the aneurism. I believe that these depositions are very often the *consequence*, not the *cause* of the disease, and that they are produced, for the most part, after the sac is formed. I have examined the arteries of a great number of horses of all ages, and I have never seen these deposits; but in a few dry specimens of aortic aneurism in the horse, all supposed to arise from violence, the aneurismal sacs were covered with atheromatous matter. The most conclusive proof, I think, is the fact that the popliteal artery in subjects under thirty years of age, is seldom atheromatous, but how common is aneurismal dilatation of this artery? The abdominal aorta, again, is more affected with these deposits close to its bifurcation, but aneurism occurs generally near the cœliac axis, a part where the caliber of the tube is diminished, and where the tunics are probably more yielding, from the number of branches that are given off at this spot.

Climate.—In addition to the remarks I have made under this head (chapter viii.) my subsequent investigations lead me to express the same opinion that I did in 1847, viz., that aneurism, owing to the laborious occupations of the people, and the use of ardent spirits, is more prevalent in England than in any part of the globe, although the *general* mortality is probably less than in any other country. From a second examination of the Registrar-General's Reports, I find that the mortality from this disease, in London, during the twelve years from 1840 to 1851 was as follows—49, 36, 24, 37, 41, 62, 55, 71, 70, 85, 89, 80. In the years 1847 and 1848, the general mortality in England and Wales, from aneurism, was 566. In the latter year, of the 307 cases, 216 were males, and 91 females; and the subjoined are the proportions in the 11 districts—London, 70; South Eastern, 35; South Midland, 10; Eastern, 14; South Western, 33; West Midland, 25; North Midland, 18; North Western, 43; York, 23; Northern, 22; Wales, 14. Of the 259 cases that occurred in 1847, 53 were under 30 years of age, 164 between 30 and 60, and the ages of 42 exceeded 60. From 1838 to 1842, according to these reports, the annual number of deaths from aneurism to 1,000,000 living, was 8, 6, 9, 8, 7, for each year. It will be seen that these figures are much below the average. I am unable to obtain the general statistics, as they are not yet completed.

The reader should be reminded that these returns, at present, are in a very imperfect state. Many of the patients were not examined after death; and probably some cases are classed under the head of aneurism when this disease did not really exist. Thus, in 1847, three aneurisms

are said to have occurred in children under twelve months of age; but as far as my enquiries have gone, our published records contain no examples of aneurism in infants. External aneurisms, too, it must be recollected, are mostly cured; a circumstance that will make a vast difference in the general statistics of sex and age.

The United States of America, where the same causes as those above-mentioned are in operation, probably afford, next to England, the greatest mortality from this disease, although the statistics at present are in a very unsatisfactory state. The reviewer of this work, in the *American Journal of Medical Science*, says, "Some difference of opinion appears to exist among the surgeons of our country respecting the prevalence of aneurisms here. Professor Gibson believes that in New York, the residence of so many foreigners, aneurisms are almost exclusively confined to them, especially to the English. Dr. Mott, on the other hand, declares that the great majority, if not all the cases which have occurred in his practice for nearly forty years, have been among his own countrymen. Indeed, he says, 'he cannot recollect a single instance of a foreigner, and that nearly all belonged to that class who had been accustomed to hard labour.'"

In addition to the interesting intelligence afforded by my friend, Mr. C. S. Webber, of Connaught Square, respecting the prevalence of aneurism in the East Indies, I may quote the following information, obtained from my brother, Dr. Henry Crisp, Senior Assistant Surgeon to H. M. 64th Regiment, Hydrabad, Scinde; he has been about four years in India; he says,—"I saw one case of aneurism of the arch of the aorta, about a month ago, in a native Portuguese; he died of suffocation a few days after his admission to the General Hospital, Hydrabad. The tumor presented on the right side of sternum; at the autopsy nothing unusual was discovered about the aneurism. Dr. Craig, who attended him, said it was the first case of aneurism he had seen, after about twelve years' residence in India, during which time he has been surgeon to several native regiments and hospitals; but the objection that the inhabitants of India have to post mortem examinations is a great bar to their discovery." My friend, Dr. Daniell, who has resided several years on the western coast of Africa, tells me that he has seen two instances of aneurism in natives. In the reports of the Chinese Medical Missionaries, which extend from 1840 to 1849, and include nearly 20,000 cases, treated chiefly at Canton, Macao, and Amoy, a case of aneurism of the external iliac is named; another of the temporal artery, and a third is entered as aneurism. It is not stated whether these aneurisms were traumatic, or otherwise. Dr. Otto, of Copenhagen, in his interesting paper in the Transactions of the Provincial Medical Association, on the medical topography of that city, says, "that only two persons died of aneurism in 1835, and none in 1836." Judging from the reports of cases in the Spanish and Italian Journals, that I have been able to procure, aneurism is much less prevalent in the south of Europe than in England.

Aneurism of the Thoracic Aorta.

Of the 67 Thoracic Aneurisms in the last table, 36 were in the ascending aorta, including six cases where the arch was also implicated.

11 of these burst into the pericardium, 3 into the œsophagus, 1 into the trachea, 1 into both pleuræ, 1 into left pleura and lung, 1 into the right auricle, 1 under pectoral muscle, 1 externally. In 16, death appeared to occur from gradual sinking, or from pressure upon the trachea, bronchus, or respiratory nerves.

Of the 21 aneurisms at the arch of the aorta, 4 burst into the trachea, 3 into the left pleura, 2 into the œsophagus, 1 into the left bronchus, 1 into pulmonary artery, 1 into pericardium, 1 died of tubercles of the lungs, 1 man committed suicide, and in 7 cases death probably arose from sinking, or from pressure upon neighbouring parts.

Of the 10 aneurisms of the descending aorta, 1 burst into the left bronchus, 1 into left lung, 1 into right pleura, 1 patient died of pneumonia, and in 6 the cause of death seemed to be from pressure upon the nerves, or surrounding organs.

The general statistics of the 241 cases of thoracic aneurism, afford the following results; 134 were in the ascending aorta, 69 at the arch, and 31 in the descending thoracic.

Occupations.—Unfortunately, in many instances, the narrators have omitted to state the occupation of the patient, but the following is the *resumé* of all the avocations that are mentioned in the 241 thoracic aneurisms. Ladies 2, wool-worker 1, nurse 1, gentlemen 14 (including 3 medical practitioners,) soldiers 15, sailors 12, labourers 8, shoemakers 5, porters 4, coachmen 4, gardeners 4, carpenters 4, sawyers 3, masons 3, tailors 3, slaters 3, bakers 2, butchers 2, travelling clerks 2, hawkers 2, mechanics 2, blacksmiths 2, innkeepers 2, locksmiths 2, policemen 2; and one of each of the undermentioned trades—silversmith, builder, glass-cutter, bricklayer, carman, nailer, cotton weaver, corn-chandler, keel-man, lath-render, waterman, cattle-dealer, quarryman, coalheaver, wheelwright, paper-hanger, smith, lock-filer, and trombone player; not stated 116. It should not be forgotten, that the occupations of those among the better classes are always named.

The subjoined account gives the termination of all the cases:—burst into the following parts; pericardium 44, (43 of these aneurisms were in the ascending aorta,) œsophagus 14, trachea 14, left pleura 14, right pleura 8, both pleuræ 1, left bronchus 5, right bronchus 1, pulmonary artery 5, right lung 3, left lung 1, superior cava 4, right ventricle 2, left ventricle 1, right auricle 3, posterior mediastinum 2, under pectoral muscle 1, burst externally 8. In 111, the sac did not give way; the greater number of these deaths being occasioned by congestion of the heart, pressure upon the trachea, bronchi, lungs, and respiratory nerves; the remainder died from serous effusion, apoplexy, pneumonia, pericarditis, dysentery, tubercle, violence, and other complications.

The most practical inference from the above statistics, is, that a vast number of patients die of aneurism, without rupture of the sac. It is a curious circumstance, too, that only 8 of these 241 aneurisms burst externally. Of the 31 patients affected with aneurism of the descending thoracic aorta, (which I believe generally arises from violence,) only three were females; one of these, (Dr. Lyon's case,) was found in the dissecting room, and the aneurism is described as a fusiform dilatation; in a patient of Dr. Walshe's, æt 28, the aneurism was in the descending portion of the arch, and in Dr. Fuller's patient, æt 33, the dilatation (about the size of a walnut,) was about an inch below the arch,

and filled with old coagula of decolorized fibrin. In my former table of 21 cases, all those affected with aneurism in this situation were males. The evidence just afforded is an additional reason for the extension of statistical inquiries.

Dissecting Aneurism.—In the last table there are four cases of dissecting aneurism; 3 males and 1 female, their ages 57, 54, 44, 38; making, when added to the 21 examples, (p. 167,) in the former table, 25 cases; 15 of them occurred in females, and 10 in males; 17 of these aneurisms (so called,) burst into the pericardium, but when these lesions, which are not strictly aneurismal, are deducted from the report, even then, this form of rupture is the most common, and if the dissecting aneurisms are included, rupture into the pericardium is thrice as frequent as into any other part.

Symptoms and Diagnosis. If we are to believe some writers on aneurism, the diagnosis of this disease is generally unattended with difficulty, and it is possible that by long practice, and an acute sense of hearing, *they* may be able to detect the presence of aneurism, although most ordinary listeners fail to do so; and, amongst this number, I class myself—for I am obliged to agree with the assertion of the great Laennec, "that there is no sign pathognomonic of thoracic aneurism, except the external tumor." I admit that a tolerable degree of accuracy may be arrived at in many instances, but there is no certainty of the existence of aneurism, unless the sac pushes out the parietes of the chest.

Dr. Bellingham, whose experience and zeal in the investigation of this disease, entitle his remarks to great attention, believes "that there is a double sound in thoracic aneurism, irrespective of the sounds of the heart; that the second sound is produced by the backward current of the blood; that the aneurism is comparatively unfilled; and that the aneurismal sac is inelastic." Dr. Bellingham, whose object, like my my own, is the investigation of truth; will pardon me, I am sure, for differing from him in all these opinions. I believe that a double *bruit*, seldom or never exists in thoracic aneurism, irrespective of the heart's sounds. What nicety of hearing can distinguish the sounds in aneurism of the ascending aorta, (the most frequent situation,) from those of the heart? How an aneurismal sac can be "comparatively unfilled," and the sac at the same time be inelastic matter, are beyond my powers of comprehension? I could amuse the reader, if I were to quote the description of the various sounds heard by different narrators, but this double sound, strange to say, has generally been undetected by English ears. Gendrin believes that a double sound *(bruit de choc)* is always present in all aneurisms of the large arteries; and, to shew how far hair-splitting may go, he says, in *true* aneurism, (?) there is but one impulse and one sound, whilst in *false*, there are two sounds and two impulses. Dr. Bellingham objects to Gendrin's explanation of the second sound, and attributes it to regurgitation into an inelastic, and unfilled sac; and he accounts for the absence of a double sound in abdominal aneurism, by supposing that a sufficient quantity of blood does not regurgitate into the sac. But, surely, in the horizontal position, regurgitation would be as likely to take place in the abdominal aorta as in the arch, or descending portion of the thoracic? It is scarcely necessary to stop to refute the opinion, that an aneurismal sac is inelastic—its very throb is an indication of elasticity: that portion of it formed by the cellular coat is inelastic; but not so the yellow tissue of which nearly all aneurisms are partly composed. In an anurismal sac now before me, which has been in Goadby's solution for several years—I find the greater part of it very elastic.

As two peas in a bushel, two pebbles on the sea shore, and two aneurismal sacs, are never exactly alike, so are the auscultatory phenomena influenced by the form of the dilatation, the quantity and consistence of its contents, the condition of its walls, and the state of surrounding organs. Let the reader examine a few hundred specimens of aneurism and then enquire whether exactly the same sound *could* be present in any of them?

When a patient, whose symptoms, lead the practitioner to believe that thoracic aneurism may exist, the first interrogation should be, as to the occupation and previous habits of the individual, and he should be particularly questioned respecting the occurrence of blows, falls, or violence of any kind; the examiner always remembering that small thoracic aneurisms may exist for a long time without producing much inconvenience, and that in some cases where the yellow tissue of the artery only is ruptured, little or no pain may be present. It is probable that many aneurisms originate in early life from the partial fracture or separation of the fibres of the elastic coat, when the artery is free from disease. After the above enquiry the blood vessels of the arms should be carefully examined, and the veins of the neck must not escape observation. The palms of the hands may then be passed over both sides of the chest, for the purpose of detecting abnormal impulse and protrusion of the thoracic walls. The stethoscopic examination along the spine is most important, but it must be recollected, that the cardiac sounds are sometimes heard in all parts of the chest. After careful percussion and auscultation, if abnormal *bruit* is detected, the effect of position and motion should be tried, and the question asked, whether the *bruit* may not arise from valvular, or other disease of the heart, from a roughened state of the arterial tube, or whether a tumor (probably malignant) may not, by interfering with the blood-current, occasion it? Œdema of the hand, pain in the arms and fingers, the condition of the voice, the appearance of the countenance, the character of the pain (if present,) state of the breathing, hæmoptysis, previous diseases, and hereditary predisposition of the patient, are all important subjects for investigation.

If the above matters are carefully attended to, I think *generally* a correct opinion may be arrived at in the diagnosis, but it is always easier to say that this disease is *not* present, than to foretel its existence.

I caution my junior readers against the inference that any sounds, alone, are to be taken as pathognomonic of thoracic aneurism. Double sounds, single sounds, in all varieties from the grating rasp of the saw, to the murmur of the gentle breeze may be heard, and yet not denote aneurismal dilatation. Common sense and pathological observation, as as in all medical deductions, are more important in this inquiry than "*sonoriety*."

Since the former treatise was written, I have had only one case of internal aneurism that terminated fatally under my care; although, during the three years that I have been Physician to the Metropolitan

Dispensary, I have seen five patients, whom I have reason to believe were affected with thoracic aneurism, but as no external tumor existed in any of them, I cannot speak positively upon the subject. Through the kindness of some of my medical friends, however, since the period alluded to, I have had the opportunity of inspecting several cases of aneurism. It would be of little benefit to add to the twelve examples already recorded, as those who are especially interested in the subject, can find abundant information of this kind in the Medical Journals; I will narrate the undermentioned case, for the purpose of showing the difficulty that attends the diagnosis of thoracic aneurism, in the absence of external bulging of the parietes of the chest.

April 30, 1847.—I was requested to see Mrs. P., Walworth, æt. 40, who has generally enjoyed good health, but has been affected for some time past with cough, and occasional hæmoptysis, and is supposed by her friends to be labouring under phthisis: she is now confined to her bed for the greater part of the day; she has not lost flesh, and her appearance is that of a strong muscular woman. The two sides of the chest are equal in size, the pulse about 80, and the pulsation of the radial arteries is similar, both as regards force and frequency. I find on a careful examination of the chest, that the heart's impulse is rather increased, but the sounds are normal; the respiratory murmur is natural on the left side, but absent on the right, where there is also dulness on percussion, especially at the upper part. I can detect no *bruit* unconnected with the heart's sounds; but my friend Mr. Taylor, of Camberwell, who also saw the patient, thought he could hear a distinct, single *bruit* on the right of the sternum; I inferred from this examination that the right lung was consolidated by previous inflammation, or that a large aneurismal tumour existed at the arch of the aorta, but I inclined more to the former opinion. She remained much in the same state, the cough being troublesome, with occasional expectoration of blood. On the morning of the seventh day, after I saw her, she expectorated about a tea-cupful of florid blood, and in the evening, my brother, Mr. F. Crisp, of Walworth, was called to her in haste, but she was dead when he arrived, having lost a large quantity of blood from the mouth. I was compelled to leave London before the inspection of the body, but Mr. Taylor has kindly furnished me with the account of the autopsy.

"Right pleuræ adherent throughout—lung carnified, like liver, no air in it—an aneurism existed in the ascending aorta, situated just under the spot where the *bruit* was heard, it was rather larger than the fist, and would contain about half a pint of blood—it communicated with the aorta by an opening the size of a half-penny. The walls of the aneurism were thin in parts, the substance of the right lung in fact forming the external wall, as the aneurism projected into it. An ulcerated opening with jagged, bony edges was found communicating with a large bronchial tube, and a portion of cretaceous deposit, the size of a nut, was present at this part. The aorta had numerous atheromatous deposits on it. Heart small, valves healthy. Left lung crepitant throughout, highly congested, and on cutting it, a large quantity of bloody serum escaped. No tubercles in any part of either lung. Liver enlarged from congestion. The other viscera not examined."

Soon after the occurrence of the above case, I saw the following, which, for the purpose of contrast, I allude to; the presence of aneurism in this instance, being placed beyond a doubt, although I lost sight of the patient and could not verify my opinion by a post mortem examination. September, 1847, G. A., a stout muscular man, a painter by trade, many years since, fell from a two-story window and injured his back, but he was soon able to resume his usual employment. He is now suffering from cough, quickness of breathing, wheezing, and occasional pain in the chest and down the right arm. The pulse at the right wrist is absent, and there is no pulsation in the brachial artery. There is a dull sound on percussion under the right clavicle, and a slight bulging of the ribs on the right and upper part of the sternum; a strong impulse is heard in this situation, but no *bruit*. The sounds of the heart rather louder than natural. He appeared to derive some benefit from the application of a Belladonna plaister to the chest, but medical treatment could be of little avail in such a case. The absence of pulsation in the arteries of the right arm, combined with the other symptoms, was almost a sure indication of the presence of aneurism at the arch of the aorta. Cases 1, 2, 3, and 11, also shew the importance of a careful inspection of the blood-vessels of the upper extremities, in all cases of suspected thoracic aneurism; a mode of diagnosis, strange to say, not specially alluded to (as far as I know) by any previous writers upon this subject.

At page 130, I have stated, that in ten aneurisms, that I had seen a short time before the publication of this work, I had only heard the double sound in one, and in this case (supposed aneurism of the aortic arch) it was not constant. When in Dublin for a few months, in 1848, I had an opportunity, on the same day (November 23rd) of seeing five patients at the Meath and St. Vincent's Hospitals, who were affected with aneurism; they were all strong muscular men; a trombone player, two sailors, a gentleman's servant, and a journeyman distiller, their ages, 40, 45, 50, 51, 60, the first was a supposed case of abdominal aneurism, near the cœlic axis; the patient had pains in the back, shooting down the legs and thighs; there was a strong heaving impulse and a single *bruit* over the tumor. The others were affected with thoracic aneurism, and in all, there was a partial bulging of the thoracic walls on the right of the sternum. In the second case there was a loud impulse over the tumor, but no murmur; in the third patient, the heart's sounds were distinctly heard over the sac, but no other sound; in the fourth case the cardiac sounds were also present over the tumor, and in the fifth, there was a single rough bellows sound almost approaching to musical. There was nothing remarkable about the heart's sounds, in any of these patients; the pulse at the wrist was natural in three; in one, the right radial artery was less voluminous than the left, and in another, there was no pulsation in the left carotid, and the pulse at the left wrist was scarcely perceptible. The radial beat did not exceed 85 in any of them; in one only, the voice was stridulous. All had been spirit drinkers, and accustomed to hard labour. One, three years before, fell with his arms erect between the wharf and a steam-boat, another first perceived pain and pulsation after carrying a heavy box to the railroad station; a third was a trombone player; the fourth accustomed to carry heavy weights, and the fifth, a sailor æt. 45, had never, so far as he remem-

bered, had a blow or a fall of any kind. I could add to these, the case of a man of intemperate habits, who had been a gentleman's servant, whom I saw at the same time, under the care of my friend Dr. Lees, at the Meath Hospital; there was impulse, and slight dulness on the right of the sternum, and the second sound of the heart was rough, and a little prolonged, but as no external tumor existed, a positive opinion could not be given. I saw the morbid parts after death, in one of these patients (the trombone player,) who had a large tumor on the right and upper part of the chest, which threatened to burst; the voice was stridulous, and he died apparently from pressure upon the recurrent laryngeal nerves, without rupture of the sac. The aneurism was as large as an infant's head, and commenced in the ascending aorta; the arterial trunks at the arch were not plugged, but the vena azygos was obliterated, accounting for a fulness which existed in the epigastric veins.

It is well to add, that the above statements are made from notes *taken at the time*, and *not from memory*. I should also remark, that some, whose opinions would receive more attention than my own, thought there was a double sound in all the last-mentioned thoracic aneurisms.

I could add several cases to the above, but I have selected these, as they were all examined on the same day. This morning, (September 28th, 1852,) through the kindness of Mr Barnard Holt, I have seen a young man under his care at the Westminster Hospital, who labours under aneurism, probaby, of the ascending aorta. There is a prominent, pointed, pulsating tumor, on the right of the sternum, with thin parietes. The radial arteries are synchronous, and their distension equal; but the pulsation, to use Dr. Billing's term, is rather resilient. On a careful examination, I can discover no *bruit*, and the two heart-sounds are distinctly heard over the tumor, the second, being a little rough and prolonged. Mr. Holt tells me, that he has never heard any *bruit* in this aneurism, an opinion confirmed by others who have examined the patient.

The reader will not misunderstand me, I hope, upon this question of double sound. I do not deny the occurrence of a double sound in some cases, over an aneurismal sac, but I think it is scarcely possible to separate these sounds from those of the heart, more especially in aneurism of the ascending aorta. It should be remembered, too, that in many aneurisms of the thoracic aorta no *bruit* is present; a certain amount of impulse (action and re-action) must exist in all aneurismal sacs, as in the arterial tubes, but this is not necessarily accompanied by *bruit*.

Abdominal Aorta.

I have but little to add to my remarks on this subject (p. 161) more than to reiterate the opinion that this aneurism arises generally from injury done to the coats of the artery from great exertion, blows, falls, and other kinds of violence, and that although in most cases a single *bruit* is present over the aneurismal sac, in some instances no abnormal sound has been heard. The present table includes fourteen cases, and the dilatation, as in the former examples, was generally seated near the cœliac axis. Of these fourteen patients 10 were males and 4 females, their ages 26, 29, 30, 32, 32, 33, 34, 34, 35, 46, 50, 51, 64, in one the age not named. Four burst into the peritoneum, four into the left pleura; one female (Mr. Holt's case) had also aneurism in both popliteal arteries, and died after ligature of the femoral; one died of Bright's disease, four others died without rupture of the sac. The reader can easily add these statistics to those of the former table. It is well to remark, that of sixty-one cases out of the seventy-three, in which the terminations are given, fourteen burst into the peritoneum, eleven behind this membrane, and nine into the left pleura.

Cerebral Aneurisms.

The additional six cases of aneurisms within the skull include four males and two females; their ages 18, 33, 35, 35, 52, in one case the age is not given. Occupations—a tailor, a painter, and a milliner. Rupture of the sac took place in all of them, producing apoplectic symptoms. In the eight cases in the former table, (p. 164,) all the subjects of this form of aneurism were males. Nearly all these aneurisms, I believe, arise from blows upon the cranium. It is scarcely necessary to remark, that there are no means of diagnosing these lesions of the cerebral arteries. As two cerebral aneurisms (plate 4) have occurred in two patients under my care, it is a fair inference that they are often overlooked, in post-mortem examinations. If I had not been attending especially to the diseases of the arteries, they might have escaped my observation.

Treatment of internal Aneurisms.—The remarks respecting treatment (p. 137) include nearly all that I have to say upon this subject: the remedial measures can only be palliative, but life may be prolonged generally, I think, by therapeutical means, and the fact that out of 241 cases of thoracic aneurism, the sac did not burst in 111, should teach us not to abandon any measures that may suggest themselves for prolonging life. Among the external means, I may mention tracheotomy, when life is threatened by pressure upon the trachea or laryngeal nerves; the application of starched linen, gutta-percha (warmed,) or plaister of Paris, when the sac threatens to burst, and attention to the position of the patient (especially in abdominal aneurism) so as to endeavour to diminish the impulse of the blood on the thin parts of the sac, may be thought of. I am not aware that this plan has ever before been suggested, but I think it will be found worthy of consideration hereafter.

In these non-depleting times the very mention of the lancet will frighten some of my readers, but the abstraction of blood in small quantities, by removing congestion and giving freedom to the circulation, is sometimes attended with marked benefit in this disease. When in Dublin, Dr. Stokes told me of a woman under his care in whom rupture of a thoracic aneurism took place externally, and was attended with enormous loss of blood; a patient in the ward, however, had the presence of mind to plug the opening with tow, and thus arrest the bleeding. The woman's life appeared to be prolonged by the hemorrhage, as she lived for some time after. A well-regulated diet, and great attention to the digestive functions, are most important in all cases. Medicine can be of little avail in this fearful disease, except "to smooth the path to the grave," but the judicious and attentive practitioner may often find means, I believe, to assuage suffering, and prolong life, although he cannot save it.

External Aneurisms.

With the exception of popliteal aneurisms, the additional cases are so few, that it will not be necessary to speak of them under separate chapters. Of the three aneurisms of the carotid, two of the patients are females, and one a male, (making in all fourteen females and fourteen males,) their ages 32, 35, 60; these last-mentioned patients all died; two after ligature by Messrs. Fergusson and Hunter, and in the other, the aneurism was mistaken for an abscess, and punctured.

I have expressed my belief (p. 213) that the comparative frequency of this form of aneurism in the female, arises from the bare state of the neck, rendering the artery more exposed to blows. As the reviewer in the American Journal of Medical Sciences remarks, "my statistics do not correspond with those of Dr. Norris," but he appears to forget that this writer has included some cases of traumatic aneurism.

Of the two subjects of aneurism of the innominata,* both males; in one (Mr. Wishart's patient) a spontaneous cure was nearly effected, and in the other case death occurred without rupture of the sac. Of the two subclavian aneurisms, both also in males, the sac burst externally in one, after oozing of blood for fourteen days. In the second case the man refused to be operated upon.

In the six additional aneurisms of the axillary artery, making altogether twenty-four cases, twenty-three males and one female; twelve were on the right side and eight on the left, and in four, the situation is not stated. The six last reported cases were all males; their ages 29, 34, 34, 47, 49, 50. In four, the subclavian artery was tied successfully, and two of the patients (Mr. Hancock's and M'Kensie's) died from hemorrhage after the ligature of this vessel. For further statistics I refer the reader to page 212.

The femoral aneurisms amount to six; five males and one female, two in the left, one in the right, side. Ages 34, 37, 56, 60, 74; the occupation of one only, a vice-man, is recorded. The external iliac artery was tied successfully in two instances. One aneurism burst externally; in two cases no operation was performed, and one man committed suicide. For the general statistics of the seventy-two cases see p. 224.

Popliteal Aneurism.

This analysis of the forty-five cases of aneurism of the popliteal artery last recorded, with the addition of the 137 examples before given, will form one of the most practical parts of this appendix, for in this investigation the deductions are generally so satisfactory, that even those most prejudiced against statistical inquiries can scarcely object to them.

Of the forty-five cases in the present table, only two were females; in three both arteries were aneurismal, in nineteen the right was affected, in twelve the left, and in eleven, the side is not stated. The femoral artery was ligatured in fifteen cases by Messrs. Adams, Clarke, Partridge, Avery, Hilton, Lawrence, Allan, Northgate, Judd, Syme, Syme, Drummond, Syme, Togood, and Holt; four of these patients died; two from gangrene, one of gangrene and tetanus, and one of suppurative inflammation. Of the eleven successful cases, one lost a leg,

* For the best account of this aneurism yet published, I refer the reader to an excellent paper by Dr. Holland, in the Dublin Journal, 1852, which includes an analysis of forty-five cases, British and Foreign.

one had gangrene of the toes, and one hemorrhage from the cicatrix and neuralgia of the foot. It must be observed, however, that some of these operations were performed under disadvantageous circumstances, and that pressure had been sometimes tried (often imperfectly) before the ligature was resorted to.

Of the remaining cases, four had amputation successfully performed without ligature of the femoral; one died from spontaneous bursting of the aneurism; one of tubercle of the lungs, (without operation,) one of hemorrhage from puncture of the sac for supposed abscess. In two others, in whom amputation was successfully performed, the sac had been punctured for supposed abscess.

Twenty-three of the above forty-five cases were cured by pressure.

General Statistics of the 182 *cases of Popliteal Aneurism.—Males* 176; *Females* 6. *Age.*—Between 20 and 30, 40; between 30 and 40, 79; between 40 and 50, 34; between 50 and 60, 11; over 60, 3; and in 15, the age is not given. In 59, the aneurism was on the right side: in 56, on the left; and 14 were affected with aneurism in both arteries. In 53, the side is not named. *Occupations.*—Sailors, and waterman 26, soldiers 17, labourers 10, porters 8, blacksmiths 4, gentlemen 3, coachmen 3, bakers 3, shoemakers 3, gardeners 3, brickmakers 3, servants 3, farmers 2, carpenters 2, tailors 2; and one of each of the following—brewer, coachmaker, miller, policeman, flesher, coalheaver, carter, horse-keeper, groom, carrier, stonemason, carman, plumber, bricklayer, rough-rider, green-grocer, plaisterer; carrier, shop-keeper, cotton-weaver, cow-keeper, mealman, brass-founder, rope-maker, organ-builder, work-house pauper, cutler, pipe-maker, watchman, skin-dresser, calico-printer, ostler, glass-blower, cooper, street-musician, sawyer, butcher, dressmaker, portress and *postillion*. In 49, the occupations are omitted. In Mr. Holt's case of double popliteal aneurism, (which has accidentally been inserted under the head of abdominal aneurism also), the woman, a portress, performed the work of a man, or rather that of a horse; and after death, a small aneurism filled with coagula was found likewise in the abdominal aorta.

Result of treatment.—Of these 182 cases, 25 terminated fatally, and 4 were cured spontaneously; the femoral artery was ligatured in 106 instances; 16 of these patients died, and in 8 others amputation was performed successfully after the ligature of the femoral. In 5, amputation was successfully practiced without ligature. For other statistics, respecting the causes of death, I refer the reader to p. 226.

Pressure.—39 of these cases were successfully treated by pressure: and although 3 of the patients died subsequently of disease of the circulatory organs, the deaths cannot fairly, I think, be attributed to the treatment employed. I would, however, suggest, as a precautionary measure, in all cases where aortic aneurism or disease of the heart is suspected, the abstraction of blood from the arm, before the application of the tourniquet.

The reader must bear in mind, that the above were spontaneous aneurisms, (so called), but if femoral and traumatic aneurisms were included, as well the unpublished cases and those recorded in the American and other journals, the number of cures would be greatly increased; indeed, if the cases of Guattani, Sabatier, Dubois, and Tyrrell, mentioned at p. 180, are added, they will not be far short of a hundred.

Mr. Tufnell, of Dublin, in his recent work "On the treatment of aneurism by compression," gives a table of 39 cases treated in Dublin,

from 1842 to 1851, and he states, "that during this period the ligature was only resorted to three times." In 30 of the patients the cure was perfect and complete. In 2 instances, the ligature, and in three, amputation was had recourse to with success. In one man the pressure was discontinued; two (above mentioned) died of co-existing disease of the heart; and one of erysipelas, but in this case (Dr.Bellingham's) Galvano-puncture had beed used."

What a pleasing contrast does the above mode of treatment afford, when compared with the ligature? How much does the investigation shew the value of statistical inference? In my first table, 1844, six cases only were included; in 1847, the number had increased to twenty-one; and now it amounts to forty-four, (all these being endogenous aneurisms.) I quote my remarks upon this subject in my Prize Essay, written in the year 1844.

"Cure by pressure has been effected in six cases of external aneurism; two in the gluteal, and four in the popliteal arteries; the time varying from six to ten, weeks. The author thinks that this mode of treatment might be more frequently resorted to with benefit to the patient. Several cases, in addition to those in the table, have been related by other surgeons." I suggested at this time the use of a spiral wire truss, a drawing of which is given, that might be made so that the apex of the pyramid pressed upon the artery. I also advised, in aneurism of the external iliac, where the ligature cannot be applied, the compression of the artery below the tumor, and the use of gentle pressure at the same time upon the sac. I cannot now refer to a successful case thus treated, which I have recently seen recorded in one of the American Journals.

After the very great success that Guattani met with, combined with the statement of Mr. Freer, of Birmingham, in his work on Aneurism, in 1807, "That such were the extraordinary advantages of compression, that when any portion of vital power remained, he knew of no exception to its use;" it is most remarkable that this mode of treatment has been so long in abeyance. To the Dublin surgeons and physicians, and to Dr. Bellingham especially, are we indebted for fairly testing and demonstrating this *great* surgical improvement. I anticipate that at no distant period, a coroner's inquest will be held upon the body of any person who dies after ligature of the brachial artery, for chronic traumatic aneurism.

Galvano-puncture.—A few words only will be necessary upon this mode of treatment, introduced chiefly by M. Petrequin, of Lyons, 1845; the result fully bears out my anticipation at page 187; for, although it has succeeded in many cases, yet the effects sometimes have been so disastrous, that it is not likely to be generally resorted to. In the *London Medical Examiner*, among the abstracts of the Foreign Journals for the last three years, several allusions to this practice are made, (vol. 1, pp. 66, 192, 236, and vol. 2, pp. 23, 137, 217, 248,) but the cases of Professors Velpeau and Scutin are of great practical interest. M. Roux has lately stated at the Paris Academy of Sciences, "that electro-puncture should be banished from the treatment of aneurism."

The next Appendix will include the statistics of the cases of aneurism in the American, French, German, and Italian Journals, with additional remarks on diseases of the Veins.

October 22nd, 1852.

REPORT

OF THE

HUNTERIAN ORATION,

DELIVERED BEFORE

THE MEMBERS OF THE HUNTERIAN SOCIETY,

AT

THE THIRTY-THIRD ANNIVERSARY,

FEBRUARY 4TH, 1852.

BY W. J. LITTLE, M.D.

PHYSICIAN TO THE LONDON HOSPITAL,

ETC. ETC.

LONDON:

PRINTED BY ROBSON, LEVEY, AND FRANKLYN,

GREAT NEW STREET, FETTER LANE.

PAGE

Sounds of the Heart—Homœopathy; Hydropathy; Mesmerism; Kinesopathy, and other Quackeries—Expectancy—Medical Charities: causes of previous want of 3-13

Venous Injections into the Veins—Transfusion: Sketch of the History of both these processes, from 1621 to 1849—Venous Injections of Saline and Alcoholic Fluids in malignant Cholera 14-34

REPORT,

ETC. ETC.

Mr. President and Gentlemen,

In responding to the call of duty, to maintain this annual custom of the Hunterian Society, I experience at the outset considerable diffidence as to the manner in which I shall endeavour to justify the confidence you have placed in me; and when we consider the great number of able men who have preceded me in my present vocation,—who have made similar occasions the opportunity for developing the hidden treasures of their thoughts, their aspirations, and their experience,—such hesitation may be excusable.

Of the manner in which my distinguished predecessors have shed lustre upon the proceedings of our Society, and have especially dignified the Anniversary Meetings, allow me to cite (and I do so with peculiar pleasure,) one single instance, as a perfect type of what, in my apprehension, a Hunterian Oration ought to be. I allude to the Oration which I heard pronounced here twenty years ago, by my teacher and friend Dr. Billing, in which he gave to the world the valvular explanation of the sounds of the heart; an explanation long combated by his contemporaries, but which they have gradually adopted; an explanation of the acoustic phenomena accompanying the heart's action, which has so strikingly facilitated the diagnosis of the diseases of that organ.

Such is the masterly example which at the present moment I would wish to follow, although with the fullest consciousness that it must be at a humble distance; for it is conceded to few of us to discover the meaning of an important fact which shall mark a period in the history of a particular disease.

This annual gathering of the members and friends of the Hunterian Society affords, amongst other advantages, an opportunity of reviewing some matters of general interest to our profession which have occurred during the past year. Amongst these we may survey the progress or decadence of empiricism, within and without our ranks. The stand is at length made against Homœopathy. It is a subject of congratulation, nevertheless, that the profession has so long left this quackery unnoticed. In the land of its origin its hollowness and falseness were soon apparent, and the monster absurdity was strangled almost in its birth. Here no master-mind like Stieglitz has until lately deemed it worthy of exposure and refutation. Homœopathy has, in fact, been abandoned to its course; left by physicians to mere contempt. In this tolerant community, however, the scorn of the profession has been mistaken for fear; but as long as the freedom of thought and action conceded to Homœopathy was confined to itself — as long as Homœopaths were content to be Homœopaths — as long as its mysteries were confined to a separate sect, who made no claim to belong to the regular ranks, but who rather boasted of their divergence from us,—it was not desirable that our profession should openly notice it; but when Homœopathy seeks to invest itself with the mantle of orthodoxy; when it aspires to invade the halls of our universities, and to instil its virus into the minds of our alumni, it is full time for the profession to oppose with energy the double imposture. This opposition has now become a necessity—nay, indeed, it has become an imperative duty; for the tendency of the delusion, with its exaggerated belief in the efficacy of drugs, and its assertion of the potency of infinitesimal doses of medicinal agents, is to engender universal scepticism as to the value of medicinal substances.

As in theology itself, so in medicine, extremes will meet: the larger and the more unreasonable the demands made upon the credulity, the imagination, and the superstition of the disciple, the greater his proneness to fall into utter disbelief.

With the profession fully alive to the tendency of the new claims of the followers of Hahnemann and others; with the

certainty that hereafter both sides of the question will be kept before the public; the ultimate result cannot be doubtful:— "Magna est veritas, ac prævalebit!"

That portion of society which has yielded to the seductions of this quackery will be undeceived, whilst another generation is springing up to imbibe perhaps some new folly, which, in its turn, will share the inevitable fate of Homœopathy — to be consigned to the contempt and derision of society, as soon as its novelty has subsided, and the halo of mystery which surrounds it has been dissipated by the steady radiance of Truth: moreover, the mere multitude of Elixirs vitæ, Homœopathy, Hydropathy, Mesmerism, and other *isms* without end, must in time bewilder the beguiled, and eventually lead to the regular physician for refuge. The number of the Homœopathic pretenders is said to have increased, yet the interest to credulous or dishonest men to continue to labour in the diffusion of these absurdities must diminish as their number is augmented; and even such men will find that legitimate medicine is equally or even more profitable in a pecuniary sense; when, of course, they will make a virtue of necessity, and return to the beaten path of science and of common sense. The spread of so many quackeries in the present day, even among educated men, has occasioned surprise; yet when the inducements to empirical practice are so numerous, and possess such solid attractions, there is little cause for any astonishment. All educated men are not proof against evil temptations, nor inaccessible to sordid motives.

Besides, every system of quackery may contain some element of good, however much that element may be obscured or abused: Homœopathy, for example, pretends to cure with globules, whilst the really effective agents which it can wield are pilfered from the armoury of the regular practitioner; and indeed, from this point of view, each so-called system is but an additional compliment to regular medicine, an unintentional homage to that true system of the healing art established by the unwearied researches of devoted inquirers on the basis of knowledge and sound philosophy. Thus each pretended system is but a gigantic hypocrisy unconsciously rendering, as it

were, the usual tribute of vice to virtue. Each consists but of a leaf taken from the venerable book of medicine—the accumulated and recorded experience of ages—and prostituted to the purpose of acquiring a distinction unattainable by honourable rivalry and competition in the arena of legitimate practice; or as an excuse for the non-possession of the education, the acquirements, or the diploma of the regular practitioner,—a leaf expanded into a huge system of credulity and imposture. These terms would be harsh if applied to the members of any sect rightly claiming to be cultivators of science; but surely those of the Homœopaths who really believe in the efficacy of infinitesimal doses,—of doses so small, a dilionth or decilionth, for example, whose very expression in figures notation can hardly bring upon its tablets, may at least be justly termed credulous; whilst the remainder, if they have not so debased their intellect as to credit the doctrine, are plainly obnoxious to the charge of imposture, for their adoption of a practice repugnant to their own common sense, merely as a profitable livelihood.

The most charitable supposition would be, that such intelligent men as have embraced Homœopathy had previously become sceptics as to the value of drugs, under any mode of administration; following in this the theory of those physicians who assert that the average duration of disease and the average mortality of mankind (all other things being equal), is the same, whatever may be the medicines administered. This scepticism of the value of drugs may, by some, be regarded as an excuse for practising with them in a harmless form, relying upon expectancy, diet, and hygiene.

Now this covert reliance upon time, diet, and hygiene, constitutes the hypocrisy of Homœopathy. It is true that expectancy, or the belief that nature alone is competent to cure every curable disease, and intuitively to teach what aliments to withhold and what to administer, has long existed as a doctrine in medicine; and when legitimately entertained and modified, it holds a place in the creed of every rational practitioner.

Another important leaf in our book teaches the use of water, both internally and externally, in the cure of disease. The belief in this agency has been universally disseminated;

a proof of which we have in the regard of every known people and tribe for those natural health-giving streams, so abundantly supplied by a beneficent Creator, and recognised and venerated by pilgrims and sages in every age and clime. Water has been employed in almost every variety of form in legitimate practice, and it is not matter of surprise, that crafty men, from the recognised value of water, have thereon constructed a pretended system, laying claim to powers well nigh miraculous.

So, again, with Mesmerism: do we not know the wonderful sanitary effects produced in numerous functional disorders by impressions made upon the mental and nervous systems, more especially upon those of susceptible individuals, and the alarmingly noxious influence at other times exercised upon the same class of persons by similar means? It is not necessary to remind you of the Wuthtanz in Germany, the Dancing Mania of Scotland, of the practising exorcists of the middle ages, and of the connexion between these phenomena, and the physiological influence exercised by the eloquence, the example, or the will of powerful spirits (noble or ignoble), either upon groups of individuals, or upon whole nations, exciting them to acts which pain, dazzle, or astonish a succeeding generation.

We do not forget nor fail to explain the cure of even structural diseases by the royal touch, nor the arrest of intermittent fever by so obscure an agent as the snuff of a candle. If we are familiar with these mental, physiological, and even curative influences of faith in the remedy, can we not comprehend the success of Mesmerism and of Electro-biology; and need we feel surprise, that an inflated leaf from our own book should pretend to the dignity of a separate science—a separate *methodus medendi?* Again, who amongst the ranks of regular medicine is not aware of the value of exercise of the body in the preservation of health and the cure of many functional and congestive disorders, the sure forerunners of disorganisation, if neglected? Who is not aware of the danger arising from the possession of luxurious couches, splendid easy riding equipages, retinues of servants, calculated to render superfluous

the use of the owner's limbs, and to leave him in full possession of Dyspepsia from negative repletion and voluptuous idleness? Which of us is ignorant how much happier in mind and stronger in body is the humble labourer, who, as Shakspeare's care-sick monarch exclaims,

> "From the rise to set sweats in the eye of Phœbus,
> But all night sleeps in Elysium!"

and how much the latter might be envied by his inactive though richer neighbour? Or, knowing these things, shall we be accused of ignorance of the value of temperate and well-regulated gymnastics in the relief of disease? Are we to be seduced into repeating the experience of that would-be medical sage who, according to Plato, first destroyed himself by exercises of undue violence, and afterwards destroyed many others by his pernicious example?

Yet in the present day a system of gymnastics, called 'Kinesopathy,' is gravely propounded for the cure of disease! Behold the abuse of another leaf from our book,—our book, I say, because "rich with the spoils of time," it is the repository of all the recorded wisdom of our noble art; and each and every pretended new system will be found, if scrutinised, to rest upon the abuse of some page, abstracted without acknowledgment from its almost exhaustless treasures.

This is the secret of the partial or temporary success of every quackery, Morrisonianism, Brandy-and-Saltism, Hollowayism, White Mustardism, St. John Longism,—quackeries in every way worthy to be classed with Homœopathy and Hydropathy.

Gentlemen, we can perhaps hardly dismiss this topic in more appropriate words than those of a profound and eloquent contemporary: "O my brother, be not thou a quack! die rather, if thou wilt take counsel: 'tis but dying once, and thou art quit of it for ever. Cursed is that trade; and bears curses, thou knowest not how long ages after thou art departed, and the wages thou hadst are all consumed; nay, as the ancient wise have written,—through eternity itself, and is verily marked in the doom-book of a God!"

But although our book contains these costly pages, each of them of such value when the proper moment of application arrives, can we each of us individually claim the merit of properly using each of these means when the occasion does present itself? Whilst our noble art is, from its innumerable resources,—the bountiful gifts of Providence,—of priceless value to mankind, we its humble votaries, the feeble instruments of diffusing these blessings, are fallible; we cannot each, individually, embrace the whole expanse of medical knowledge; we are unable to be expert in the prescription and administration of every means within our reach. On the contrary, we find in practice that the most successful physicians are often those who have learned how best to employ certain agents, whose confidence in their weapons thus becomes perfect, and who work wonders by the skill and experience they have thereby acquired.

This habit of employing chiefly certain means, and the success consequent thereon, often engender neglect of other means, perhaps of the highest value; hence Homœopathy, Hydropathy, Morrisonianism, &c. remind and admonish us not to be too exclusive in our choice of agents; but as far as practicable to combine experience or regular empiricism with rational medicine,—a combination the neglect of which has led already to scepticism in our own ranks; thus Pneumonia has been adduced as an instance of a disease being unaffected in its progress by medicinal agents. It has been asserted that in whatever mode this disease be treated, the physical changes in the lungs will advance or decline, disappear or kill, without appreciable hindrance from medicine, and that the average mortality from this cause will be the same.

This opinion has been countenanced by a too exclusive attention to physical examination of the patient during life, and of the lungs after death,—the observation having been conducted rather as a matter of natural history than of true clinical study.

It is erroneously asserted that, if one hundred cases of inflammation of the lungs be treated by blood-letting, or by mercury, tartar-emetic, nitre, &c., the sufferings of the patient

will be similarly protracted in every case, and death result in an equal number of instances, whichever of these plans of treatment be adopted.

We admit that, if one hundred cases of inflammation of the lungs, taken indiscriminately, be irrationally treated upon one and the same plan, merely because the disease be inflammation of the lungs, irrespective of age, sex, habits, temperament, previous disease and complications,—the duration of the disease, and the mortality produced by it, will be the same, whichever plan of treatment be selected. But if the physician avail himself both of physical examination, to determine the nature of the disease, and also of his knowledge of morbid anatomy, to remind him in what direction the disease tends; if he at the same time profit by the experience of his own and of former times; if he dismiss preconceived notions of the disease necessarily demanding a certain treatment, and investigate each case upon its own merits;—he will assuredly treat each case according to its obvious and peculiar indications: he will wait upon nature in one case; will bleed or use tartar-emetic in a second; depend upon mercury in a third; trust to nitre, salines, and alkalis in a fourth; he will even support a fifth through the disease (an habitual tippler, for example,) by the aid of brandy; or he will combine several of these remedies at different stages of the same case, and will unquestionably find that the average duration of the disease, and the mortality from it, will be far below the estimate of those who treat all cases alike. In fact, in this as in every other disease, setting aside the infallible necessity of death at some period or other, recoveries are in proportion to the care with which the cases are investigated, watched, and treated,—in which the observation of the nurse is combined with the intelligence, the experience, and the varied resources of the physician.

It has often been justly observed, that medicine would indeed be a simple art, if it were only necessary to arrange or enumerate the symptoms, to find the name of the disease, and to search out, in a sort of Encyclopædia, for a particular remedy for a particular form of disease. No; our art is not so easy. Nay, its very difficulties, its very uncertainty, have exposed us to the not over-flattering criticism of the wits and witlings of every country and every period, from Terence to Molière, Le Sage, and Dean Swift. Even brave old Michael Cervantes himself has a fling at us. Is it not in *Don Quixote* that the intendant of the hospital, consulting the chief physician, says, "What, sir, shall we do this week?" "What," demands the doctor, "did we do last week?" "We physicked the north ward and bled the south." "Then bleed the north ward and physic the south."

It may be supposed that the very earnestness of the pursuit, in which the members of our profession are engaged, and the consequent zeal with which doctors have maintained their opinions, independently of the influence fairly attributable to egotism, have indelibly impressed our differences of opinion upon the public mind, and furnished poets and satirists with the opportunity of ridicule at our expense. In truth, we have been selected as the butt of derisive mirth, because the exercise of our art has necessarily been conducted openly, in the presence of the people, ever shrewd enough to detect the weak side of individual conduct, if not of medical practice. Thanks, however, to the agency of education, and to the wonderful discoveries by which mind and matter are so rapidly and extensively moved in the present day, the turn of other learned professions, to be similarly tested and weighed in the balance of public opinion, has at length arrived. The screens behind which other professions performed, to a great extent, their share in the multiplex drama of life, have fallen down one after another, and the public have become aware, that not doctors alone disagree, but also theologians, judges, barristers, naturalists, engineers—men mighty, both in conception and achievement, notwithstanding that all these professions have to deal with matters supposed to rest upon unchangeable *Leges scriptæ*, or upon the fixed forms of the animal, vegetable, and mineral kingdoms, and the immutable laws of mathematics and mechanics. That all these differ as widely as doctors differ, the world is, in fact, beginning to comprehend; that the very variety of intellects, the incalculable number of different proportions in which the formative elements of mental existence are combined, no two persons being identical in

mind, any more than in body, induce the tendency to examine every branch of knowledge under different aspects; this analysis, as it were, of every department of human inquiry, finally leading to agreement, because leading to truth.

The past year is also memorable as that in which the greatest effort has been made to found and maintain charities for the relief of decayed and unfortunate members of our profession. It will ever appear remarkable, that whilst our profession has been mainly instrumental in the establishment of charitable institutions for the relief of nearly all "the ills that flesh is heir to," we have hitherto incurred the opprobrium of having effected little for the succour of our own suffering brethren, their wives and families. The Society for the Relief of Widows and Orphans in London and its vicinity is a noble and successful illustration of what effectual provision may be secured for unforeseen destitution. Who can presume to say that himself or his widow or his children may not one day require the relief thus extended to others? Still, this society, like the British Medical Fund, is mainly based upon the principles of a provident association, and not upon those of a purely benevolent institution.

The Provincial, Medical, and Surgical Benevolent Fund, a purely benevolent establishment, has strong and peculiar claims upon the support of every medical practitioner. But the earnest attention of the profession has been fully awakened to the necessity of further combined exertion, with the aid of the general public, by Mr. Propert's excellent scheme of a Medical Benevolent College, which has been commenced under the best auspices, and for whose perfect success the best augury has already been pronounced.

The enthusiasm with which this admirable scheme has been received, the unanimous approbation both of our profession and of the public, certainly indicate that the previous want of such a charity has not originated from the absence of generous emotion among our members; from no indifference to the examples offered us by other professions and commercial bodies; nor even from the fact that, owing to a large portion of the labours of medical men being unrequited in a pecuniary sense, and the comparatively large expenses of our

literary, intellectual, and social position, we remain essentially a poor profession, incapable of insuring ourselves and our dependants from the casualties of this life. No! It is rather a fact which exhibits the weak side of our body politic.

We are an unorganised profession—not that we have not medical corporations in excess; each, however, having established some claims to our respect, by the higher scale of qualifications they have demanded of candidates for honours: in this respect acknowledging the necessity of keeping pace with the general demand of the present day for improved education. But unfortunately these corporations are not governed by the thousands who belong to them; and I believe that one great cause of the absence of benevolent institutions, worthy of a great and noble profession, springs from the want of sympathy and the want of habitually active concurrence in matters of moment to the profession between the represented and the unrepresented,—the want of the habit of appealing in matters of common interest to the thousands of intelligent, industrious, and influential surgeons and physicians distributed over the whole kingdom. If we glance even at the numerous ancient and originally trading corporations of this metropolis, we find that, under different schemes of organisation, the representative system has prevailed—the wants, feelings, and wishes of all classes have influenced, and ultimately determined, the conduct of the governing bodies; and amongst other admirable results of that principle (which is, in fact, the vital principle of responsibility), we find the endowment of educational establishments on a princely scale, and the establishment of benevolent funds for the accidental misfortune or decadence of their members.

It can scarcely be doubted that, with such an extended intercourse between all classes of the profession as would be created by a full representation in our corporations, there would result an increased influence of these bodies in the promotion of science, in the advancement of the intellectual and social claims and position of the profession, and (co-equal in importance) the active recognition of the duties of our body towards its less fortunate members.

Many subjects of minor importance may be passed over. No great medical discovery has been promulgated during the past year, although a silent progressive improvement is perceptible in every department. I believe, indeed, that in no profession a more healthy striving after truth is apparent than in that of medicine at the present moment.

Gentlemen, I am unwilling to occupy your time entirely with a review of matters affecting merely the economy of our profession; I shall, therefore, solicit your attention for the remainder of the hour to a matter of more scientific interest.

It is acknowledged to be serviceable to the progress of our art, occasionally to trace the rise and fluctuating reputation of a mode of treatment not generally recognised, but which has been applied in some of the emergencies of practice. And, therefore, whilst placing upon record some facts illustrative of injection into the veins of the human subject, I will avail myself of this opportunity to bring to the notice of the Society some details from the history of this operation and of a subject closely allied thereto, namely Transfusion of Blood. I shall not attempt a complete sketch of the history of venous injection, or infusion into the veins, as it was more commonly termed, still less of that of transfusion of blood; but these two processes have mutually exercised a great influence, and their history is, as it were, so much intermixed, that in reviewing the origin of one of these therapeutic processes, I am almost necessarily led into some details concerning the other. I will refer those of my hearers who desire further information on the subject to the works of Scheel, Dieffenbach, Birch, Denis,—to whom, amongst others, I am indebted for many statements I shall lay before you.

The earliest advocates of the method of introducing blood or other substances into the veins, have endeavoured to invest the matter with additional importance by tracing its discovery to the remotest times. The origin of venous injection is, in fact, involved in the obscurity common to so many other inventions: conjectures are not wanting in the absence of facts.

In the middle ages, the friends of infusion and transfusion were delighted to refer their discovery to the renowned magician Medea, whilst their opponents stigmatised venous injection and transfusion along with worse deeds of this fabulous personage. Jason having implored Medea to prolong the life of his aged parent Æson, she is said to have restored him to youth by drawing away the old blood from his veins, and refilling them with the juices of certain herbs. The words of Ovid are—

"Quæ simul ac vidit, stricto Medea recludit
Ense senis jugulum; veteremque exire cruorem
Passa, replet succis, quos postquam combibit Æson
Aut ore exceptos aut vulnere; barba comæque
Canitie posita nigrum rapuere colorem."

Ovid is also supposed to allude to transfusion of blood, when he relates the delusion by which the daughters of Pelias were led to the commission of parricide:

"Quid nunc dubitatis inertes?
Stringite ait gladios: *veteremque haurite cruorem,*
Ut repleam vacuas juvenili sanguine venas."

Whilst, on the one hand, the silence of other ancient authors, and the exceeding improbability that at so early a period of the world's history as that of the Argonautic expedition, this bold operation could have been performed, lead us to regard Ovid's account simply as a creation of his poetic fancy, affording an early instance of a powerful imagination anticipating by ages the discovery of an important truth; it is, on the other hand, probable that, at a subsequent period, Ovid's assertion may have suggested the actual performance of the operation.

The prevailing theories of the sixteenth and seventeenth centuries respecting the nature and treatment of disease, and one article of faith derived from the Mosaic writings, namely, that the seat of life is in the blood, as well as the internal use of blood or medicines prepared from blood—a practice revived in the present day in the proposition to use dried ox-blood in anæmia,—combined to suggest transfusion and injection of materials into the blood, as incomparable methods of acting promptly and immediately upon the source of life, the heart.

Probably some of the superstitious arts of sympathy then practised may have conduced to the same result. I may here mention one of these singular practices, worthy to be ranked with the pretensions of that modern art, by which the electric telegraph is superseded by the agency of snails. I allude to the ancient art of enabling two persons residing at any distance apart instantaneously to communicate with each other. Both persons made a slight wound in any convenient part, and permitted a few drops of each other's blood to be reciprocally instilled into the wounds, which were then allowed to heal. It was believed that if either pricked his own scar, the other instantaneously felt the same, and by a previous arrangement as to the number of pricks, understood the meaning of the other.

The first distinct proposition to effect transfusion of blood was made in an anonymous publication in Germany, at the beginning of the seventeenth century. The mode of operation proposed by the anonymous discoverer, supposed to be Pergelius, a physician at Rostock, was fully described in 1615 by Libavius, a physician at Halle. The honour of the discovery belongs, therefore, to Germany.

The more important part of the history of infusion and transfusion commences with the epoch of the great discovery of the immortal Harvey. His doctrine of the circulation of the blood satisfactorily demonstrated the possibility of total renewal of the circulating fluid of an animal, and that a remedial agent injected into the veins would gradually affect the whole of the blood, and be thus distributed to every part of the body. Harvey's discovery also taught the most direct mode of performing these operations, whilst transfusion itself was regarded as a confirmation of Harvey's doctrine.

We may picture to ourselves the influence exercised by the coarse humoral pathology of those days, and the extraordinary experiments to which it led. It was asserted that old and worn out animals could be fitted for the chase; that the sterile woman would be made fruitful; that the old women who had long ceased to bear children would be again rendered capable of conception; and even that princes would be rendered wise by the operation. The most enthusiastic expectations both of infusion and transfusion were formed by the leading physicians in most countries. But unfortunately so large a number, not only of useless, but of unreasonable and mischievous experiments were carried out both upon men and animals, that instead of attributing the unfavourable results of infusion and transfusion to the erroneous modes in which they had been employed, physicians ultimately lost all hope of obtaining through them results beneficial to the art of healing, and suffered them almost to lapse into oblivion. Nevertheless, eager disputation respecting the merit of the discovery ensued, for which almost every nation advocated its claim.

The actual performance of the operation of infusion first occurred in Germany, in 1642, twelve years after Harvey's discovery. In every country the injection into the veins of medicinal and of other substances preceded the operation of transfusion.

The canine race afforded the subjects of the first operation. Infusion was first employed as an amusement by a German sportsman. The fluids injected consisted of wine and brandy. The animals slept off their innocent debauch, and experienced no evil consequences from the operation. This is perhaps a solitary example of an important physiological discovery resulting from an otherwise disreputable trifling with animal feeling.

Strict justice, which awards to the regular consequential working out of a discovery the palm of invention, attributes to our own countrymen the honour of having, in 1656, first successfully introduced venous injections and transfusion of blood to the notice of the scientific world.

As early as 1638, Potter had fruitlessly proposed transfusion.

Wren, the celebrated architect, spontaneously discovered and practised infusion into the veins of a dog at Oxford, in 1656. The well-known natural philosopher Boyle witnessed Wren's experiments, and described the effects of the injection of a solution of opium dissolved in sherry, which was followed by brief stupor and perfect recovery of the animal.

Boyle subsequently tried Crocus Antimonii (oxysulphuret) upon a criminal. He dissuaded from the use of strong substances, and recommended cordials, antidotes, and alteratives, and mentioned even diuretics, as proper materials for injection.

Clarke tried injection of milk, beer, whey, broth, and attempted transfusion.

Lower, commencing his experiments with infusion, found that beer and wine could be introduced without danger; and, in 1666, he was the first who successfully effected transfusion in an animal. In this satisfactory instance of transfusion, Lower directly connected the cervical artery of one dog with the jugular vein of another, and gradually introduced the whole of the blood of the one animal into the vein of the other, allowing at the same time the blood of the receiving animal to escape in a proportionate quantity, until he had successively parted with and gained a quantity of blood equal to his own weight. The receiving dog afterwards leaped down from the table, caressed his master, and in order to clean himself from blood, rolled in the grass, as he would have done after leaving the water. The dog remained perfectly well.

Some years after our countrymen had zealously and perseveringly laboured to determine the merits of the new discovery, the French embraced it with all the ardour of their genius, and had the courage to effect transfusion in man, from which operation the rest of the European nations had hitherto shrunk. Their bold experiments promptly diffused an universal knowledge of transfusion; but the unreasonable manner in which it was advocated, and the low cabals of its opponents, contributed speedily to extinguish all consideration for the operation.

Bourdelot, the first French *claimant* for the honour of the discovery, alleges singular reasons for the proposal of transfusion. He states that the idea occurred to him after a conversation with Gallalei, respecting the injection of sundry purgative and alterative medicines; and adds, that if it be possible to cure paralysis by plunging the part into the reeking entrails of a slaughtered animal, transfusion of blood must needs be more effective. This extraordinary reasoning did not abash

him. It is not remarkable that he should have believed, with the priests of the temples of antiquity, that a paralysed limb might be cured by immersion in the warm trunk of an animal, —an opinion and practice not yet exploded.

The first French physician whose labours in this new field attained great notoriety was Denis; he first effected transfusion in the human subject in 1667, having successfully introduced small quantities of the blood of calves and lambs into the veins of five persons, who experienced no injurious consequences from the operation. The enemies of Denis asserted that in one case fatal results had ensued. The minds of the profession and the public were much excited by the exaggerated pretensions of the friends of the method, and the startling denunciations of opponents. Legal proceedings were even taken against Denis; but he succeeded in clearing himself from the imputations of his persecutors, and was afterwards appointed physician to the king.

The court of justice now decreed that the operation should only be performed with the sanction of a member of the Parisian or other competent faculty; and at a subsequent period, it was erroneously stated that the court of law had even interdicted the operation.

Denis transfused the blood of animals in a case of diseased brain with soporose symptoms; in a healthy person; in an insane; in a woman affected with paralysis (hemiplegia); and in a nobleman who had laboured for three weeks under bilious diarrhœa and vomiting. The last case, apparently the least favourable for the experiment, was nevertheless the most interesting, as an illustration of the capabilities of transfusion of even the blood of animals. Denis, believing in the existence of organic disease, refused to operate until the physicians already in attendance had declared the case to be hopeless. By the injection of six ounces of calf's blood, the patient was rallied from a state of speechless prostration and unconsciousness; he took nourishment, slept, and for twenty-four hours appeared to be going on favourably, after which, however, the diarrhœa and prostration returned. Denis unwillingly yielded to the solicitations of the patient's family, and repeated the operation, which afforded only twelve hours further respite from death.

An impartial survey of the literary effusions of the period leads to the conclusion that Denis experienced much injustice at the hands of his medical brethren, and that notwithstanding his exertions to promulgate the new operation, its repute fell so suddenly and so completely in Paris, as powerfully to influence its fate in other countries.

After the fate of transfusion had been sealed in France, the public and the profession went, as usual, from one extreme to the other, and rejected even the good which might have been obtained from a limited and careful employment of transfusion in the art of healing.

In France, the less assuming operation of infusion into the veins received little attention; and the experiments that were performed, consisting of the injection of concentrated acids and alkalies, melted tallow, and undiluted alcohol, represent experiments calculated rather to determine in what manner the introduction of these substances would destroy life, than to illustrate their use as a means of preserving it.

The anxiety entertained in our own country lest the life of a human being should be endangered by an experiment, and perhaps also the fear of judicial proceedings, easily instituted in a country where the value of human life is with justice highly estimated, delayed the performance of transfusion in England.

At length, in 1667, a crack-brained Cambridge undergraduate, thus described by Dr. Lower, "hominem amabili quadam vesania affectum," volunteered himself for the experiment. Dr. King operated in the presence of numerous distinguished personages, and introduced into the veins from eight to twelve ounces of calf's blood. No evil effects were experienced. Three weeks afterwards, the operation was repeated with the same result. Coga, the subject of the operation, subsequently addressed the Royal Society in testimony of his recovery. As might have been expected, his mind was not affected, beneficially or otherwise, by the proceeding.

The news of the discredit into which the operation of transfusion had fallen in France did not extinguish in our country all interest concerning it; but rather diverted the attention of

physicians to infusion of medicinal substances into the veins of animals. Death was found to result from injection of olive-oil, muriate of ammonia, salts of tartar, saltpetre, alum, camphorated spirit of wine, and tincture of hellebore; whilst the animals recovered or experienced no injurious effects from the injection of vinegar, sugar, common salt, urine, opium in small doses, or diluted alcohol. Injection of senna-infusion was succeeded by vomiting and purging.

At the same period the Germans occupied themselves with venous injections. Major, in 1668, first applied this operation in the human subject. He inserted into the veins "liquorem quendam nervino-balsamicum:" the patient is said to have recovered, but no further particulars of the case are upon record.

Elsholz successfully injected three soldiers: the first with aqua plantaginis ℥j, for the cure of a long-standing ulcer; the second with infusion of carduus benedictus ℥ss for a fever; and the third with infusion of cochlearia for scurvy. The recovery of these three persons without unfavourable symptoms after the injections cannot be contested. We may without injustice to Elsholz believe that the small quantity of materials used had little or no direct share in the recoveries, but that moral and other (physical) agencies more powerfully contributed to cure.

Elsholz, in his conclusions on the value of the method, recommended injection of stimulants in fainting from heart-disease, and of refrigerants in fever; thought it might be usefully employed in apoplexy; in hysteria; in anginas impeding deglutition; in consumption, on account of the facility with which the remedy could be introduced almost immediately into the lungs; and, as a climax, suggests the reciprocal transfusion of a few ounces of the blood of married couples, or of brothers who have quarrelled, in order to insure future harmony between them.

Garman suggested that asphyxiated new-born infants might be restored to life by injection of a few drops of wine by the umbilical vein. He tried the experiment in an asphyxiated puppy, and successively introduced some wine, laudanum, and a purge. The animal was restored. Garman recom-

mended transfusion after profuse hemorrhage, and sagaciously dissuaded from injection in consumption and marasmus.

Dr. Schmidt, better known by the cognomen of Fabricius, a physician at Dantzic, received permission from the authorities of that city to make experiments at the public hospital. He selected a robust soldier suffering from advanced secondary syphilis. He injected eight grains of scammony in ʒiij of tincture of guaicum. The patient experienced vomiting, but recovered. The second case was one of epilepsy, which was similarly treated, with equal benefit. In three cases, namely, rheumatic gout, apoplexy, and plica polonica, he employed with success, in the same manner, alterative agents.

The history of this period abounds in curious illustrations of the fact, that at every stage our profession has been infested with scientific quackeries. Such appears to have been the nature of the practice of one Hoffmann,—whether the inventor of the celebrated anodyne that bears his name, I am unable to say. Hoffmann undertook to cure diseases by transfusion according to a comparatively safe method, consisting merely of the introduction of a few drops only of the blood of a healthy person, which he asserted acted as a new ferment "quasi per insitionem," by which the entire mass of diseased blood was converted into healthy blood.

Kauffmann effected some successful as well as some unsuccessful transfusions of sheep's blood into the human subject. He speaks, however, of the unsuccessful living for years afterwards, affected with what he termed "sheep's melancholy."

Purrmann, who had assisted Kauffmann, remained so fully convinced of the value of venous injections, that in 1670, whilst suffering from obstinate cutaneous disease, he caused himself to be injected with a few spoonfuls of aqua cochlearia and spiritus theriacalis, after preliminary blood-letting. During the operation he was seized with syncope, which he attributed to the too rapid injection of the fluid; and he also sustained some suppuration of the vein

Upon another occasion, whilst engaged at the siege of a fortress in Pomerania, after suffering sixteen weeks from ague and dysentery, he caused the operation to be again performed

upon himself with a few spoonfuls of aqua cardui benedicti, and recovered in a few days, without either syncope or suppuration. In 1670-80, at the Halberstadt hospital, Purrmann successfully injected three patients affected with intractable epilepsy. He repeated the operation three times on each patient; the materials employed being small quantities of vegetable infusion, alcohol, and ammonia.

In 1691, Müller first injected quicksilver in animals, and observed the formation of points of suppuration in the lungs around the metallic globules,— an experiment repeated by Dr. Haighton of this metropolis in 1799, and revived a few years since by Cruveilhier.

Infusion and transfusion were much used in Italy for physiological purposes. The renowned Baglivi expressed surprise that the method had not made greater progress in the cure of disease. He observed that injection of cold water induced rigors, and made numerous experiments with introduction of air into the veins. The danger attending the presence of air in the heart became thenceforward fully known throughout Europe; and its forcible injection by the jugular vein was practised as a humane method of slaughtering animals, because of the instantaneousness with which death resulted.

Some courageous physicians at Marseilles, in 1721, whilst pursuing their investigations concerning the dependence of plague upon a morbid state of the biliary fluid, communicated that disease from man to dogs by venous injection of the contents of the gall-bladder.

Jean Marie Regnaudot, in a dissertation, published at Leyden in 1778, entitled *De Chirurgia Infusoria renovanda*, described a case of Oriental leprosy, the cure of which he attributed to venous injection of senna, guaicum, and mucilage.

Fuller, in 1785, recommended venous injection as a last means of restoration in cases of suspended animation from drowning,—an idea carried out at Halle by Meckel, the father of the illustrious physiologist of that name. Meckel succeeded in effecting vomiting, as a means of reaction, by injection of two grains of tartar-emetic into the veins, and thus rescued

a woman from the asphyxia consequent on an attempted suicide by drowning; other appropriate remedies for suspended animation having been simultaneously adopted.

A remarkable instance of recovery of a youth, aged fifteen, from the effects of the bite of a viper, by the injection of a drachm of hartshorn into the veins after the symptoms were desperate, is recorded by Valisnieri.

In 1776, Kohler illustrated, in a remarkable manner, the value of venous injections of emetic substances. He was called to a man in whose œsophagus a large piece of gristly flesh had become impacted, the dislodgment of which by ordinary surgical means had failed. The man was speechless, with fluttering pulse and cold sweat. Dissolution seemed imminent. Kohler having witnessed some experiments with venous injections, instituted for physiological purposes by the celebrated Lieberkühn, injected six grains of tartar-emetic into a vein. Half an hour after the operation the piece of gristly flesh was ejected with great violence. The man recovered, and was alive twelve years afterwards.

Balck, a military surgeon at Potsdam, who had heard of Kohler's case, was, in 1784, hastily summoned to a young soldier, who had swallowed a large piece of flesh, containing, as the result shewed, a projecting bone. The mass had become impacted midway in the œsophagus. Balck repeated the attempt which had been made with the probang to dislodge the mass; but the sufferings of the patient were aggravated by the operation. The man was unable to swallow any thing, even a drop of fluid, and resembled an apoplectic patient; his face was turgid with dark blood, his eyes projected from the sockets, dyspnœa was distressing, anxiety and restlessness were indescribable, and, in Balck's judgment, speedy death was inevitable. In this extremity Balck injected a solution of tartar-emetic into the right median vein: a quarter of an hour afterwards nausea, followed by violent vomiting, set in, during which the sufferer indicated by signs that the obstructing mass had been ejected. The vomiting notwithstanding continued, and occasioned much exhaustion; and was only finally arrested by the exhibition of laudanum and ether.

On the following day the man was well, with the exception of the feeling of weakness.

Two years after this occurrence, Balck was called into consultation in a similar case: a woman, upwards of fifty years of age, who had endeavoured in vain to dislodge an impacted mass by attempting to swallow several hard crusts. The probang, and other usual remedies, had been tried without success. Some hours having been passed in a state of severe suffering, Balck resorted to injection into the veins, as in the last case, and with equally good effects.

A fourth case of this kind occurred in 1796, to Knopf, at Freistadt: it differs only in the rapidity with which vomiting was excited. The lump of flesh ejected was as large as a hen's egg.

The well-known Berlin surgeon, Gräefe, was equally successful in a precisely similar case at Berlin in 1816.

Considering the comparative infrequency of death from this obstruction of the gullet, whilst gluttons abound every where, it is fairly open to inquiry whether some of these semi-strangulated persons might not have recovered without the venous injections. It should, however, be remembered that the alarming symptoms present before the venous injections, have been detailed by evidently competent persons. Gräefe, for example, was personally known to living surgeons in this country as a great ornament to his profession. In accounting for the large number of five similar cases, it should also be remembered that they are spread over a period of forty years, and that the diet of a nation may increase the liability to this particular accident.

Hemman, also a German, published some very rational views respecting venous injections. He regarded as suitable all medicines which are readily miscible with the blood, and which effect no visible change in the mixture and relations of this fluid. He successfully illustrated his views by injecting an ounce of aqueous solution of musk into the veins of an epileptic girl: this was followed by restoration of catamenia and recovery.

Hemman's second case was more remarkable. A man,

æt. 39, previously of robust constitution, lay, on the twelfth day of typhus fever, soporose, and with involuntary flow of urine and fœces. He had ceased to swallow, the pulse was scarcely perceptible, and the trembling of the flexor tendons, added to the above symptoms, shewed that he had not long to live. Hemman prepared an infusion of bark, one ounce to a quart of water, to which, after thrice filtering, he added two drachms of hartshorn. He injected into the veins three ounces of this fluid: half an hour afterwards the corpse-like face shewed some signs of colour; large drops of sweat stood upon the forehead; the pulse rose; the skin, previously dry and shrunken, became moist; a profuse perspiration covered the surface; and in three hours later the patient aroused from the sopor as if from a deep sleep; he was then able to swallow a few spoonfuls of wine, and appeared generally improved. The same night delirium again supervened, the patient became restless, the skin was again dry, and the pulse fell. As the symptoms continued unfavourable, on the following day Hemman prepared a solution of ʒj of "the essential salt of quinia" in four ounces of distilled water, repeatedly filtered it, and added 15 grains of carbonate of ammonia. He reopened the vein at the elbow, and after allowing the escape of four ounces of blood, injected three ounces of the prepared solution. One hour after this second injection the pulse rose, and the patient appeared better; copious perspiration ensued, and, during the succeeding night, an offensive diarrhœa. A trifling suppuration occurred at the bend of the elbow; but from the moment of the second injection the patient gradually and completely recovered.

Darwin, in his *Zoonomia*, proposed transfusion in threatened starvation from stricture of the œsophagus; but the patient, Atticus-like, wisely preferred to die unrelieved.

Lynn, a celebrated surgeon at the close of the last century, witnessed much relief from the injection of ʒj tobacco-infusion into the veins of a horse labouring under lock-jaw, but the animal died before the operation could be repeated.

Viborg, of Copenhagen, a veterinary surgeon, obtained a series of interesting results from venous injections employed

therapeutically in animals. The problem to be solved was the production of vomiting and perspiration, acts excited with difficulty during the diseases of pachydermata and ruminantia. He ascertained that by injections of solutions of hellebore into the veins of horses and cows, vomiting and perspiration were readily induced; from which a powerfully salutary influence in the cure of many of their diseases was derived.

Some experiments of Ortel of Leipsic, published in 1804, may be briefly passed over. Among other cases, he recovered a person from sopor of fourteen days' duration; a case for which the usual remedies had been used, including counter-irritation and actual cautery, and for which trepanning had been contemplated. The recovery was effected by the injection of six grains of camphor triturated with mucilage.

Dieffenbach, who ably reviewed Ortel's cases, expresses surprise that so much camphor should have been tolerated without fatal disturbance of the respiration. It is, in my opinion, a matter of equal surprise, that although some animals have survived the injection of these substances, the individual in question should have borne with impunity the introduction of mucilage into the blood, a substance of all others likely to occasion mechanical stoppage of the circulating fluid and coagulation of blood. It is not improbable that coagulation of blood in the veins, thus induced, may have prevented the camphor and mucilage reaching to the vena cava, the heart and lungs, and thus have averted fatal consequences.

The elder Hufeland and Horn, both well-known physicians of Berlin, instituted an instructive series of experiments with venous injections at the Charité Hospital in that city. They thus treated insane, hysterical, melancholic, and epileptic subjects; and selected those in whom medicines could in no other mode be introduced, or whose condition suggested that improvement might result from a prompt and powerful revolution of the entire organism. Tartar-emetic, camphor, nux-vomica, ammonia, opium, carbonate of potash, and henbane, were the chief substances employed. Some patients were injected several times without a single fatal result; a large number of the patients were decidedly improved. The injec-

tions were practised with every precaution, and witnessed by numerous competent observers; it is, therefore, the more to be regretted that ample details of these cases were never published.

Percy and Laurent, authors of the article on venous infusion in the *Dictionnaire des Sciences Medicales* (1813), state that they tried venous injections in the cases of eight Russian and French soldiers affected with tetanus, at a period when nearly all cases treated in the ordinary way proved fatal. Of the eight cases injected, five recovered. The authors thus very justly express themselves respecting the value of venous injections in tetanus: "Which, among all the remedies which have been successively vaunted in this formidable and fatal disease, offers so much encouragement as the plan we have described?"

Well might they experience gratification and hope, after obtaining in tetanus five recoveries out of eight cases! They injected wine, stramonium, extract of bark, digitalis, and valerian.

It is a subject of regret that no perfect and detailed statement of these cases, whether successful or unsuccessful, was published by them.

Krahe of Berlin, in 1820, recovered a girl from epileptic apoplexy by injection of tartar emetic. Magendie's well-known laudable, yet futile attempt, to save a hydrophobic patient by venous injection of two pints of tepid water, occurred about this time. Although unsuccessful, it is, as Magendie stated, a remarkable fact, that a sufferer in the worst stage of this terrible and rapidly mortal disease, was placed in a state of perfect calm during eight days by the injection of two pints of water. Possibly the result might have been different, if Magendie had been aware of the liability of pure water to cause destruction of blood-corpuscules, when brought into contact with them. Microscopic observation teaches that only fluids of suitable density can be brought with impunity into contact with blood-corpuscules. Had salt water been used, therefore, the issue might have been otherwise. It is impossible to over-estimate the impulse that may be given to the practice of venous injections, by judicious application of the knowledge of the properties of the component

parts of the blood, displayed by microscopic and chemical investigations.

Froriep, in 1826, vainly endeavoured to rescue a woman affected with hydrophobia, by injecting an infusion of belladonna; and Gaspard, for the same disease, uselessly injected five ounces of tepid water.

M. Coindet unnecessarily injected about seven grains of of white poppy-juice into the veins of a girl labouring under hysterical trismus: the patient recovered, without injury from the process.

An unfavourable opinion may be passed upon the case of Meplain, a physician of Donjon, who injected four grains of tartar-emetic into the veins of a child convulsed from intestinal worms; the worms were expelled by vomiting, the convulsions ceased, and the child recovered notwithstanding.

Dr. Cholmely's unfavourable instance of transfusion, in a case of marasmus from schirrhus of the pylorus, at Guy's Hospital, is deserving of mention only as an opportunity of deprecating the operation in a case of evident organic disease: for although it is reported that no abdominal tumour was discernible during life, the extreme gradually occurring, marasmus of itself too clearly indicated the existence of organic disease in some part of the economy.

The temporary beneficial influence of the transfusion may, however, have suggested its trial by Dr. Blundell, whilst attached to Guy's Hospital. Dr. Blundell's experiments and writings on transfusion are too well known to my hearers to need full mention here; his praiseworthy efforts to disseminate a knowledge of that operation having been crowned with success. He had the happiness to save a woman flooding to death; an example successfully imitated by Dr. Uwins and Dr. Waller.

But venous injections were most numerously prosecuted during the cholera epidemics of 1831-2 and 1848-9.

Dr. O'Shaughnessy, after careful analysis of the blood in cholera, being aware of the previous application of venous infusion in other diseases, proposed to re-oxygenate the dark carbonised blood of cholera by venous injection of a solution

of chlorate of potash. He subsequently recommended trial of weak alcoholic solutions.

Dr. Latta of Edinburgh has the merit of having taken the initiative in the practical recognition of Dr. O'Shaughnessy's recommendation of venous injection in cholera. Dr. Latta had the boldness to open a vein in the elbow, and to infuse pint after pint of a solution of common salt and carbonate of soda, salts of which the blood had been deprived during the vomiting and purging of that disease. The reviving influence exerted by the injected fluid was so strikingly apparent, and the expression of his relief by the sufferer so much exceeded the most sanguine expectation, that Dr. Latta was encouraged to persevere; hundreds of ounces were thrown into a single patient in the course of a few hours. In nearly every case the same extraordinary relief was obtained, although in the majority the improvement was only temporary. The journals of the period shew, that of fifteen cases of cholera-collapse operated by Dr. Latta, one-third recovered. I will here quote Dr. Latta's description of the phenomena observed during the injection. His words are these:

"There is at first but little felt by the patient, and the symptoms continue unaltered until the blood mingled with the injected liquid becomes warm and fluid. The improvement in the pulse and countenance is almost simultaneous; the cadaverous expression gradually gives place to appearances of returning animation; the horrid oppression at the præcordia goes off; the sunken, turned-up eye, half covered by the palpebra, becomes gradually fuller, till it sparkles with the brilliancy of health; the livid hue disappears; the warmth of the body returns, and it acquires its natural colour; words are no more uttered in whispers; the voice first acquires its true cholera tone, and ultimately its wonted energy; and the poor patient, who but a few minutes before was oppressed with sickness, vomiting, and burning thirst, is suddenly relieved from all distressing symptoms."

Every physician practically conversant with venous injection in cholera, will admit that this forms no exaggerated picture of the primary effect of the process upon many apparently hopeless cases of cholera-collapse.

Many practitioners followed Dr. Latta's example. I may

mention Dr. Tweedie, in whose hands one case in four recovered; Dr. Craigie, who had one recovery and one death; Dr. Murphy, two recoveries and one death; Dr. Girdwood, four recoveries and three deaths; and Mr. Arthur, thirteen recoveries and five deaths. My own experience of venous injection in 1832 was not favourable, as regards the permanent recovery of the sufferers. Three times in 1832 I had recourse to this method, and as often assisted others. One patient only finally recovered.

In 1832 I was induced to add alcohol to the injected solution, in consequence of the small and transient re-action I had in some cases observed after the injection of common salt, carbonate of soda, and chlorate of potash. The result of my experiment with alcohol convinced me that it constituted an additional and powerful means of arousing the dying heart and nervous system. The reaction after injection of a mixture of hot water, salines, and alcohol, was more lasting than after injection of water and salines only.

In 1848 I could not but witness with regret the impending return of cholera. Yet, as a medical man, retaining a vivid impression of the effects of venous injections which I had seen in 1832; convinced that an agent capable of so beneficially modifying the human organism even for a time, would, after due investigation, be found to possess a therapeutic value; and equally convinced that no other means yet discovered possessed the faculty of even occasionally rescuing from death persons already moribund,—these considerations did, to me, invest the approach of cholera to our shores with an interest which only the members of our profession, accustomed to successful encounters with death, can appreciate. At that period of public anxiety, the belief in the value of venous injections was a ray of hope amidst the depressing emotions and the gloom produced by the re-appearance of this fatal sickness. I did not anticipate the cure of cholera by venous injections, either with or without the addition of alcohol; but I trusted that, as in the last stage of typhus, when the fever has actually departed and the patient is sinking from inanition, the tottering balance inclining to the side of death, the patient is often

rescued by the timely administration of stimulants;—that as, upon the battle-field, many a wounded combatant has been saved from exhaustion and death by the heavenly esteemed gift of a timely draught of water;—or that, as a syncope which might otherwise prove fatal is removed by opportune lowering of the head, by means of which a feeble yet adequate flow of blood is transmitted to the brain,—so, in like manner, fortified by the experience of 1832, I hoped that in the approaching cholera we might be able, by venous injection, at least to save lives that were inadequately struggling into reaction.

The greater number of physicians who have resorted to venous injections in cholera have recommended their employment at a comparatively early stage, before the collapse of this terrible disease has become most intense; and probably the larger proportion of the recoveries sometimes witnessed has resulted from this practice.

Favoured by the excellent arrangements adopted by the authorities of the London Hospital on the approach of the epidemic, and also by the zeal of the officials, from the house-governor Mr. Hill, the resident medical officer Mr. Birch, to the humblest nurse, I was, during the height of the epidemic, August 1849, induced to inject a few cases. I preferred observing the effects of the treatment in a few of the worst kind, to trial of its efficacy in a larger number of mixed cases. The total number injected was eight; of these, four were so completely moribund that they exhibited no temporary amendment; of the remaining four (all extremely collapsed), one died and three recovered.

In any estimate of the value of venous injections, I should feel justified in omitting the first four cases, because it is well known that, unless a sufferer is already partly dead,—unless perhaps coagulation of blood has already commenced at the heart,—venous saline injection never fails to produce at least well-marked temporary amendment.

The three patients who recovered were males, aged respectively 22, 33, and 50 years. The first of these was injected four times to the extent of 250 ounces, or more than twelve imperial pints of fluid, each pint containing two drachms

of alcohol in addition to the salines.* This patient, therefore, received into his veins in the course of a few hours a quantity of alcohol equal to that contained in six ounces of brandy.

The second survivor was similarly injected twice to the extent of 45 ounces; and the third, once only to the extent of 40 ounces. I have recently seen the first and second of these patients; they have remained perfectly well, and have suffered no evil effects either from the cholera or from the operation.

The only instance of venous infusion effected by another practitioner in 1849, that has come to my knowledge, was that successfully operated by Mr. Howlett.

These are the "few facts" I have desired on this occasion to add to the mass of evidence already recorded of venous injections in general. I have refrained from dwelling upon my own experience; but I have ventured to intrude upon your patience by placing before you a chain of historical facts, extending over a period of two hundred years, illustrating at least the safety with which remedial agents may be introduced directly into the circulating system.

Time will not permit me this evening to state the conclusions at which an unprejudiced view of this interesting subject may enable us to arrive: I will, therefore, with your permission, postpone, until one of the ordinary meetings of the Society, the consideration of the diseased conditions in which, in my opinion, venous injections are justifiable, the materials which may be injected, and the mode in which the operation may best be performed. I shall be satisfied if, on the present occasion, I shall have succeeded in demonstrating the safety of the plan, and in awakening in your minds a more than transitory interest in the subject; such an interest as may lead to further research, and to the establishment of rules for its application.

The Hunterian Society, one of the oldest established medi-

* The saline solution employed consisted of—

Hydrochlorate of soda	℥iij
Carbonate of soda	℈ij
Water	℔xxx
Temperature	F. 110°

cal Societies of this metropolis, boasting a long line of members distinguished by their social and professional position, has ever been characterised by the pursuit of inquiries into the nature of diseases and their treatment, in the spirit of the immortal Hunter.

" I thank you much for the attention with which you have listened to this discourse. I confidently expect that, when the science and experience of our Society shall be applied to the elucidation of the best mode of using venous injections, a further step in the progress of our art will be accomplished,—a step calculated to increase our means of alleviating the sufferings of humanity, at least in some of those appalling diseases—cholera, tetanus, hydrophobia, and others,—in the treatment of which our experience has been hitherto the most often baffled; the diseases, therefore, in which additional means of relief are the most desiderated.

THE END.

LONDON:
PRINTED BY BOBSON, LEVEY, AND FRANKLYN,
Great New Street, Fetter Lane.

A CASE

OF

VERY EXTENSIVE GELATINIFORM

CANCER OF THE PERITONEUM,

INVOLVING THE LYMPHATIC GLANDS OF THE ABDOMEN AND THE OVARIES, AND SIMULATING ASCITES.

By EDWARD BALLARD, M.D.,

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Received Feb. 19th—Read March 14th, 1848.

Jane Last, aged 55 years, residing in the neighbourhood of Burton Crescent, placed herself under my care at the St. Pancras Dispensary, on January 17th, 1848. The history of her illness taken at that time is as follows :—

She is a native of Boston, but has resided in London during the greater part of her life. She was married at 29 or 30 years of age, and her husband is still living. In two years time she aborted at the third month, and never again became pregnant. The catamenia appeared rather early, namely, between 11 and 12 years of age, and she used frequently to suffer from dysmenorrhœa. She has never undergone any privation, and has always been extremely temperate and regular in her habits. No particular hereditary tendency can be traced.

Previously to her miscarriage she had suffered from no abdominal symptoms, but three or four years before had undergone an attack of typhus. At the time of the miscarriage, however, there was such an amount of pain and tenderness

at the epigastrium as to induce the medical man who attended her to apply leeches and a blister. From that time forward she has been in the habit of suffering from flatulent distention of the abdomen after meals, especially if she partook of any kind of pastry, raw vegetables, or soup.

At 45 years of age, that is to say, ten years ago, the catamenial periods became irregular, the interval varying from two to six weeks, the discharge on each occasion being excessive, and lasting for one or two weeks, accompanied as usual by severe abdominal and lumbar pains. This menorrhagia continued up to the final appearance of the catamenia between the ages of 51 and 52 years. She never suffered in the intervals from any leucorrhœal discharge.

At the time of the menorrhagia (the exact period I have been unable to determine), she consulted a practitioner in her neighbourhood, who cupped her over the sacrum, and applied leeches to the cervix uteri, and informed her that there was a tumour connected with the liver, which could be readily felt beneath the margin of the ribs on the right side. Attention was drawn to this in consequence of the uneasiness and tenderness at the epigastrium having so much increased that she was unable to bear the ordinary pressure of her clothes; and she occasionally experienced the "sensation as of a knot," referable to that region, which was commonly relieved by a dose of aperient medicine, and the evacuation of mucous stools mixed with long membranous shreds.

On the cessation of the catamenia, her health became so much improved that both her friends and herself anticipated in a short time its complete restoration. This improvement was only interrupted, about two years ago, by what she was told was "inflammation of the veins" of both legs: no clear account of this attack could be obtained.

In June 1846, she accidentally slipped from some steps. A few days after she began to complain of a sense of bearing down, and soon discovered a prolapsus of the womb. On account of this a ring pessary was introduced, but as there was much trouble in keeping it in its place, and it gave her

great inconvenience, after a three months' trial its use was finally abandoned, and the uterus has not prolapsed since.

Just at this time she perceived that she was passing a smaller quantity of urine than was natural to her, and that her abdomen was beginning to enlarge. The enlargement was first noticed at the lower part, and was accompanied by such a sense of weight there that she was in the habit of supporting it with her hands while walking. She also noticed a tumour above the pubes, "about the size of a turkey's egg," which, with that beneath the margin of the ribs, subsequently disappeared as the abdominal enlargement increased.

At Michaelmas in the same year, while walking in the city, she inadvertently struck her abdomen against a post with considerable violence, and ever since that time the epigastric uneasiness and sense of a knot there have been much more troublesome; but there has been throughout no lancinating pain.

The abdomen continued to increase in size, and in October last she began to vomit her food. At the beginning of November she entered King's College Hospital. The bowels up to this had been regular, but now became constipated, a change which she ascribed to the administration of opiates for the relief of her pain. Vomiting soon became of nearly daily occurrence, accompanied by violent epigastric pain, the matter thrown up being greenish and sour, but never fæcal. Solid food or broth induced violent cramp-like pain at the epigastrium, and sense of distention, remaining till she was relieved by vomiting after her tea. While in the hospital she began to suffer from œdema of the ankles, which subsequently extended to the thighs and lower part of the abdomen. Three weeks before leaving it she had an attack of diarrhœa.

She left the hospital of her own accord on December 29th. Since then the vomiting has continued, but the bowels have been more regular. She has been complaining very much of chilliness, and is compelled to lie between the blankets for warmth. She has been losing flesh ever since the

prolapsus, and presents a considerable degree of emaciation, where it is not disguised by œdema. The countenance is expressive of great anxiety; the complexion is not more sallow than is natural to her, and the cheeks are marked by fine purple veining.

There has been almost complete orthopnœa for five months past. She states that a sense of dragging and of cramp-like pain at the epigastrium is produced when she lies on the left side, and to a less extent when on the right. The pulse is small and ninety-eight, while the respirations are thirty, in the minute. There is great tenderness at the epigastrium and below the margins of the ribs, especially on the right side, and the tenderness extends as high as the dulness of the liver. There is tenderness also above the pubes, and deep in the left iliac region. There is much thirst, and the tongue is clean and smooth down the centre.

On examining the abdomen, its enlargement was found to be general and rounded, the epigastrium very full, and the umbilicus remarkable as not being prominent but stretched and flattened out. There was no enlargement of the superficial veins, but the cuticle was exfoliating in large pieces. The abdominal wall was tense, except at the epigastrium, where it yielded readily to the hand. By sliding the hand over the surface, there could be felt in the abdominal wall, about its upper part, seven or eight small tumours, about the size of a nut, all unconnected with any solid organ beneath, except one, which was situated near the cartilages of the ribs on the right side of the epigastrium and not far from the ensiform cartilage: this one was felt to be continuous with a solid tumour, about the size of an egg, which passed upwards under the cartilages of the ribs, and apparently belonged to the liver.

On the most careful examination no other tumour could be felt, except one just above the pubes, and this with some difficulty. It rose about three inches above the pubes, extended equally on either side to the total width of about four inches and a half, was immovable, and lobulated.

Pressure over any part of the abdomen produced great increase of the dyspnœa. There was a little resonance on percussion at the epigastrium and along the margin of the ribs on the right side, but in every other situation the dulness was complete, and there was the most perfect fluctuation in every part. The dulness of the liver was perfect as high as the fifth rib. The abdomen measured forty inches and a half in circumference, two inches above the navel.

In front the pulmonary resonance on percussion was good, and the respiratory murmur natural, but accompanied by some sibilant and mucous rhonchi. The heart's dulness was of natural extent, but the apex was beating beneath the third rib. Posteriorly the dulness of the liver rose as high as the lower angle of the scapula, and the resonance on the left side extended only about two fingers' breadth lower. There were no physical signs of pleural or pulmonary disease.

On digital examination the vagina was found to be altered in direction, passing upwards and forwards behind the pubic symphysis, and was broad and capacious. The cervix uteri was not to be detected, but the os uteri terminated the vagina opposite the upper extremity of the pubic symphysis, and was of natural size. By means of Simpson's sound the cavity was noticed to pass forwards in the direction of the tumour, felt in the middle line above the pubes, and the organ was immovable. In addition to this, there could be felt through the posterior wall of the vagina an irregularly nodulated elastic tumour, engaging the upper part of the pelvic cavity; it did not give to the finger any impression which led me to believe that it contained fluid.

It will occupy too much space to enumerate the grounds on which my diagnosis was based, but the conclusions I arrived at were as follows:—1st. That there was ascites. 2ndly. That cancerous disease existed most probably about the inferior part of the liver and transverse fissure, and probably also about the larger divisions of the postal vein. 3rdly. That there were cancerous nodules in the abdominal wall. 4thly. That there was fibrous tumour of the uterus.

5thly. That there probably was encysted ovarian disease. The more important parts of this diagnosis were communicated to my colleagues, Dr. Stewart and Mr. Walton.

I ordered her no medicine, but directed that she should take nothing but a table-spoonful of strong beef-tea every ten minutes, and that an enema of warm water should be used every morning. As I anticipated, this had the effect of removing the vomiting, and very much lessening the pain, while the bowels were acted upon regularly.

As the poor woman expressed a desire to be tapped, and there was a prospect of temporary relief to the dyspnœa being afforded by the operation, I requested Dr. Stewart to visit her with me, and as he saw no reason for deferring it, it was performed on the 24th, by Mr. Walton. Both these gentlemen felt equally convinced with myself, that there was fluid in the peritoneum. The trocar was introduced just below the navel, in order to avoid any tumour which might extend higher from the pelvis than we had physical evidence of. Nothing, however, passed through it, but about a tea-spoonful of clear gelatinous matter, which at the time struck me as so similar to that of gelatiniform cancer, that I remarked it to Mr. Walton. Still the idea that the peritoneal sac could be full of this matter was at once exploded; and under the belief that possibly the trocar might have penetrated a cyst of some kind, we agreed to another puncture being made, a couple of inches higher, in the hope of finding fluid; but here, again, the result was similar. A little opaque white matter accompanied the jelly from the second puncture. The patient was accordingly put to bed, and in the evening, when I visited her, though plainly not fully recovered from the shock of the operation, no unfavourable symptoms had arisen.

On the 25th, I found her complaining more of epigastric pain; there was no tenderness about the punctures, but much thirst; she had not vomited, and the bowels had acted spontaneously; the pulse and respirations were as before the tapping. After passing a very restless night, however, during

which she suffered severely from epigastric pain, she died on the morning of the 26th.

The body was examined twenty-six hours after death.

Head.—The dura mater was universally adherent to the calvarium; the brain, slightly congested.

Chest.—The lungs were small, and did not reach, in front, below the level of the fourth rib. Here and there, near their apices, a little cheesy tubercle and some gritty matter were discovered. The left lung presented a little congestion posteriorly. The heart was healthy, but its apex was situated opposite the third intercostal space. There was more or less atheromatous deposit throughout the whole length of the aorta.

Abdomen.—On opening the abdomen, the peritoneal cavity was discovered filled with gelatinous matter, and in the course of the examination no less than six quarts were, with more or less difficulty, removed: the difficulty arose from its remarkable tenacity, which was greater in the deeper parts than near the surface. It emitted a very disagreeable, faint, sickly odour, resembling that commonly perceived in the contents of the stomach after death. It possessed a ruby tint superficially, but in the deeper portions, and pelvis, it was less clear, and was streaky, some portions being pearly, and others having the colour of crude rock salt. In the epigastric region, there was some reddish-brown opaque liquid, containing some shreds of lymph. On turning aside the abdominal wall, the peritoneal lining was seen to be infiltrated with colloid, in the form of nodules and little masses, intersected with little fibro-cellular bands forming loculi. This gelatinous matter was not separated by any membrane from that in the cavity. There were a few spots of ecchymosis, also, about the lower part of the wall. In its upper part, the nodules felt during life were found to consist of small, hard, flattened tumours, about a quarter of an inch thick, and measuring three-quarters of an inch in diameter, placed beneath the peritoneal lining. There were shreds of lymph attached to the wall and organs in the epigastrium and hypochondria.

When the loose jelly was sufficiently removed, the following appearances were presented:—

1st. The epigastric tumour was found to depend upon a colloid infiltration of the peritoneal investment of the left lobe of the liver. 2nd. The suspensory ligament was infiltrated with it. 3rd. The diaphragm was adherent to the right lobe of the liver, over a great part of its upper surface, and to the spleen; and where unattached, its peritoneal lining was infiltrated in the same manner as the abdominal wall, and contained also small tumours of the same growth. 4th. On the right side of the abdomen lay a large tumour, consisting of colloid, and nodulated on the surface with gelatinous matter. It was broad above, where it was separated by a moderately deep furrow from the liver, but narrow below, where it adhered to a tumour in the hypogastrium. Its entire right border was free, as was also the lower half of the left. From its left side, above, a prolongation extended outwards and backwards, to become continuous with some colloid matter on the surface of the spleen, and was connected by peritoneum with the great curvature of the stomach. The upper part of the tumour presented tuberous elevations on it, and consisted of the mesocolic glands, in a state of colloid degeneration. The remainder, with its prolongation, was formed by the omentum in a similar condition. 5th. In the hypogastric region, a solid tumour was situated, adherent on its anterior surface, and on the right side to the abdominal wall, and there was colloid matter in the adhesions. This tumour was largely lobulated, and was connected to the colloid omentum by means of one of its large globular elevations. Towards its left side and upper part, it was connected with a fibrous sac, presently to be noticed. The dimensions of the tumours were those observed during life. 6th. On the left side of the abdomen, extending upwards nearly to the margin of the ribs, and downwards to the iliac fossa, were situated the remains of a large sac, whose cavity extended, to the right, behind the great lateral tumour of the omentum, and communicated with the general cavity of the peritoneum in the

right flank. This sac, when perfect, could not have contained less than three pints of matter. Its contents were a jelly, less transparent and more opaline than what was lying free in the peritoneum, and very firm. Its colour was like that in the deeper parts of the cavity of the peritoneum. The sac itself, as it appeared anteriorly, consisted of a very dense fibrous tissue, and presented sacculi. It was more or less vascular in all parts, but the part attached to the hypogastric tumour was very much so, the vessels being abundant, running in a vertical direction, and giving a red colour to the surface. There were openings in the sac, by which it communicated with the peritoneal cavity, and the gelatinous contents communicated also with that of the peritoneum, the transition in firmness and colour from one to the other being gradual. One remarkable opening was situated on the anterior part of the sac; it was about three inches in diameter, and rather rounded in form. The sac was also broken down towards the left side, and again at the right side of the colloid omentum. It was connected with the uterine tumour, and with the left edge of the omental mass. 7th. A coil of small intestine was situated in front of that part of the omental mass which proceeded towards the left side, to be connected with the spleen:—the convolutions were adherent by lymph not very recently effused.

The liver was of the ordinary size; its capsule, opaque and thickened; its peritoneal coat, infiltrated with colloid, in most parts of the lower half of the organ. In the lower border of the left lobe, this had occurred to some extent, and constituted the tumour felt during life in the epigastrium. The parenchyma of the organ was healthy. The glands about the transverse fissure were enlarged, and converted into masses of colloid.

The spleen was enveloped in a capsule of colloid, with the exception of the upper and inner portions. The stomach was empty, flattened and contracted, especially towards its pyloric extremity. The mucous membrane was healthy. A small deposit of colloid was situated on the peritoneal surface

posteriorly. There were some portions of colloid connecting the tumour of the mesocolon with that on the surface of the liver, crossing the pyloric extremity of the stomach. The greater part of the small intestines was situated in a small space behind the portion of the tumour which represented the transverse mesocolon, and in front of the right kidney. They were here rather contracted in diameter, but presented no indication of peritoneal inflammation. The mucous membrane was natural. Tubercular elevations of colloid, varying in size from that of a hemp-seed to that of a half-walnut, were scattered over the peritoneal surface. The mesenteric glands were similarly infiltrated with colloid, but were not affected to the same extent as the mesocolic glands. There were nodular deposits of colloid also on the peritoneal surface of the cæcum and colon, and the appendices epiploicæ and the mesocolic glands were deeply implicated; the latter, very much enlarged, and converted entirely into colloid matter. The transverse colon was traced passing in a tortuous manner through the great lateral tumour, marking off the portion above as belonging to the mesocolic glands, and that below as belonging to the omentum. Its diameter was contracted, but the mucous membrane, so far as it could be examined, was healthy. Near the extremity of the cæcum, one colloid mass passed through the muscular coat, and its structure could be seen through the thin layer of mucous membrane which covered it internally. This was the only part of the whole canal where so deep a penetration of the disease had taken place. There were masses of colloid also attached by peritoneum to the sides of the rectum. The pancreas was completely free from disease.

The fundus of the uterus presented some rounded globular elevations before noticed, of the ordinary fibrous character. The ovaries could not be discovered; but in place of them, at the sides of the uterus, and connected by the lateral ligaments to the wall of the pelvis, were fibrous sacs, presenting, externally, a more or less lobulated appearance. This external lobulation corresponded to an internal division into sac-

culi or cells, of rounded form, and each distinct in its cavity from the adjoining. The external ones were large, and embraced several of the smaller. The large sac or cyst, seen on first opening the abdomen, appeared to belong to the ovary of the left side. All these cysts contained a gelatinous, firm matter, which was adherent to their wall. No part of the cysts on either side extended into the pelvis far beyond its brim; but on both sides they pressed upon the iliac vessels and nerves lying on the psoas muscle, especially on the right side. On the right side a small portion of the cyst passed through the upper part of the great sacro-sciatic foramen. The bladder was small and compressed between the uterus and pubic symphysis, but otherwise healthy. The kidneys were healthy, but pale.

A microscopic examination of some of the locular masses, the gelatinous matter of the abdomen and that of the ovarian cysts, was made by Dr. Garrod and myself, and also by Mr. Marshall. The characteristic cells of cancer, simple, compound and caudate, were discovered in all; and, on adding acetic acid, they were found to be held together by a very delicate membrane, which tincture of iodine demonstrated to be very finely granular in its texture.

Remarks.—Although I have searched diligently through the principal periodicals and systematic works, and have moreover looked through several museums, I have failed to meet with a single case presenting the extraordinary characters of that which has just been read. The extent of the disease is one of its most remarkable features, the microscope having proved the cancerous nature of all the gelatinous product found within the abdomen.

It is not unusual for gelatiniform cancer to attack the *omentum*; and sometimes it has, as in this instance, acquired a considerable bulk; but it has mostly occurred

secondarily to cancer of the stomach; while it is remarkable here as lying so completely upon the right side of the abdomen.

The occurrence of colloid of the *liver* is not admitted by Dr. Walshe, Dr. Budd, or M. Cruveilhier, and I only know of a single specimen of it, which was presented by Mr. Kiernan to the Museum of University College. It there forms large masses in the parenchyma of the organ. In the case now before the Society, the peritoneal investment alone is implicated.

I have been able to discover no example of colloid affecting any part of the *spleen*.

The *lymphatic glands* have suffered much more from the disease than those parts of the alimentary canal with which they are connected—throughout which there is no part where the entire wall has been infiltrated; the colloid being thinly scattered over it, and only on the peritoneal surface.

The *peritoneum and its lymphatic glands* constitute the chief seat of the affection. I am unacquainted with any case in which the lining of the *abdominal wall* was so extensively infiltrated as in this. A few years ago, a case of extensive gelatiniform cancer, affecting most of the organs of the body, was, I believe, communicated to the Society by Dr. M. Hall; but I have been unable to discover how far it agreed with mine, inasmuch as only an outline of it is to be met with in the periodicals of that date. I may refer to the *ecchymoses*, in the midst of the colloid infiltrating the lower part of the wall, as something unusual in this variety of cancer. The occupation of almost the entire *peritoneal sac* by colloid matter is, so far as I have been able to discover, without a parallel in the records of medical experience.

Dr. Walshe remarks that he has never himself "observed colloid in the *ovaries*, nor met with any faithful description proving that other observers have been more successful."

Cruveilhier, on the other hand, admits its occurrence; and although, as all must allow, on very insufficient grounds, yet I have reason to believe that his opponents are called upon to modify, in some degree, their complete condemnation of his views.

Dr. Walshe remarks, that "one of the distinguishing attributes of the colloid species of cancer is its apparent insusceptibility of assuming such variation of internal arrangement as to call, in this point of view, for the establishment of varieties."

In the present instance, however, three forms are clearly discernible:—one, is the ordinary *locular form*; another, is the *encysted form*; and the third, is a *free or unincluded form*. Regarding the first, nothing further is required to be said; but the other two call for a few observations. The term "encysted gelatiniform cancer" originated with M. Cruveilhier; who, taking into consideration the gelatinous appearance of the matter sometimes contained within ovarian cysts, and its enclosure within cavities having fibrous or fibro-cellular walls, instances of which abound in the cases recorded by medical writers, rather hastily sums up his conclusions in these words:—"In short, it is with gelatiniform cancers that we must class the ovarian cyst, which only differs from gelatiniform cancer of the stomach and other tissues by the size of its meshes."

Few pathologists have been found to coincide in this opinion; but, nevertheless, it appears probable that the other extreme, which denies a malignant character to any ovarian cystoid disease, is likely to be no less erroneous. At all events, in the case before us, microscopic examination has demonstrated in the gelatinous matter of the cysts the same minute structure as was found in that from the loculi, so that if we admit the malignancy of the latter, we cannot deny it in the former. But this case does not stand alone, since I would direct attention to one brought before the Medical Society of London, a few years ago, by Dr. Frederick Bird. In that instance an ovary had become converted into a large cyst, containing a firm gelatinous matter, in which Dr. Golding Bird and others, by microscopical examination, discovered cells agreeing in appearance with those found in

other forms of malignant disease. To say the least of it, these two cases deserve to be taken into consideration, in connection with the frequent conjunction of ovarian cystoid affections with cancerous disease of the pelvic or abdominal organs, before we decide finally against the completely malignant character of the former. I am aware that another explanation of the existence of cancer-cells in the gelatinous matter of the cysts in the present case may be afforded, namely, that the contents of the cyst might at first have been non-malignant, and that they might have acquired the cancerous character subsequently to the bursting of the cyst, by contamination from the neighbouring diseased tissues. Nor would the view of its original malignancy be in any way strengthened by the discovery of cancer-cells in the smaller and more recent cysts, although their absence in the latter, had it occurred, would have weighed strongly against it. Still the admission of this explanation does not destroy the fact of malignancy, which is all I argue for; much less does it affect in any way the inference to be drawn from Dr. F. Bird's case.

I have already stated that I have failed in my search after another example of colloid matter lying free within the peritoneal sac. How it got there is a question of interest. Three modes may be imagined—1st. By escape from the ovarian cyst on the occasion of its rupture. 2nd. By growth from the diseased part of the abdominal wall. 3rd. By the occurrence of fibrinous dropsy and its subsequent conversion into colloid matter. An objection fatal to the first of these views is, that at the time the rupture of the cyst is believed to have occurred, the abdomen was but little enlarged; and other evidence is in favour of its accumulation at a subsequent period. Besides, the fibrous cyst was inelastic, and at no time could have contained the vast quantity of matter found in the peritoneal sac. In deciding between the two remaining modes, the question resolves itself into this: viz., whether the plasma from which the cancer arose was effused merely from the diseased surface, or from the entire peritoneal lining,

healthy and diseased. Vogel admits "that the fluid of fibrinous dropsy is capable of organization," and that "it is indifferent whether the fibrin be in a fluid or coagulated state;" that from it "the most different forms of tissue, either normal or pathological, may be evolved:" and he enumerates cancer among the latter. Dr. Walshe, too, says, "that the blastema of cancer, there is every reason to suppose, is, like the ordinary exudation-matter, essentially composed of liquor sanguinis, but of liquor sanguinis modified in its vital properties;" and moreover agrees with Dr. Carswell, that in rare cases the free surface of serous membranes may secrete cancerous matter. It must be borne in mind, moreover, that there was present extensive disease about the large divisions of the portal vein and in the omenta, which would dispose to effusion into the peritoneal cavity.

Still I do not feel inclined to regard this as its origin, for the following reason; namely, that the parts furthest from the diseased portions of the abdominal wall were much firmer in consistence than those near to them, and were deficient in the faint and sickly odour so strongly marked in the latter. The pelvis, too, contained a matter nearly as firm as that in the largest cyst, and the surface of this portion was in no degree softer than the inner part of it.

One word more with respect to two of the signs observed during life. The very perfect character of the fluctuation was such as to lead the minds of those who saw the patient, to the opinion that the matter in the peritoneal sac was sufficiently fluid to pass through an ordinary trocar. I do not know in what way the fluctuation of gelatinous matter can be distinguished from that of ascitic fluid. Cruveilhier relates a case where an ovarian cyst containing gelatinous matter was tapped ineffectually, and acknowledges his inability to avoid a similar accident in future. In Dr. F. Bird's case, fourteen medical men were convinced, from the perfection of the fluctuation, that the cyst was distended with fluid. I was well aware too that even a solid organ will impart sometimes a sense of fluctuation to the hand, recollecting a

case a few years ago under the care of Dr. Williams, in University College Hospital, where it was very evident over a surface corresponding to an exceedingly enlarged spleen. Perhaps in any future case, should a similar one at any time arrive, the diagnosis might be assisted by the condition of the umbilicus;—the opening out, stretching and flattening of its fold, without prominence, being unusual, where ascitic fluid is of the ordinary character, and the abdominal wall free from disease.

[*From Transactions of the Medico-Chirurgical Society*, Vol. xxxi.]

RICHARD KINDER, PRINTER, GREEN ARBOUR COURT, OLD BAILEY.

PAMPHLETS

69

69