Parkes Pamphlet Collection: Volume 19

Publication/Creation

1849-1860

Persistent URL

https://wellcomecollection.org/works/evk64ypj

License and attribution

You have permission to make copies of this work under a Creative Commons, Attribution, Non-commercial license.

Non-commercial use includes private study, academic research, teaching, and other activities that are not primarily intended for, or directed towards, commercial advantage or private monetary compensation. See the Legal Code for further information.

Image source should be attributed as specified in the full catalogue record. If no source is given the image should be attributed to Wellcome Collection.

wellcome collection

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

ROYAL ARMY MEDICAL COLLEGE LIBRARY S.W.1.

Black —	Broncho-pulm.y mem.b
Jenner —	Congestion of Heart
Parkes	Discharges in Chole
Parkes.	Early cases of Chole in London.
Scott.	Mercury on bile
Schunk.	Indigo blue in Urin
Reynolds.	Vertigo
L. Parker	Affections of bone.
Davy	Urea
Harvey.	Fœtus in Utero
Reynolds	Laws of life.
H. Dobell.	Influence of White Ligh
Humphry	Coagulation of the blood
Harris —	Amyloid Degenera
Symonds —	Habit.
Paget.	Rythmic motion of h
Dal'ois Reymond.	Letter to Benco Jone
Bond.	Rheumatism
Jenner	Emphysema
Langer.	Ague
Ringer.	Diabetes Mellitus
Lister	Arteritis
R. Hall.	Bronchial tu's.
R. Hall	Spirometer

The pamphletts in this volume are of various dates from 1849 to 1880. But the majority are dated towards the latter period.

get —	Chronometry of Life
urchison.	Stomach fistulæ.
ister —	Visceral Nerves.
durchison.	Gastro-colic fistula
ouch.	Ventilation
ughton.	Constants of Urine
ley —	Supra renal capsules
ain.	Rectal tumour
der Lindsay.	Transmission of dis. to lower animals
"	Hard waters on lead

Bequeathed by Dr. E. A. Parkes.

ROYAL ARMY MEDICAL COLLEGE LIBRARY.

With the Author's compliments

36

Bequeathed
by Dr. E. A. Parkes.

PREFACE.

In offering the second part of this work to the Medical Profession, the author has to remark that, inasmuch as it treats only of the pathology of pulmonary tuberculosis, it is necessarily incomplete. To render it otherwise, an account of the melanotic and malignant diseases of the bronchio-pulmonary membrane would be necessary. These, however, are *comparatively* of such infrequent occurrence, that the author has purposely omitted them until his own field of investigation shall have supplied him with sufficient data respecting such diseases. It is not by a *few*, but by *numerous* carefully conducted investigations, that the pathological truths of a disease become established axioms in medical science; and it were much better not to publish at all than to seek to establish certain views which are supported by insignificant data only, and which have not the guarantee of a sufficient number of personal investigations and experiments of both a *positive* and *negative* value. This view has, from the first, determined the author not to treat of any disease of the bronchio-pulmonary membrane, which has not afforded him the amplest opportunities for its full investigation. Whatever, therefore, he has published on this subject is to be regarded as the result of long and laborious research; but, in saying this, he has no desire either to court favourable criticism, or to hurl defiance in the face of those who may honour his present publication with a notice. He, on the contrary, leaves the work either to stand or to fall by the confirmation or refutation of the pathological views which it sets forth.

Chesterfield, Dec. 1854.

ROYAL ARMY MEDICAL COLLEGE LIBRARY.

WE10.
13.25.e

DESCRIPTION OF PLATE.

FIG. 21.—1. Fibrous tissue of the pulmonary cells.
2. Cells showing slight thickening of their basement structure.
3. Cell showing a further degree of basement thickening.
4. Pulmonary vesicles filled with tuberculous exudation, which has germinated and formed cells.

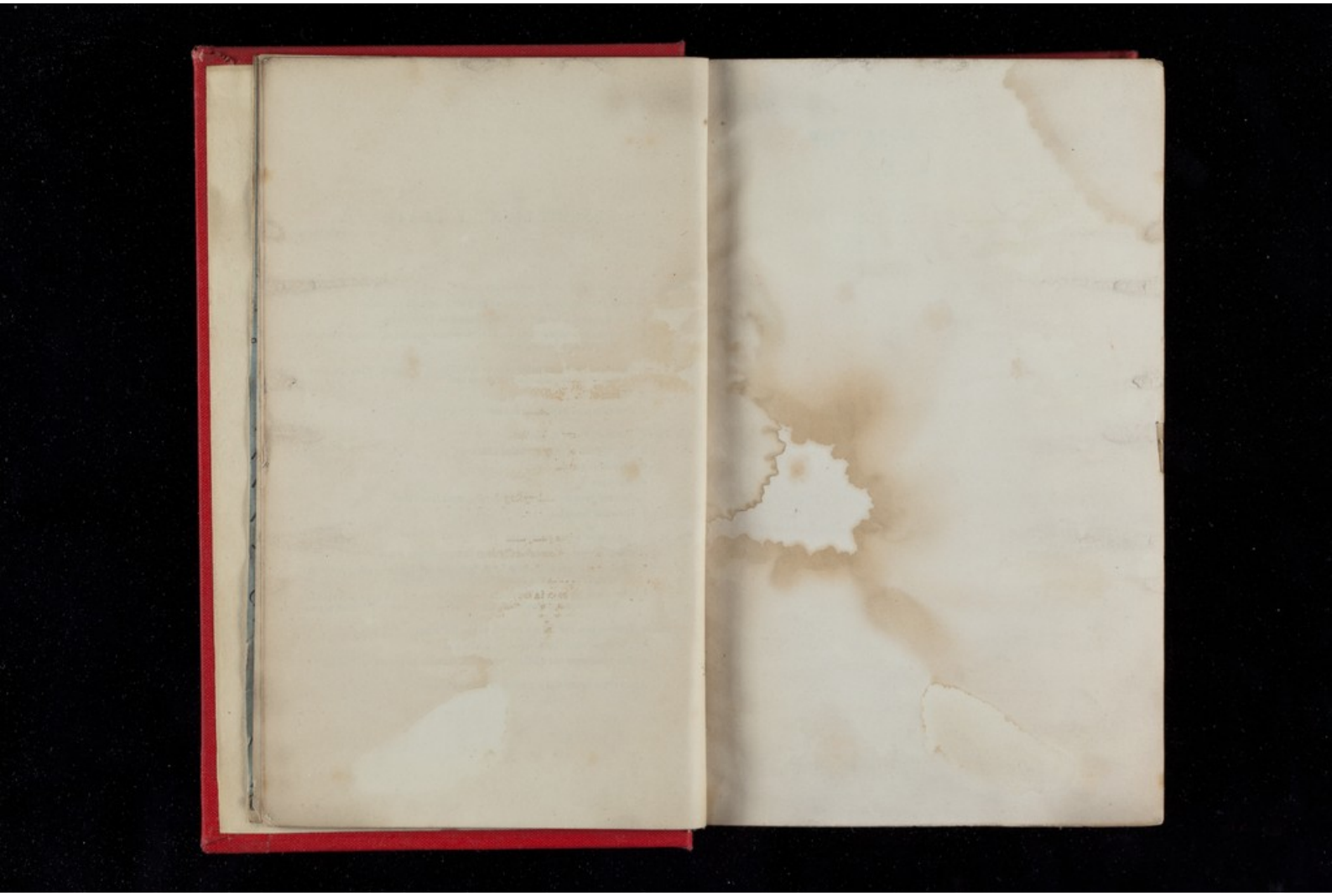
FIG. 22.—1. Granular epithelial patch seen in the sputum of commencing germination of acute pulmonary tuberculosis.
2. Mucus-cells.
3. Granular and molecular matter.

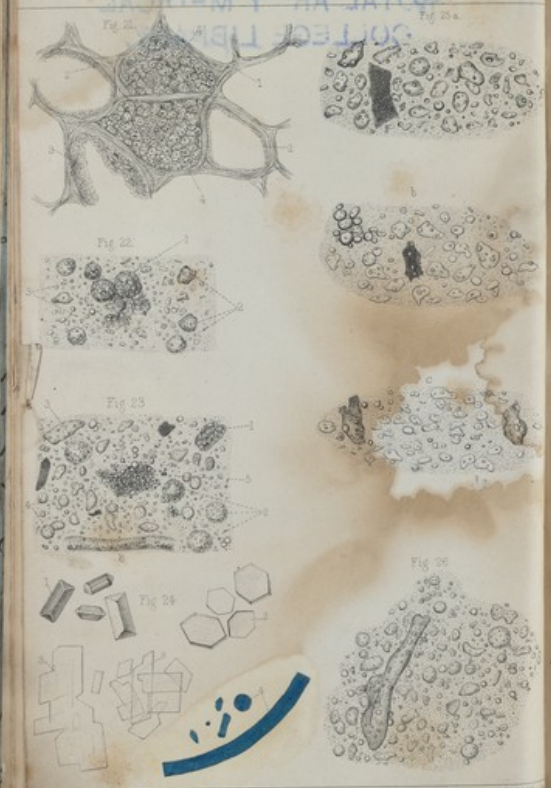
FIG. 23.—1. Patch of young epithelium.
2. Mucus-cells of very granular character.
3. Basement patch.
4. Pus-cells.
5. Exudation-mass undergoing development into cells.
6. Granular bronchial cast.

FIG. 24.—1. Crystals of triple phosphate.
2. Plates of cystine occasionally seen in tuberculous sputum.
3. Distinct and confused plates of cholesterine—the latter of more frequent occurrence than the former in tuberculous sputum.
4. Blue particles seen in the sputa of cases numbered 9 and 11 of Table I. of the sputum, manifesting the chemical reactions of Prussian blue.

FIG. 25.—*a*. Tuberculous pus from a cavern in the left lung.
b. The same after the addition of acetic acid.
c. The same after the addition of a weak solution of potash.

FIG. 26.—Appearance of the flaky portion of tuberculous pus.





ROYAL ARMY MEDICAL
COLLEGE LIBRARY.

PART II.

NON-INFLAMMATORY DISEASES

OF THE

BRONCHIO-PULMONARY MEMBRANE.

Tuberculosis of the Bronchio-Pulmonary Membrane.

The origin and subsequent course of tuberculosis of the bronchio-pulmonary membrane comprise three pathological conditions of that structure, which are recognised as—

1. The stage of local predisposition.
2. The stage of deposition.
3. The stage of germination.

Stage of Local Predisposition.

The evidences of the predisposition in the bronchio-pulmonary membrane to tuberculous deposit, are found in a deviation from the healthy standard of blood which the vessels of that structure contain. This deviation is, in many instances, apparent to the naked eye; but to the powers of the microscope it reveals itself in a more definite and intelligible manner. To the unaided sight the membrane, in well-marked cases, is apparently more puffy and of a deeper coloration than natural; whilst by the microscope its vessels are observed to have a greater average diameter than natural, owing to the overplus of blood with which they are surcharged. The local predisposition, which this overplus of blood constitutes, may be limited to even a pulmonary lobule, a lobe, or it may involve the greater portion of one or both lungs. The upper lobes, however, are so frequently affected in pulmonary tuberculosis, that these portions of the lungs have been thought to possess an elective affinity for the localization of tubercle; but such does not appear to be the

case. Their more frequent invasion by tubercle is more probably due to their deficient mobility, as compared with that of the middle and lower lobes, during the respiratory movements. The greater the mobility of a part, the more quickly is its circulation accomplished. If, then, different parts of an organ manifest differences in the degree of their mobility, the vessels of the less mobile will fail to transmit their blood in a ratio equivalent to that of the vessels of the more mobile parts, and thus a comparatively partial retardation of the flow of blood through the former vessels takes place. Now, it is evident that, when these different parts are supplied with blood by branches of the same vessel, the blood sets out with equal velocity to each part, and that if, in the extreme vessels, that velocity is more efficiently maintained by the mobility of one part than by that of another, a *comparative* retardation must necessarily take place in the vessels of the latter. Hence, this normal condition of the circulation in the upper lobes of the lungs is more favourable to the occurrence of tubercle than is the circulation through the lower lobes; and hence the probable reason why, under the exciting causes of the localization of tubercle, the deposit more frequently takes place in the former than in the latter portion of the lungs.

In the establishment of the local predisposition to tubercle in the bronchio-pulmonary membrane, it is important to know, whether this predisposition is indicated by any alteration or modification of function in the organs of which that structure forms a part. Now, it is evident that, as the bronchio-pulmonary capillaries of the affected part contain more blood, and, therefore, occupy more space than in parts not thus locally predisposed to tubercle; and as this increased occupation of space must necessarily be at the expense of a corresponding diminution of the diameter of the pulmonary cells and bronchial tubes, some modification of the function of the part, however slight, takes place. The appreciability, however, of this modification will depend on the degree and extent of the local predisposition, and on the comparative difference, in this respect, between the corresponding portions of the two lungs. If the condition of the bronchio-pulmonary capillaries is the same in corresponding portions of both lungs, the physical signs bear the relative ratio of health, notwithstanding a local predisposition may exist. If, however, one lung is, and the other is not, predisposed to tuberculosis, or if, in this respect, there is a difference between the two, the physical signs of the one not thus predisposed are more strongly marked or intensified than those of the other. In other words, the percussion-note of the lung predisposed to tuberculosis does not, under the same circumstances, attain that pitch which is elicited from the other; neither is the vesicular murmur so clear and audible, as in the lung not predisposed to tuberculosis. On the contrary, a greater distance seems to intervene between the ear of the observer and the site of the respective sounds; but such in reality is not the case. The apparently greater distance of the vesicular sound of the lung

predisposed to tuberculosis, is not owing to a diminution in the conducting power of the lung, but to the reduction, during each respiratory movement, in the quantity of inspired air, and to the partial want of that active resistance which is manifested by the fibrous tissue of the pulmonary cells at the very extreme of inspiration, when the genesis of the vesicular murmur is at its climax. Thus, then, the local predisposition to tuberculosis of the bronchio-pulmonary membrane does modify, to a certain extent, the normal function and the physical signs of the lungs. To this statement it will probably be urged, that many cases of predisposition to pulmonary tuberculosis exist, in which the most careful and accurate investigation fails to detect the slightest evidence of disease in any one of the above particulars. To such an objection I oppose the negation of the existence of the alleged predisposition. A constitutional predisposition does not necessarily impose a local predisposition. The former is a condition of the system in which every structure and every fluid are alike affected, as to the degree of vitality of the former, and the healthy constitution and elaboration of the latter; yet how many thousands inherit this predisposition and pass unscathed through a long life! A local predisposition may, on the contrary, be either engrafted on a constitutional predisposition, or it may be acquired without any such constitutional connection; but in either case it is the result of a cause operating directly or indirectly upon the part itself. A person born of phthisical parents, is not necessarily predisposed to pulmonary consumption, but to a tuberculous disease, which, according to the local predisposition engendered by causes apart from constitutional considerations, may result in glandular enlargement, pulmonary consumption, caries, abscess, or a squamous affection of the skin. Thus, in a person constitutionally predisposed to tuberculosis, the continued exposure of the neck to the influence of a cold air, will, by first unusually exciting and thereby subsequently impairing, to a certain extent, the healthy tonicity of the capillaries, allow the latter vessels to admit more blood than usual, by which condition a local predisposition is established, and actual disease results on the continuance of the cause. The same remark applies to the other diseases just named, all of which require the previous establishment of a local predisposition before actual disease can be said to exist. This view resolves the establishment of a local predisposition into the genesis of disease; and as the genesis of disease predicates an aberration or deflection from the standard of health, the local predisposition to tuberculosis in the bronchio-pulmonary membrane, must and does give rise to a corresponding modification of function, not only in the structure immediately concerned, but also in the organs of which that structure forms a part. Some of the evidences of this modification have already been detailed. They arise in immediate dependance on the local predisposition which has been established, and are amongst the first to arrest the attention of the physician. Other modifications,

equally significant, are present in the vital manifestations of the lungs, and are read in the influence of the respiration on the different parts of the system.

Thus, the reduction in the breathing-power of the lungs which necessarily attends the establishment of the local predisposition to tuberculosis, leads, as has been before shown, to a falling off in the bulk of the body until the required oxygenation of the latter is brought down to a relative ratio with the respiratory capacity. During this lost balance between the respiratory function and the required oxygenation of the system, the imperfect aëration of the blood leads to a defective stimulation and nutrition of every organ, the function of which undergoes a proportionate modification. Thus the deficient aëration of the blood begets the sensation of the want of more air, which sensation is, according to physiological laws, referred to the organs by which the aëration of the blood is accomplished. It is true that this sensation may be so slight as to be only noticed on exertion; but still it is present in proportion to its cause, and, when carefully analysed, is found, even in the quiescent state, to give evidence accordingly. The very slightest deflection from health in an organ is invariably accompanied by some degree of modification in the conditions necessary for the due performance of its function, but such modification of the conditions and the consequent departure from the standard of perfect action must influence other organs before the attention of the mind can be arrested by them. It follows from this explanation that, provided the local predisposition to tuberculosis in the bronchio-pulmonary membrane does not so far reduce the volume of air which enters the lungs as to impress somewhat powerfully, through the imperfect oxygenation of the blood, the brain proper, the sensation of the want of air is not excited in the mind; or if it is, such sensation is so feebly excited there that the mind can take cognizance of any other object of sensation to the exclusion of the former. But the medulla oblongata, by virtue of its government over the respiratory function, feels the slightest diminution in the volume of air admitted into the lungs, and it at once endeavours to establish a compensatory action, although the cause may not resolve itself into an object of sensation. Hence the mode of breathing may be altered, and the mind be scarcely conscious of it. This is often the relative state of the respiration and of the mind in the very gradual occurrence of the local predisposition before named. A long process of indigestion is generally the precursor of the gradual establishment of a local predisposition to tuberculosis in the bronchio-pulmonary membrane; and this condition it not only induces by the depraved blood-plasma to which it gives rise, and to the consequently defective nutrition and deficient tonicity of the pulmonary capillaries, but probably also by the impediment which is offered to the expansion of the lungs, owing to the frequent and continued distension of the stomach and to a congested liver, which prevent the proper descent of the diaphragm during inspiration.

As the local predisposition may, in this way, be gradually brought about, the absolute encroachment on the respiratory function observes an equally slow progress—the nutrition of the body declines in equal ratio—the sensibility of the brain is imperceptibly diminished, and thus the mind fails, for a time, to take cognizance, under ordinary circumstances, of the necessarily altered breathing consequent on the diminished vital capacity of the lungs and the deficient aëration of the blood. When, however, the local predisposition, which corresponds to the first pathological condition of bronchitis and pulmonary cellulitis, is suddenly induced, or when the slow establishment of this local predisposition is suddenly aggravated by any cause which immediately and at once increases to any great extent the congestion of the pulmonary capillaries, the brain is so powerfully impressed by the incident impression transmitted from the lungs, that the mind becomes conscious of a deficient aëration of the blood, and thus arises the desire for the admission of more air into those organs. Now, the inference to be drawn from the foregoing considerations is, that the causes of this local predisposition are various; whilst experience sufficiently attests that the local predisposition itself may exist in different degrees of extent and intensity. To the account of causes may be referred dyspepsia, which operates slowly, and cold, or the inhalation of air loaded with dust, minute particles of foreign bodies, or impregnated with irritant gases, which act more or less suddenly in the induction of pulmonary congestion. In the slow establishment of the local predisposition to pulmonary tuberculosis, the dyspepsia which precedes it, is to be regarded as the first tangible point only in the link of causation; inasmuch as it itself is but the effect of a cause, which is referrible either to the original constitution and vital endowments of the digestive apparatus, to a depressing action of the nervous system on the latter, to the quantity and quality of the food, or to a more or less combination of these several circumstances. These facts appear to me to have received too little attention at the hands of Professor Bennett, who, in his recent work on "Pulmonary Tuberculosis," seems to centre the sole cause of tubercle in functional aberration of the digestive organs. By doing so, to the exclusion of the efficient causes of such aberration, he seems to lose sight of that general predisposition to tuberculosis which is traceable in very many of such cases, and which is characterised by a primitive delicacy in all the tissues of the body, as the result of hereditary transmission. Does not this constitutional delicacy of the digestive organs render them more liable than they otherwise would be, to suffer from the exciting causes of dyspepsia? That such is the case it is impossible to deny; therefore, in treating of the pathology of tuberculosis, due weight must be attached to this constitutional predisposition, although its consideration may but slightly affect the treatment. Again, in all but exclusively referring tuberculosis to the derangement of the digestive function, Dr Bennett is more or less opposed to the experience of many observers, who have not unfrequently witnessed the

deposition of pulmonary tubercle without its having been preceded by any dyspeptic symptom whatever. I have not unfrequently observed in a constitution predisposed to tuberculosis, a sudden localization of tubercle in the lung follow undue exposure to cold, where there was not the slightest ground for supposing the previous existence of dyspepsia, and where the subsequent course and microscopic characters of the exudation left no doubt as to its real nature. Now, it is evident that, in exudation thus suddenly poured forth, we must look for the exciting cause of its localization in whatever induces undue congestion of the pulmonary vessels, which thus constitutes the local predisposition to tubercle. When the local predisposition is thus suddenly established, it arises in any portion of the lungs, and, if not immediately relieved, it is followed by an exudation which, in its progressive tendency, often runs a rapid course, thus constituting acute tuberculosis or "galloping consumption;" whereas a local predisposition, preceded by dyspepsia, is generally followed by a slow deposition, which selects, as it were, the upper lobes of the lungs, observes an equally slow progress, and is, therefore, properly designated by the term—chronic.

The general symptoms and the physical signs indicative of these two forms, which approach each other by cases of intermediate severity, vary in the relative degree of their intensity. In the one—the acute form—they approach the general symptoms and physical signs of the first pathological stage of simple inflammation of the lungs; in the other—the chronic form—they are at first referrible to derangement of the general health, accompanied by little or no febrile movement, to which the pulmonary signs and symptoms gradually succeed.

Stage of Deposition.

After the continuance for a time, in different degrees of severity, of the local predisposition before detailed, exudation takes place from the affected vessels in quantity proportionate to the extent and degree of their previous distension. Hence the amount of exudation may vary from a mere overplus of the natural demand to a quantity equal to that of simple inflammation of the lungs.

The former is the more frequent occurrence of the two; but the latter arises in tolerable frequency, and is more particularly referrible to the periods of adolescence and early manhood than to the later epochs of life.

The exudation in pulmonary tuberculosis, like the exudation in simple inflammation of the lungs, leaves the vessels in a fluid form, infiltrates the basement structure of the pulmonary mucous membrane, and thus tends towards the interior of the cells and minute bronchi. When the quantity of exudation is small, as in chronic pulmonary tuberculosis, it leads to a gradual thickening and hypertrophy of the basement structure, and consequently to a gradual

encroachment on the diameter of the pulmonary cells; but when, on the other hand, the exudation is copious, as in the acute form of the disease, it passes into the interior of the pulmonary cells, which it may obliterate, and in this way a certain degree of consolidation of the pulmonary tissue is produced. In escaping into the pulmonary cells, however, it does not pass by imbibition through the layer of epithelium; but it, on the contrary, raises this from its connection with the basement structure in a manner analogous to the elevation of the cuticle by a blister.

The above declaration, as to the seat of tubercle, may be verified by microscopic examination of fine sections of lung affected with tuberculous deposit. These generally show that some of the cells are completely obliterated; others partly so; whilst we find, as the examination recedes from the seat of the greatest amount of exudation, the vesicular structure approaches more and more its normal condition. When the deposit is confined to a single lobule, as is sometimes the case, all the cells of that lobule may be entirely obliterated, and in that case the transition from perfect occlusion of the cells to their normal condition, on the confines of the deposit, is abrupt.

Microscopic observation further shows that the exudation seldom takes place, to any great extent, amongst the fibres of which the cell-walls are in part composed.

A reference to Fig. 21, Plate I., drawn from several morbid specimens of lung variously affected with tuberculous deposit, will elucidate the above statements, with respect to the seat and extent of tubercle.

Now, the physical signs and general symptoms attendant on this stage, are commensurate with the extent and rapidity of the tuberculous deposit. When the basement membrane throughout the whole or the greater portion of one lobe of the lungs, has undergone even a very slight degree of thickening, the physical signs, indicative of such a condition, become appreciable; but when this amount of thickening is limited to one or to a few lobules, its existence is scarcely to be recognised by the most careful and delicate auscultation. Percussion also fails to indicate it; neither does the state of the respiration excite suspicion. When, however, the degree and extent of the basement thickening render the disease detectable by auscultation, it is found that the relative duration of the inspiratory and expiratory murmurs is altered, and that their *quality* is likewise modified. Thus the duration of the expiratory to the inspiratory murmur is lengthened, the former being to the latter in the relative ratio of 1·5—2—2·5 to 3. The cause of this alteration in the relative duration of the expiratory to the inspiratory murmur is found in the impediment which is offered, by the hypertrophied basement structure, to the free movement of the fibres of the pulmonary cells during expiration.

This impediment is both of a *negative* and *positive* character—

negative, from a diminution in the quantity of air admitted into the affected pulmonary cells—from their consequently diminished inflation and the deficient excitation, relatively considered, of their fibres—and *positive*, from the feeble contraction of such fibres, as the result of defective excitation—from the slowness of such contraction, as the result of such feebleness—and from the passive obstacle which the thickened basement membrane simply and abstractedly offers to the moving fibres of the cells.

The difference in the *quality* of the inspiratory and expiratory murmurs is detected in the roughness or harshness of these murmurs as compared with those of health. Of the two murmurs, clinical experience ascribes a greater amount of harshness and intensity to the inspiratory than to the expiratory, the reason of which appears to be, that the inspired air enters the pulmonary cells with greater force than it quits them, and that, therefore, the generating causes of the vesicular murmur, during inspiration, are in greater intensity than those of expiration. The existence even of a vesicular expiratory murmur has been denied, by many authors, to the healthy and most complete action of the lungs; but a closer investigation of the subject, and a more correct appreciation of the sounds of the chest, confirm the existence of this murmur. In the morbid condition of the pulmonary cells above-named, the "prolonged expiration" leaves no doubt as to the existence of such sound during the movement of the cells in expiration. Whilst the pitch or intensity of this vesicular murmur depends on the intensity of the causes producing it, its *quality* depends, to a certain extent, on the nature of the surface over which the entering and emergent columns of air pass. If the interior surface of the pulmonary cells is rendered irregular by the unequal (Fig. 21) deposit of tuberculous exudation, the uniformity of pitch in the vesicular murmur is destroyed; because, during inspiration, the air does not impinge with equal force on every point of the interior surface of such cells. Hence, the pitch is irregular, and this irregularity impresses the auscultator with the idea of roughness and waviness.

It has been already stated, that when basement thickening is slight and of very limited superficial extent, the modification in the respiratory murmur is so trifling as to be scarcely recognised by auscultation. When, however, this increase of thickness pervades the basement structure of one or more entire lobes, the physical signs of the chest generally are altered. Thus, a comparison between the two sides of the chest shows, that percussion elicits a duller sound over the diseased portion of lung—that the vesicular murmur, although rough, is of lower intensity than in the corresponding portion of the opposite lung—that a forcible inspiration evokes a small, sharp bronchial note sounding through the diseased pulmonary tissue—that a short, hacking, and dry or moist cough is more or less present—that the vital capacity of the chest is sensibly reduced—that the breathing, less deep than natural, is slightly

accelerated, and particularly so under excitement or exertion—that the ribs are observed by the stethometer to move less over the diseased lung—and that a degree of flattening of the surface of the chest at this point exists. To these signs must be added that of a reduction in the weight of the body, which reduction always and invariably progresses in proportionate ratio to the advance of the pulmonary disease. It, indeed, is impossible for the vital capacity of the lungs to be encroached upon without being attended by a corresponding reduction in the weight of the body; therefore the consideration of weight is of constant and primary importance in the estimation of the progress of pulmonary tuberculosis.

Now, during the gradual thickening of the basement structure just detailed, there is often a sensible change in the quantity of mucus eliminated. The increase is in proportion to the extent of membrane which receives a more than ordinary supply of blastema. Thus, when this inordinate supply is limited to the membrane of a few lobules only, the quantity of mucus expelled but slightly exceeds that of the healthy action of the membrane; but when the membrane of one or more lobes has its action thus increased, the quantity of mucus expelled in twenty-four hours not unfrequently amounts to several ounces. The identity between this condition of the membrane and that which characterises the second pathological condition of epithelial bronchitis and of pulmonary cellulitis, is apparent, and the analogy thus shown to exist between chronic pulmonary tuberculosis and simple epithelial inflammation of the bronchio-pulmonary membrane itself, will be still further made manifest when I shall compare the severe form of bronchitis with acute pulmonary tuberculosis.

The sputum expelled in this stage of the disease, is more or less tenacious and ropy, of a light or dirty drab colour, at first somewhat aërated and of regular consistence, afterwards less frothy and of irregular consistence. Microscopically examined, it is found to consist essentially of large well-developed mucus-corpuscles, varying in size from $\frac{1}{2400}$th to $\frac{1}{2100}$th of an inch in diameter, more granular, and the granules frequently larger than those of the ordinary mucus-cells of the membrane. With these are associated a moderate quantity of epithelial patches, the cells of which have all but attained perfect growth, but which yet cohere by their edges, and thus prove by this fact, and by the granular appearance of each cell, that in the elimination of the natural secretion of the membrane, the epithelial becomes the true mucus-cell. It would, I conceive, be indeed difficult for any observer who has carefully watched the microscopic characters of the sputum to arrive at any other conclusion.

In the very mildest form of this stage of pulmonary tuberculosis, there are seldom other bodies than those just named in the sputa; but in those cases which are intermediate in severity between this form and acute pulmonary tuberculosis, exudation-cells, similar to

those present in the sputa of simple inflammation of the bronchio-pulmonary membrane, exist in more or less limited quantity.

If we now compare the pathological condition of the membrane, in this comparatively slight stage of deposition, with that of the second pathological condition of epithelial bronchitis, we find that the analogy between the two diseases is still maintained. Thus, in both, the membrane of the affected part is of deeper coloration than natural, more spongy, and besmeared with an excess of mucus; whilst here and there, over its surface, are small superficial patches of increased redness, which are owing to the shedding of the epithelium at these points.

The presence of a more than ordinary quantity of mucus on the surface of the affected membrane, gives rise to various rhonchi of a crepitant, subcrepitant, submucous and mucous character, according to the particular site of the origin of such sounds. Of these rhonchi the subcrepitant is the most frequent, and when heard with some degree of persistence in the upper lobe of one or both lungs, it affords, in conjunction with other signs, a tolerably sure indication of the presence of pulmonary tuberculosis to the extent above-named.

Now, the pathological condition of the bronchio-pulmonary membrane at this stage of the disease, and the particular microscopic characters of the sputum, show that recovery, under such circumstances, is very possible; and that such is frequently the case, the experience of every practical physician, accustomed to treat such cases, abundantly testifies. The indications of improvement are observed in a gradual diminution and cessation of expectoration, in the change from moist to dry rhonchi, in a certain degree of improved breathing, and in an improvement of the general health. The cough, however, does not so readily subside; yet it is less frequent than before, and is now chiefly occasioned by any cause which suddenly increases the vascular condition of the membrane. Although the moist rhonchi may subside, yet the roughness of the respiratory murmur, and the prolonged expiration before named, are still present, because the thickened, irregular condition of the cell-walls, on which these physical signs have been shown to depend, still exists. So also the flattening of the chest remains even after the complete cessation of cough and of the re-establishment of the former vital capacity of the chest, by a compensatory action of the unaffected portions of lung. The cases most likely to undergo this improved change, are those of acquired pulmonary tuberculosis, because there is, to a certain extent, an absence of that natural delicacy of the tissues which is imposed upon the organism by hereditary predisposition. Nevertheless, clinical experience shows that even many such cases, in which the hereditary taint is well marked, recover again and again from distinct attacks of tubercular deposition. If such attacks occur somewhat suddenly, they are apt to be regarded in the light of bronchitis or pneumonia, and so far as the nature of the symptoms

and the seat of disease are concerned, such terms are sufficiently expressive; but, in restricting the disease to these terms, the deficient vitality of the blood-plasma, which constitutes the very essence of it, is apt to be overlooked. Hence, the term "inflammation" does not comprehend the whole pathological import here involved; and since it fails to define the essentiality of the disease—*i.e.*, the deficient vitality of the blood—it can only be regarded as an expression of the mode in which such disease acquires a localised or tangible form. It may here be asked whether or not, on the cessation of deposition and on the apparent restoration of the general health, the thickened basement membrane is ever reduced to its original diameter. So long as this hypertrophied basement structure does not press upon the subjacent capillaries, and thereby interfere with the circulation and vital process of the part, it may undergo gradual disintegration and removal without any breach of structure; but when, by its pressure, it arrests, as in the more advanced forms of deposition, the flow of blood through the affected parts, their normal tissues die, the exudation itself germinates, is cast off, and thus a breach of structure is produced. There are two ways in which the hypertrophied basement structure may be reduced to its normal thickness—the one, by its slow disintegration and absorption into the vessels—the other, by its gradual disintegration at the epithelial surface of the bronchio-pulmonary membrane. In the mere thickening of the basement membrane above described, the tuberculous exudation is gradually liquified by the natural fluid transudation from the vessels, and it is then either taken up by the veins and absorbents, or carried forward to the epithelial surface, whence it is eliminated by the epithelial cells. If, however, the tuberculous exudation is, in the natural waste of the basement membrane, brought to the surface of the latter, and, therefore, within the range of the application of this structure to its growing nuclei, the latter do not readily appropriate the tuberculous matter in the production of their cell-walls, and thus imperfectly developed cells arise, and are cast off with the unappropriated tuberculous matter, in the form of dark, granular basement patches. Before, however, such removal of the tuberculous exudation can be perfected, another local predisposition may arise in the same part, to be followed by another exudation and a return of all the previously described conditions in a greater degree. Thus does the basement membrane, by repeated attacks of this kind, become at length so thickened, that it can no longer furnish the pabulum for epithelial growth; mucus-cells, consequently, cease to form on its free surface; and this absence of waste, in conjunction with the continual addition which is taking place at the attached surface of the basement membrane, soon produces closure of the affected pulmonary cells, as shown in Fig. 21, Plate I.

In another form of chronic pulmonary tuberculosis there are no periods of well-marked deposition; but the disease is, on the contrary, gradually progressive from the first invasion of the pulmonary

tissue. In such cases there is, for an indefinite period, a gradual decline of the general health and strength, accompanied by dry, short cough, slightly hurried breathing, quick, small pulse, frontal headache, vomiting of food, and a train of dyspeptic symptoms. The stethoscopic signs reveal a generally diminished respiratory murmur throughout the upper lobes of the lungs; and the roughness of the inspiratory murmur before noticed is more particularly observed during forced inspiration. As yet the loss of symmetry between the two sides of the chest is not manifest; nevertheless the vital capacity of the lungs is found by experiment to be reduced, and the breathing, although but trifflingly accelerated during perfect quietude, is visibly accelerated by exertion. Percussion elicits the same sound on comparison of the two sides; but this sound, when compared with that of a healthy chest of the same vital capacity originally, is somewhat duller than the latter. These signs arise out of the generally hypertrophied basement structure of the affected lobes, by which the pulmonary texture is rendered denser, and its static and dynamic conditions are thereby altered. Soon, however, this progressive equality in the advancing pulmonary consolidation is destroyed, and the upper half of one lung proceeds more rapidly than the other to more or less complete occlusion of the pulmonary cells. There is now no longer any equality in the physical signs of the two sides of the chest. Inspection at once detects flattening of the chest and diminished mobility of the ribs on the advanced side, greater dulness on percussion, absence of vesicular murmur, rough bronchial breathing on forced inspiration, and increased resonance of the voice. When the consolidation is limited to the middle lobe, and is in close contiguity to a large bronchus, the transmission of the voice is sometimes so audible and so articulate, that bronchophony might easily be mistaken for pectoriloquy, were the determination of the latter left to the mere nature of the breathing and of the voice. As the consolidated lung becomes a better conductor of sound than the natural spongy texture of that organ, the heart's sounds are heard with unnatural clearness where any amount of tubercle is deposited.

In acute pulmonary tuberculosis, the stage of deposition is rapidly brought about. It is also more general throughout the lungs than is the chronic form, and it not unfrequently progresses with almost equal rapidity in both the upper and lower lobes. It affects alike the basement membrane of the minute bronchi and pulmonary cells, and thus developes, in its etiological characters, the physical signs and symptoms of bronchitis and pneumonia.

In this form of pulmonary tuberculosis, the exudation is always copious, and not only does it augment the natural diameter of the basement structure, but it also passes into the submucous tissue and upon the denuded surface of the mucous membrane of the bronchi, where it coagulates and forms irregular masses of exudation, or casts of the minute bronchi, according to the quantity poured out in proportion to the caliber of the affected tubes. It likewise passes into

the interior of the pulmonary cells, which are thereby more or less obliterated, and sometimes it is effused amongst the fibres of which the walls of such cells are in part composed. Thus are the pulmonary cells obliterated, the tissue of the lungs more or less consolidated, the minute bronchi narrowed and more or less occluded, and the functional balance between the respiratory and general systems destroyed. The sudden encroachment on the respiratory function leads to a rapid falling off in the weight of the body as before explained—the pressure of the exudation upon the pulmonary nerves excites an almost constant cough; whilst pain in any part of the chest, but more particularly in the side, is often present as the consequence of such cough, or of sympathetic irritation, or of both these conditions conjointly. As the deposition generally involves both lungs, and often in a nearly equal degree, the loss of symmetry between the two sides of the chest is not at first so palpable as in the chronic form of the disease; nevertheless the mobility of the ribs is less than natural, whilst the number of respirations is manifestly increased. Owing, too, to the partial consolidation of the pulmonary tissue, the percussion-note of the chest is reduced; nevertheless there is, on comparison between the two sides, frequently wanting that marked difference which is so significant of the relative condition of the two lungs in chronic pulmonary tuberculosis. With the occurrence of exudation in acute pulmonary tuberculosis, the stethoscopic signs quickly pass from a dry to a moist character. Thus, in cells, but partly filled with exudation, the crepitant rhonchus is heard; whilst the subcrepitant and submucous rhonchi are first, and afterwards the mucous rhonchus is, present in the affected bronchi. In the last mentioned tubes a deep and forcible inspiration, by suddenly stretching their fibres, excites a partial spasm, which adds, for a few seconds, various modifications of cooing, chirping, and whistling to the other morbid sounds. When from closure of the pulmonary cells by exudation, or from collapse caused by occlusion of the minute bronchi, the function of one or more lobules is destroyed, the adjoining cells may adopt a compensatory action, which, owing to the increased pressure of the inspired air upon the interior surface of such cells, and to the impediment offered to the contraction of their fibres by the thickened basement structure, not unfrequently leads to their enlargement, which thus constitutes one form of emphysema.

With this general deposition in the texture of the lungs, there is frequently the same kind of exudation upon the surface of the pleura, the physical signs of which are thus added to those already noticed. In tracing the pathological anatomy of such exudations, microscopic observation clearly shows that analogous changes to those observed in mucous membrane, occur in the pleura and other serous structures. Thus, before exudation occurs upon the free pleural surface, a shedding of epithelium takes place in extent proportionate to the exudation, and this result is an invariable concomitant of a copious

exudation, as generally occurs upon the pleura in acute pulmonary tuberculosis. When, however, the exudation is small in quantity, as occasionally occurs in chronic pulmonary tuberculosis, a gradual thickening of the basement membrane of the pleura and of the subserous tissue takes place, after which the epithelium is cast off, disintegrated, and probably removed by absorption into the vessels. This condition of the pleura is not only necessary for the occurrence of exudation upon its *free* surface, but also for the formation of adhesions, which are of frequent occurrence in chronic pulmonary tuberculosis. When these are recent, microscopic observation shows them to consist of fibrillæ often of irregular thickness, the average diameter being $\frac{1}{16.000}$th or $\frac{1}{14.000}$th of an inch, short, friable, and interspersed with granular matter and minute exudation-cells, varying in diameter from $\frac{1}{9000}$th to $\frac{1}{5000}$th of an inch. If the exudation has been copious and rapid, as is sometimes the case in acute pulmonary tuberculosis, there are in addition plastic and pus-cells generally of diminutive growth. The production of the last-mentioned cells is not nearly so frequent upon the pleura as upon the mucous surface of the bronchi and pulmonary cells, and this fact is readily explained by the greater access of oxygen to the exudation in the latter situation, by which the adipocerous degeneration, necessary for the conversion of the plastic into the pus-cell, is more readily accomplished. When such metamorphosis occurs in the protein compounds of a tuberculous exudation either upon the pleural or peritoneal surfaces, there is a strong disposition in such exudation to separate into its proximate and elemental constituents. To this cause may be ascribed the existence of large, well-defined prismatic crystals of the triple phosphate of lime, which I have frequently observed in both pleural and peritoneal exudations. That such crystals were formed without the vessels, is proved by the fact that none were less than $\frac{1}{2000}$th, whilst most of them equalled $\frac{1}{1000}$th of an inch in their longest, and $\frac{1}{2700}$th of an inch in their shortest diameter. Such crystals, granting that they might be propelled along the capillary vessels, were manifestly unable to pass through the molecular spaces of their walls. Their formation, therefore, must either depend on crystallization from the fluid portion of the exudation, or, what is more probable, on the metamorphosis of such exudation, which allows the relative constituents to assume more or less a separate and isolated form.

In reviewing all that has been said in reference to the stage of deposition of acute pulmonary tuberculosis, it would be a matter of surprise were such mischief as has been observed to take place in the pulmonary organs, and to be characterized by the signs already enumerated, to exist without its being attended by considerable disturbance in the general system. The nice adjustments of the system, the dependence of one part on another, and the community of action of the whole, forbid such a supposition. On the contrary, the general system feels both the blood-disease and local affection, and these it

symptomatizes by a hot skin, occasionally bathed with perspiration; quick, small pulse; red, fiery tongue, or red at the edges, tip, and along the middle, with a thin, white fur over the remainder of its dorsum; thirst, loss of appetite, irregularity of bowels, often diarrhœa, high-coloured, scanty urine, depositing urate of ammonia on cooling, and great and rapidly-increasing muscular debility. These general symptoms, as well as the physical signs, vary in their intensity in different cases; but in all they are well marked, and as a general rule, terminate only in the death of the patient, which generally occurs between the sixth and the sixteenth week. Such cases are almost invariably connected with hereditary predisposition to tubercle; but now and then an instance occurs in which no such predisposition can be traced, and where the disease seems to be acquired by causes which, for a long time, undermine the general health, and which are then followed by typhoid fever. Should death occur during the stage of deposition, the morbid appearances further enable us to trace the analogy which has been before shown to exist between this disease and bronchitis and pneumonia. Thus, in the smaller bronchi, there are increased coloration of the membrane, epithelial desquamation at different points, exudation upon the basement membrane laid bare by such epithelial desquamation, as well as into the submucous tissue, and more or less occlusion, by exudation, of the terminal bronchi; whilst, in the cellular portion of the lungs, we find consolidations of more or less extent, accompanied by hyperæmia, and, consequently, by increased depth of colour. Hitherto, then, pulmonary tuberculosis has been shown to observe the same minute process in its development as does simple inflammation of the lungs. Its specialty of character, which has been acknowledged from the infancy of medical science, cannot, therefore, depend on the particular mode of etiology which it may observe, but upon the innate character of the blood, from which all exudations take place, and which determines the specific nature or otherwise of all diseases characterized by an excess of blood-plasma in the affected parts.

Stage of Germination.

The last vital phasis, which tubercle assumes in the pulmonary organs, is that of germination and growth—a condition which corresponds to the "softening" of authors. This phasis, instead of being denominated the "death" of tubercle, ought, in my opinion, rather to be regarded as its greatest manifestation of life, which having been attained, is, as is the case with all other exudations obtaining no higher degree of development than that of cells, immediately followed by death and the expulsion, if possible, of the effete product from the system. The period at which the stage of germination succeeds that of deposition, varies considerably in chronic pulmonary tuberculosis; but, in the acute form of that disease, the

former succeeds the latter so closely that there is no appreciable interval between the two. In the chronic form, however, distinct intervals of very indefinite duration frequently occur, during which the patient may regain a degree of health and strength which approaches more or less to that of the original condition of the system. Such improvement depends on the return of the pulmonary capillaries engaged in the work of deposition to their original diameter—on the consequent removal of an excess of blood from the seat of disease—and on the absorption of the fluid portions of the exudation, by which the respiratory capacity is enlarged, and thereby enabled to maintain, in healthy waste and repair, an increased bulk of the body, which always accompanies the improvement just indicated. The degree of this improvement in the respiratory capacity must, of course, be in the inverse ratio of the amount of tuberculous deposit and of the obliteration of the pulmonary vesicles. Hence, a very limited deposit permits the system, after the stage of deposition has passed away, to recover, in a great measure, its wonted health and strength; but where the tuberculous deposit continues to be extensive, the natural strength and weight of the body are never regained.

Now, the first appreciable sign of the germination of pulmonary tubercle is more correctly indicated by the microscopic characters of the sputum than by the auscultatory phenomena of the chest, or by general symptoms. As the first evidence of the germination of tubercle in the lungs, there is discharge from the bronchio-pulmonary membrane, which is invariably present during this stage, but which, nevertheless, is not diagnostic of that occurrence, inasmuch as it has been shown to exist in many cases during the stage of deposition. When, however, the stage of germination succeeds that of deposition which has been unaccompanied by discharge from the bronchio-pulmonary membrane, the sputum first expelled is thin, frothy, generally more or less aërated, of a light drab colour, of tolerably equal consistence, and presents under the microscope epithelial patches and well-developed mucus-corpuscles floating in a very granular fluid menstruum. This kind of sputum corresponds to that of epithelial bronchitis, except in the cells and epithelial patches being generally somewhat more granular than those of the latter. In the sputum first discharged in acute pulmonary tuberculosis, the mucus-corpuscles and epithelial patches are still more granular than those of the chronic form. This is well shown in Fig. 22, Plate I., which represents the first sputum in a case of acute pulmonary tuberculosis, which, in a young stone-mason, twenty-six years of age, ran its course, and ended fatally in eleven weeks.

Very shortly, indeed, after the establishment of expectoration, the sputum becomes thick, heavy, irregularly consistent, of a dirty drab, yellowish-white, or yellowish-green colour, pencilled with yellow or yellowish-white lines, and sometimes streaked or mixed with blood. It is viscid, tenacious, leaves the vessel in a thick, ropy

mass, is neutral or faintly alkaline to test-paper, and is generally expelled in quantity from three to four, but sometimes as many as eight ounces in twenty-four hours. Microscopically examined, it exhibits numerous highly-developed, and, generally, very granular mucus-corpuscles, varying in size from $\frac{1}{2400}$th to $\frac{1}{1700}$th of an inch in diameter. There are also present patches of epithelium, basement patches, exudation masses, and isolated exudation-cells from $\frac{1}{7000}$th to $\frac{1}{2500}$th of an inch in diameter, plastic and pus-cells in variable numbers, together with bronchial casts and dark pigmentary matter. This appearance of the sputum shows that ulceration has already commenced in the bronchio-pulmonary membrane—a fact of which the stethoscopic signs can as yet give no satisfactory indication. By and by small whitish, yellowish-white, semi-solid, cheesy-looking particles are superadded in variable numbers to the sputum. These have long been regarded as softened tuberculous matter; but microscopic observation demonstrates that they are sometimes minute portions of pulmonary tissue studded with tuberculous exudation. It is true that these masses[1] consist essentially of tubercle; but not unfrequently they imbed some one or other of the normal tissues of the lung, which, by the pressure of the tuberculous exudation, has lost its vitality, and is thus cast off during the growth of the exudation into cells. The most frequent appearance of one of these minute masses represents more or less of cell-development, mingled with much granular matter, with here and there a patch of imperfectly developed epithelium. With these are sometimes associated irregular nodules of pigmentary matter, isolated portions of broken fibrous tissue of the pulmonary cells, and, now and then, several whole pulmonary vesicles, some of which are entirely, and others but partly, filled with tuberculous matter. Between the cells thus observed, a light greyish coloured band pursues a definite course, and in this band the natural fibrous tissue of the lungs is more or less distinctly seen. On adding liquor potassæ the albuminous portion of the contents of the cells is dissolved, and the fibrous tissue rendered more distinct (Fig. 23, Plate I.) To the presence of these fibres in the sputum, M. Lebert attaches considerable importance in a diagnostic point of view; and it is certain that, in this respect, his observations are of the greatest value. I have, however, more frequently observed these fibres in the sputa during the actual formation of caverns, than when such excavations have existed for some time. Their presence, nevertheless, in the sputa, at any period, indicates certain destruction of pulmonary tissue; but

[1] Minute particles of bread, and frequently milk coagula are associated with the above-mentioned masses, and are generally more numerous than the latter in the sputum at this stage. The particles of bread in question cannot be distinguished from tuberculous masses by the *naked eye*, but the milk coagula frequently admit of recognition in this way. The microscope, however, at once determines the real nature of both. As a general rule, the number of tuberculous masses in the sputum is comparatively small.

their absence, on the contrary, by no means shows that such destruction has not taken place. The presence of basement patches is of much more frequent occurrence, and they, at the same time, indicate an earlier stage of ulceration. Hence, considerable diagnostic and practical value attaches to the microscopic examination of the sputum during the germination and growth of pulmonary tubercle.

Such, then, are generally the microscopic characters of the cheesy-looking portions of tuberculous sputum. Of an aggregation of such minute masses the larger masses of pulmonary tubercle are composed. Hence each distinct mass, which results from such aggregation, is intersected by numerous portions of pulmonary tissue; and if the vessels of each of these portions could transmit their blood, the process of germination or softening would take place simultaneously at all these points by direct exudation from the vessels; but as many of the vessels involved in a large tubercular mass are, by reason of the pressure of such mass, more or less incapable of transmitting blood, the exudation necessary for germination and growth is supplied by the nearest vessels of the mass which can transmit their blood, and the fluid portion of the exudation being taken up by simple imbibition, and transmitted as it were from particle to particle, the whole simultaneously, or nearly so, begins to germinate and thus to develope cells. As, however, the conditions of cell-growth are more favourable at the circumference than at the centre of the mass, the process of germination proceeds more rapidly at the former than at the latter point of the seat of disease. The opinion of Laennec—"Ce ramollissement commence vers le centre de chaque masse, où la matière tuberculeuse devient de jour en jour plus molle et plus humide, caséiforme ou au moins onctueuse au toucher comme un fromage mou, puis acquiert la viscosité et la liquidité du pus. Le ramollissement gagne peu á peu la circonférence, et devient enfin complet"—is not borne out by the most rigid application of the important aids to the investigation of morbid tissues which we have more or less acquired since his time. His statement that the circumference of the tuberculous mass is gradually engaged in the process of softening subsequently to the centre, may acquire an *apparent* but not a *real* truth from the fact, that an increased exudation always accompanies the process of germination or softening; that this exudation takes place around the circumferences of the already existing masses of tubercle; that whilst the latter readily imbibe the liquid portion of the exudation, as one of the necessary conditions of germination, the solid portion of such exudation is deposited in the contiguous and as yet comparatively uninvaded pulmonary tissue; and that when this last-mentioned deposit does germinate, its period of doing so is posterior in time to that of the original deposit. Hence an *apparent* truth in Laennec's remark above quoted, which, however, disappears when it is remembered that the deposit which takes place in a portion of lung contiguous to

a germinating mass of tubercle is to be considered as separate and distinct from the latter. This subsequent deposit may or may not germinate, according to the particular conditions of the system. Should it not do so, it limits the extent of pulmonary excavation occasioned by the softening and expulsion of the original deposit, and thus far contributes to recovery even after excavations have been formed in the lungs. The question may here be asked, whether or not, at the above-mentioned stage of germination, perfect recovery can take place. Experience proves that such a result is of very frequent occurrence, and microscopic investigation is certainly favourable to the possibility of the healing of the minute ulcerations of the bronchio-pulmonary membrane, which, as a general rule, characterise this stage of the disease. When the number of exudation-cells is small throughout the sputum, when also there are comparatively few patches of imperfectly developed epithelium, and when the quantity of fluid thrown off by the affected membrane does not visibly increase from day to day, recovery is very frequently brought about. These microscopic objects show that, at present, ulceration is limited to the bronchio-pulmonary membrane, which will, under favourable circumstances, heal as readily as tuberculous ulcerations of other tissues. When, on the other hand, the cheesy-looking portions of the sputum contain whole pulmonary vesicles, or portions of the fibrous tissue of the cells, or of the minute bronchi, it is certain that the pulmonary tissue itself is being broken down, and that the probability of recovery is less than in the previously mentioned cases. The degree of rapidity with which such destruction of the pulmonary tissue takes place is indicated, to a very great extent, by the number of exudation-cells present in the sputum, in which, in acute pulmonary tuberculosis, they are quite as numerous as in the sputum of acute bronchitis. They, indeed, may be taken as a measure of the rapidity with which the disease is progressing at this stage; but when accepted in conjunction with the evidence afforded by other portions of the sputum, by the physical signs of the chest, and by general symptoms, they render the prognosis, which is the legitimate deduction of such conditions, all but certain of realisation. This prognosis is, in acute pulmonary tuberculosis, always unfavourable, owing to the great extent of pulmonary tissue which is more or less suddenly involved, and to the direct effect thereby produced on the respiratory function, and, through it, upon the general system; whilst, in chronic pulmonary tuberculosis, it is more or less unfavourable, according to the particular conditions above stated. The auscultatory signs present at this period are those arising out of consolidation of the pulmonary tissue and of the presence of fluid in the pulmonary cells and minute bronchi, to which must be added the loss of symmetry, deficient movement of the ribs on the affected side, and increased dulness on percussion, as noticed in a previous stage of the disease.

The following table represents the chemical composition of the

sputum in sixteen cases of commencing germination of pulmonary tubercle:—

IN 100 PARTS.

No.	Organic Matter.	Chlorides of Sodium and Potassium, Alkaline Sulphates and Phosphates.	Salts of Lime.	Water.
1	4·40	·36	·17	95·07
2	3·94	·70	·23	95·13
3	9·31	·83	·25	89·61
4	4·30	·56	·19	94·95
5	4·37	·62	·10	94·71
6	8·12	·51	·28	91·09
7	3·56	·56	·16	95·72
8	4·95	·64	·24	94·17
9	3·92	·92	·36	94·80
10	2·37	·55	·70	96·38
11	3·55	·77	·18	95·50
12	3·65	·80	·35	95·20
13	3·60	·55	·05	95·80
14	3·65	·60	·47	95·28
15	4·49	·78	·18	94·55
16	3·01	·74	·22	96·03
	71·39	10·49	4·13	1513·99
Average	4·46	·65	·25	94·62

On comparing the average of the above table with the averages of healthy mucus and of the sputa of acute and chronic bronchitis and of pulmonary cellulitis, the following tabular relation is observed:—

AVERAGE IN 100 PARTS.

Healthy Mucus.		Sputum of Acute Bronchitis.	Sputum of Pulmonary Cellulitis.	Sputum of Chronic Bronchitis.	Sputum in commencing germination of Pulmonary Tubercle.	Sputum in advanced germination of Pulmonary Tubercle.
Organic matter,.....	·68	2·15	3·68	1·72	$4·46\frac{5}{16}$	$5·80\frac{6}{15}$
Chlorides of sodium and potassium; Alkaline sulphates and phosphates;	·12	1·10	1·47	·92	$·65\frac{9}{16}$	$·61\frac{6}{15}$
Salts of lime,.....	a trace.		·05		$·25\frac{3}{16}$	$·22\frac{4}{15}$
Water,.....	99·20	96·75	94·80	97·36	$94·62\frac{1}{16}$	$93·35\frac{1}{15}$
	100·00	100·00	100·00	100·00	100·00	100·00

It thus appears that the sputum of commencing germination of pulmonary tubercle exceeds, in organic matter and the salts of lime,

that of all the other pulmonary diseases, which have as yet been considered; whilst, in reference to the presence of alkaline salts, it affords a less quantity than even the sputum of the early stage of chronic bronchitis. These facts are in close harmony with the views entertained by pathologists, as to the chronic character of pulmonary tuberculosis, as well as with the statement made in an earlier part of this work, respecting the relative ratio which the alkaline salts bear to the degree of cell-growth in exudations poured out by the vessels of the bronchio-pulmonary membrane. The greater the degree of cell-growth in such cases, the greater is the amount of alkaline salts in the sputum. Hence, as in inflammatory exudations from this membrane the degree of cell-growth exceeds, as a general rule, that of tuberculous exudation from the same structure, the reason why the sputa of the former diseases exceed that of the latter in the amount of the alkaline salts. This constant ratio between the above-mentioned constituents of morbid sputa may, by legitimate deduction, be regarded as a measure of the activity of disease. According to this statement, then, pulmonary tuberculosis is less active in its progress than the early stage of chronic bronchitis; yet the latter, owing to the marked remissions and to the total arrestment which it, from time to time, undergoes, may allow a greater prolongation of life than the former, which, when once it has reached the stage of germination, is not so amenable to treatment, and consequently pursues a more steady progress towards death. This remark, however, does not apply to acute pulmonary tuberculosis, which, classified in point of activity by the above-mentioned particulars, holds an intermediate place between acute and chronic bronchitis. It has already been remarked that tuberculous sputum, at the commencement of germination, contains a greater amount of organic matter than do inflammatory sputa. This is evidently owing to the breaking down of the pulmonary tissues, and to their presence in the sputum in masses which may yet retain, more or less, their organized form, as well as in molecularly disintegrated particles, as the result, not so much of active cell-growth, as of the partial death of such pulmonary tissues by the mechanical pressure of tuberculous exudation in a quiescent state, and of the further loss of vitality by the subsequent disposition of the exudation to germinate.

As to the presence of an increased quantity of lime in tuberculous sputum, as compared with the ratio of this salt to the other constituents of inflammatory sputa, the source whence the increased quantity is derived in pulmonary tuberculosis is not, perhaps, manifestly determined. As there is no evidence to show that the normal constitution of the blood of tuberculous patients contains an excess of this salt, it seems pretty certain that it is derived, during the progress of disease, from the molecular disintegration and decomposition of one or other tissue of the body. Laennec says:—"Les os ne perdent pas de leurs dimensions en longeur; mais il m' a souvent paru

que leur circonférence diminuait quand le marasme durait longtemps. Ils deviennent spécifiquement moins pesans, et il en est sans doute de même de tous les autres organes." It is probable that this statement correctly indicates the source whence the increased quantity of lime in the sputum is derived, and this probability receives additional support from the fact, that the same excess of lime occurs in the urine of such patients. If the above statement of Laennec is a positive truth, the quantity of lime in the sputum and urine may be accepted as an indication of the degree of waste which the osseous system is undergoing; and as we know that this system is one of the last, if not the last, to be thus influenced, the probable fact of its being so influenced in such cases, would alone impress us with the idea of the gravity of the disease with which we have to contend. Some portion of this excess of lime occasionally assumes a prismatic crystalline form, and as such it becomes a beautiful microscopic object of the sputum. But crystals of the triple phosphate are to be regarded as *accidental* and not as *regular* constituents of the sputum. In this category, too, I may enumerate cholesterine, urate of ammonia, biliphein, cystine, urea, and certain blue particles, the exact nature of which have, in two instances, been determined. (Fig. 24, Plate I.) Cholesterine in clearly well-defined tables is seldom present; but in confused and irregular plates, it is of more frequent occurrence. When the solid residue, left after the evaporation of tuberculous sputum, is boiled for a short time in alcohol, strained whilst hot, and subsequently allowed to evaporate, it deposits a reddish-brown, uncrystallized mass. On treating this mass with concentrated nitric acid, aided by a gentle heat, the cholesterine is dissolved, and by allowing the fluid to cool, and on adding water, a light yellow substance (cholesteric acid), not unlike butter, is immediately formed. This substance readily combines with the alkaline and other bases to form salts, all of which are more or less coloured. It is also found to have precisely the same chemical reactions as the cholesteric acid obtained, by the process above-mentioned, from human bile.

With respect to the presence of urea in the sputum, one important fact deserves especial mention—namely, that it is preceded by a marked diminution of the usual quantity of urea in the urine, and this diminution by the occurrence of albuminuria. These conditions more frequently arise, as will hereafter be shown, in the advanced than in the early stage of the germination of pulmonary tubercle, and they then become the sure harbingers of death. To separate the urea from the sputum I adopt the following method:—A thousand grains of sputum are evaporated to dryness over a water or sand-bath, the dry residue is triturated in a mortar, and subsequently boiled in alcohol. The alcoholic solution is poured off as soon as the more heavy sediment has subsided, and is then carefully evaporated. The residue of the evaporation, consisting of the fatty matter and urea, is next treated with pure nitric acid, aided, for a few

minutes, by a gentle heat. The fatty matter and urea are in this way dissolved, forming a golden yellow solution. The heat is now withdrawn, and the solution allowed to cool. If, after cooling, a sediment subsides, the supernatant fluid is poured off, and to the latter cold distilled water is added until white or yellowish-white flocculent masses cease to form. The fatty matter is thus allowed to subside and to collect at the bottom of the vessel, after which the fluid portion, holding in solution the nitrate of urea, is passed through a filter, and to the filtered fluid carbonate of potassa is added until all effervescence has ceased. The solution is now carefully evaporated, and the dry residue, left after evaporation, is heated with absolute alcohol, which dissolves the urea and leaves the nitrate of potassa as an insoluble residue. The alcoholic solution, poured off, is allowed to evaporate, and the urea is thus obtained free from impurity.

As to the presence of the blue pigment above-mentioned, it appears in small, irregular, isolated masses of a rich blue colour, and sometimes in regular elongations as though it formed the contents of a vessel. It sometimes appears simultaneously in both the sputum and urine, in the latter of which it is best found in the sediment which subsides after some hours' repose. Numbers 9 and 11 of the previous table were instances of this kind. The sputum and urine of the former case were sent to me, at my request, from the hospital for consumption, at Brompton, by Dr R. Quain, to whom my thanks are especially due for his kindness in supplying me, during my investigations, with other specimens of sputa and urine from the same source. Liquor potassæ, added to the blue pigment under the microscope, converted it into a bright yellow, which was again changed into a rich blue by the subsequent addition of the tincture of the sesquichloride of iron. Both the physical appearance and the chemical reaction of the blue pigment were, in this instance, as also in number 11, those of Prussian blue; and with the view of ascertaining how far its constituents were in number 9, derived from sources exterior to the system, I addressed a letter to Dr R. Quain, asking him whether or not, at the time the urine and sputum were collected, any preparation of iron was being administered to the patient. I received from him the following reply:—"The patient to whom your inquiry refers, was taking, at the time the urine and sputum were collected, the tincture of the sesquichloride of iron." This reply of Dr Quain, while it lends a degree of conformation to the recognition of the blue pigment in question as Prussian blue, shows also the source whence one of its constituents was, in all probability, derived.[1] It must not, however, be supposed, that this salt exists in every specimen of sputum or urine in which blue particles

[1] Number 11 had been taking the sulphate of iron in conjunction with the disulphate of quinine upwards of three weeks before the blue particles in question were observed in the sputum and urine.

are observed by the microscope. Minute portions of soot, floating in the atmosphere of an apartment, are apt to fall into open vessels containing urine and sputum, and these under the microscope exhibit more or less a blue colour. It will, therefore, be more prudent and much more conducive to the ends of science, to discard all idea of the presence of Prussian blue, when the chemical reactions of this salt do not confirm the physical appearance of the object under investigation.

The following table shows the composition of the urine in sixteen cases of commencing germination of pulmonary tubercle. The numbers correspond with those of Table I. of the sputum:—

TABLE I. OF URINE.

No.	Quantity in 24 hours.	Urea in	Uric Acid and Vesical Mucus in	Fixed Alkaline Salts in	Phos. of Lime and Magnesia in	Animal Extractives and Am. Salts in	Water in	Albumen in
	Fluid oz	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000grs
1	40	3·100	2·000	5·900	1·000	13·950	970·05 0	4·000
2	32	5·130	4·800	7·500	3·100	23·660	950·810	Nil.
3	30	8·160	1·900	7·400	1·850	18·440	962·250	,,
4	60	23·765	4·800	10·550	1·250	5·635	954·000	,,
5	70	9·200	3·200	12·100	2·060	14·400	960·040	,,
6	50	7·920	1·060	9·000	1·100	2·420	978·500	,,
7	60	6·720	1·100	4·800	4·200	22·000	961·060	,,
8	60	7·250	2·015	7·100	1·763	15·412	966·460	,,
9	40	14·000	1·400	8·000	2·200	2·800	971·600	,,
10	26	7·325	1·520	6·935	1·964	11·214	971·042	,,
11	25	9·576	3·042	9·421	2·110	13·472	962·379	,,
12	16	18·201	2·740	3·114	2·054	8·217	960,674	,,
13	40	5·621	1·132	7·420	1·943	10·532	973·352	,,
14	32	23·900	1·520	3·500	1·400	14·600	954·330	,,
15	40	20·865	4·000	9·963	·962	7·221	956·989	,,
16	30	4·887	1·800	8·804	1·862	13·264	969·383	,,
	651	174·620	38.029	126·567	30·818	202·497	15423·469	4·000
Average	40·687	10·913	2·376	7·910	1·926	12·656	963·966	·250

Note.—In estimating the quantity of urinary solids the following method was adopted:—A thousand grains of urine were evaporated in a previously weighed capsule over a sand-bath, until no further weight was lost. The capsule and its contents were then placed over strong sulphuric acid, under the exhausted receiver of an air-pump, until the last drops of water had evaporated. Their aggregate weight was next noted immediately on their removal from the air-pump, and the subsequent deduction of the weight of the capsule alone showed at once that of the solid residue of the 1000 grains of urine.

The following table shows the average quantity of urinary constituents excreted in twenty-four hours for the above table:—

Urea.	Uric Acid and Vesical Mucus	Fixed Alkaline Salts.	Phosphate of Lime and Magnesia.	Animal Extractives and Ammoniacal Salts.
grs.	grs.	grs.	grs.	grs
208·949	45·493	151·451	36·876	242·322

If we compare the average of the above table with that of the following analyses of healthy urine by Simon, Berzelius, Marchand, Lehmann, and Miller, we arrive at the following important conclusions:—

	Simon.	Berzelius.	Marchand.	Lehmann.	Miller.	Average
	1000 grs.	1000 grs.	1000 grs	1000 grs.	1000 grs.	1000 grs.
Water, ...	956·000	933·000	933·199	937·932	956·9000	943·326
Urea, ...	14·578	30·10	32·675	31·450	14·2300	24·606
Uric Acid, ...	·710	1·00	1·065	1·021	·3700	·833
Fixed Alk. Salts, . .	13·77	15·29	14·074	...	13·158	14·073
Phos. of Lime & Mag.	·654	1·00	1·210	1·132	...	·999
Animal Ex. & Am. Salts,	12·940	...	14·355	...	15·0474	14·114
Silica, ...	trace.	0·3	...	...	...	...

1. That in the stage of commencing germination of pulmonary tubercle, the amount of urea excreted by the kidneys is, for equal quantities of urine, diminished by more than one half as compared with that thrown off during health. According, however, to Dr Golding Bird, the average quantity of urea excreted, in twenty-four hours, during health, is 270 grains; whilst Dr Becquerel fixes it at 255 grains. By adopting either of these averages, there is, nevertheless, on comparison, a *manifest diminution* in the quantity excreted during the germination of pulmonary tubercle.

2. That, under the above-mentioned circumstances, there is a sensible increase of uric acid in the urine.[1]

3. That the fixed alkaline salts are considerably less than those of healthy urine.

4. That the amount of phosphate of lime and magnesia is almost twice that of healthy urine.

5. That there is slight diminution in the amount of extractive matters.

These facts, which have been ascertained by the most careful and rigid analysis of the urine, harmonize with our knowledge of the physiology of urea and of the influence which inflammatory diseases of the lungs exercise on the amount of the salts of the urine. It is well known that a deficient exercise and a diminished quantity of food produce an immediate and sensible effect in reducing the amount of urea excreted by the kidneys. Both these conditions are almost invariably present in the stage of commencing germination of pulmonary tubercle, and to these circumstances must, in part, be attributed the manifest diminution of urea observed in such cases. But the restricted respiration occasioned by disease, evidently produces, irrespective of the influence of exercise and diet, a diminution in the amount of urea; for we shall hereafter find that, in the

[1] The uric acid and vesical mucus were calculated together; but it was nevertheless evident that the joint increase of these two constituents over those of healthy urine, was chiefly owing to uric acid.

more advanced cases of pulmonary tuberculosis, in which excavations exist in the lungs, and in which the tolerance of exercise and the appetite for food are not unfrequently equal to those of an earlier stage, whilst the vital capacity of the lungs is nevertheless undergoing a gradual reduction by the steady progress of the disease, the average quantity of urea, as compared with that of the stage of commencing germination, is decidedly less. Whatever share, however, these three conditions—*i.e.*, deficient exercise, diet, and restricted respiration—may, as it were, *separately* exert on the amount of urea, it is certain that, *conjointly*, they produce a manifest diminution in the quantity excreted by the kidneys.

As regards the reduction in the proportion of the alkaline salts of the urine, this fact supports, to a certain extent, the statement already advanced and confirmed by several authors, namely, that during the exudative stage of pneumonia, there is a total absence, or nearly so, of the chlorides from the urine. It is true that, in pulmonary tuberculosis, such absence never wholly takes place; nevertheless the marked diminution which is observed, not only in the proportion of chlorides, but also of the other alkaline constituents of the urine, manifestly shows that pulmonary disease exerts a wonderful power of modifying, in this respect, the eliminating action of the kidneys. As in the commencement of the germination of pulmonary tubercle the saline constituents of the urine are never reduced to the same extent as in inflammatory affections of the lungs, so by reference to the tables of the respective sputa, it will be found that they never exist in that increased proportion in the sputa of the former as in those of the latter diseases. If the view previously expressed, as to the measure of the activity of pulmonary disease by the proportion of alkaline salts in the urine, be applied, in a negative manner, to that of the alkaline salts of the urine, to indicate the same thing with respect to the lungs, we find that the greater proportion of alkaline salts in the urine of pulmonary tuberculosis assigns less activity to this affection than to the inflammatory diseases of the bronchio-pulmonary membrane, and therefore places it in precisely the same position, in relation to these diseases, as was assigned to it by a comparison of their respective sputa. The former method may be termed the *negative*, and the latter the *positive*, measure of the activity of pulmonary disease. Both should be employed, and from their use an improved knowledge of pulmonary disease might be anticipated.

Stage of Advanced Germination.

When from the minute ulcerations of the bronchio-pulmonary membrane, which characterise the early stage of germination, the disease proceeds to the further destruction of the pulmonary tissue, so that excavations now exist in the lungs, the stage of advanced

germination has been attained. In progressing to this extent the growth of the tuberculous matter, fed by an increased exudation from the neighbouring capillaries, is locally the real pathological cause. Hence, as the growth of one mass of tubercle and its subsequent removal take place, a fresh quantity of tuberculous matter is deposited in the adjoining pulmonary tissue, which, by and by, germinates and is subsequently cast off; whilst fresh exudation again occurs in the tissue of the lungs beyond it. In this way the gradual destruction of those organs progresses until, by the encroachment on their function, life can no longer be maintained. When treating of the stage of deposition, it was shown, that the disease did not observe an equal progress throughout the pulmonary tissue invaded—that, in one place, total occlusion of the pulmonary cells, by tuberculous deposit, took place; whilst, in another, the basement membrane of their mucous lining was no more than thickened. It is equally true that, in the advanced stage of germination, the same inequality of progress is observed—that the more completely consolidated portions of lung have first become the seat of caverns; whilst the less affected parts have only acquired that degree of consolidation, which precedes their complete disintegration. To comprehend the mode in which a mass, about to undergo complete disintegration, is broken up and subsequently removed from the lungs, it must be remembered that such a mass consists of a multitude of pulmonary vesicles, each of which is filled with tuberculous matter, and between which the walls of such vesicles form so many septa or partitions, giving lodgment to the pulmonary capillaries, from which the original exudation took place, and which now, according to the degree of their capability to transmit blood, convey to it the materials necessary for its germination and subsequent removal. Such masses, therefore, may be regarded as an aggregation of smaller masses, of one of which latter a single vesicle, studded with tuberculous exudation, is the correct representative. The aggregate masses vary in size according to the number of pulmonary vesicles of which they are composed. When small, they may not consist of all the vesicles which constitute a single pulmonary lobule; when large, they may, on the other hand, involve many lobules. In the breaking up of such aggregated masses, each lobule of such masses, and each vesicle of such lobules, provided their respective capillaries are able to transmit blood, become so many centres of action, in which the whole mass is, as it were, simultaneously engaged. If, however, the pressure of the tuberculous exudation prevents the transmission of blood through the capillaries of any portion of such mass, then such portion receives its proper stimulus to germinate by a species of imbibition of the fluid thrown out by the nearest vessels which are patent. In almost every large tuberculous mass this particular condition exists, the effect of which is, to retard the germination of those portions of tubercle which are the farthest distant from those parts of the mass in which the circulation is yet maintained. Hence the

reason why, in a tubercular mass of any size which is in process of "softening," a uniform consistence is not always observed—one part of the mass having become diffluent, whilst another portion of it has but as yet acquired a lardaceous consistence. As this unequal action progresses, and as the cells resulting from the growth of the tuberculous exudation, together with the debris of the broken down tissues involved in such exudation, are expelled through the minute bronchi which terminate in such masses, and which are themselves implicated in the disease, caverns of small but variable size occur. These are, in masses of the above size, at first divided by septa of diseased tissue, which are undergoing the same process of disintegration, and which, continuing to disintegrate at their cavernal surfaces, are ultimately destroyed. In this way the small caverns in a large tubercular mass ultimately become one. It often happens that those portions of the septa, which are the farthest removed from the surface of the lung, are the first destroyed, so that when an inspection is made after death, there may be, at first sight, no apparent destruction of the pulmonary tissue, only a tubercular consolidation, which however, when incised, reveals in its interior excavations which communicate one with another. In small masses of tubercle the same process of cell-growth and disintegration is observed—each pulmonary vesicle becoming a centre of action so far in point of activity, and so far only, as its capillary circulation is maintained; whilst the pulmonary tissue around such germinating masses is itself receiving a deposit of tuberculous matter, which, as the caverns extend beyond the limits of the original deposit now in the process of growth, will undergo the same germination and subsequent removal. Thus, by the lateral extension of caverns, formed by the germination and disintegration of separate and isolated tubercular masses, a large excavation is produced. Life, however, frequently fails before such conjunction of caverns can take place. It is, therefore, very common to find after death, several caverns of variable size separated by intervening portions of pulmonary tissue more or less consolidated by tuberculous exudation. The form of "softening" here described, is more particularly observed in the more chronic cases of pulmonary tubercle, in which the manifestation and progress of both the constitutional symptoms and local disease have been slow. If, in such cases, the tuberculous deposit is of limited extent, a cavern may cease to extend, or it may even contract and gradually close, provided an improved condition of the blood occurs, and any further deposit of tubercle ceases to take place in the neighbourhood of such cavern. When, for the above reasons, a cavern ceases to extend, its internal surface may become covered with a layer of plastic exudation, which is subsequently organised into an adventitious membrane, which forms a perfect lining for the cavity, and which, on microscopic examination, is found, in the generality of cases, to consist of a fine fibrous tissue, presenting little or no trace of epithelium. With this formation there seems to be a limit placed

to the extension of the cavern on the one hand, and to its attempt at closure on the other. From the pouch-like form which such caverns frequently present, and from the mode in which they communicate with the bronchi, they might readily be mistaken for dilatations of the latter tubes.

When caverns do not become thus lined by an adventitious membrane, and when, nevertheless, a curative tendency is observed in the disease, successive layers of plastic exudation are thrown out on the inner surface of the walls of the cavern, which become organised, contract, and ultimately obliterate it entirely; whilst a dense fibrous cicatrix, involving a retraction of the pleural surface, is left to mark the previous site of the excavation. By the increasing approximation of the sides of the cavern, a degree of tension is made on the surrounding pulmonary tissue, the cells of which, owing to the complete or partial paralysis of their fibres produced by virtue of such tension, and, sometimes, by implication of such cells themselves in the disease, gradually yield to the distending force of the inspiratory current, enlarge, and thus constitute one form of emphysema.

As masses of tubercle germinate and break down in the manner now described, a communication is established with one or more bronchi, through which the effete and disintegrated matters are, from time to time, expelled. It, however, sometimes happens that the bronchi leading to such masses, are themselves completely obliterated by tuberculous exudation, in which case the successive portions of diffluent matter, formed by the disintegration of those masses, are retained. In this way a vomica is formed, which by pressure ultimately bursts into a neighbouring tube either by forcing the plug of exudation which closes it, or by causing absorption and ulceration of its coats. This event gives rise to a sudden discharge of purulent fluid, which not unfrequently amounts to several ounces.

Another mode in which tubercular masses are removed from the lung, is by the absorption of the organic portion and by the consequent formation of cretaceous masses. The necessary condition for this occurrence appears to be, that there shall be no patent bronchus communicating with the mass. This condition obtained, it would seem that, in consequence of an improved condition of the blood, a healthier exudation takes place in the immediate circumference of the tubercular mass, which, becoming organised, encloses the latter in a cyst, that subsequently a gradual transudation of the watery constituents of the blood effects the disintegration of the organic portion of the tuberculous exudation, and that, during such disintegration, its constituent salts of lime are thrown down, whilst the soluble salts and organic matter are reabsorbed and removed from the system. Whether the removal of the organic matter is preceded by its germination and growth into cells, or whether it results from an absolute loss of vitality occasioned by its complete isolation caused by the formation of a cyst around it, and by a consequent separation into its ultimate elements and their re-combination in other forms,

we have no positive data to show. In either case the same ultimate results would occur—the products of the decomposition of the organic matter, whether the latter is considered simply as having, or not having, germinated and formed cells, being the same. As by one or the other of these modes, the organic matter is probably removed, a corresponding retraction of the walls of the cyst takes place, which, when it has occurred to the full extent on the removal of the whole of the organic matter, constitutes puckerings similar to those observed after the healing of a cavern. It must not, therefore, be inferred, from the existence of cretaceous masses in the lungs, that they have been preceded by caverns having communication with the bronchi. They, nevertheless, indicate the previous existence of an organic deposit, of its subsequent removal, and of a certain vacuity left by its removal; whilst they undoubtedly seem to show, that the site of such exudation had no direct communication with the external air; for had such been the case, the continual emptying of the cavern would inevitably have prevented the gradual accumulation and formation of cretaceous masses. These bodies are, nevertheless, sometimes found in caverns having a direct communication, by means of one or more bronchi, with the external air; but, in all such cases, it is pretty certain, that the communication was established at a time posterior to the removal of the organic matter and the formation of cretaceous masses. In point of size, these bodies vary from that of a hemp-seed to that of a small horse-bean. They are generally of somewhat irregular figure, slightly rough on their surface, of a pale yellow colour externally, white and friable within, without exhibiting any trace whatever of laminæ. They give out an animal odour when heated, and effervesce with the mineral acids, thus showing the presence of a carbonate. The following was the composition of a cretaceous mass, which was taken from a cyst in the apex of the lung of a patient:—

Weight of calculus, 6 grains.

Organic matter, ...	·75
Cholesterine, ...	·40
Salts of lime, ...	4·12
Water, ...	·73
	6·00

Thus far the formation of caverns has been traced in the most chronic cases of pulmonary tubercle, in which distinct and isolated masses occupy the lungs. There are, however, many cases in which it has already been shown that the course of this disease is intermediate in point of severity and duration, between that already described and the acute form of the disease. In such cases the formation of caverns is not preceded by that perfect and limited consolidation, which obtains in the most chronic form of the disease. There is, however, a greater extent of disease; but it does not lead

to that amount of occlusion of the pulmonary vesicles as is observed in the form previously described. There are, nevertheless, considerable thickening of the cell-walls and complete closure of many cells by tuberculous deposit, so that the physical signs are materially altered, and at once arrest the attention of the physician; but notwithstanding this fact, the vesicular murmur, although inaudible during natural respiration, is distinctly heard during a forced inspiration where the pulmonary vesicles are not closed by deposit, and is then accompanied by a moist crepitant or subcrepitant rhonchus, whilst mucous rhonchi are more or less present in the bronchial tubes. In such cases it is demonstrated by successful injections of the pulmonary tissue, that the capillary circulation through the affected parts is, to a very great extent, capable of being carried on, that there is not that degree of isolation of the tuberculous deposit from the capillary circulation as occurs in the form already described, and that the pulmonary tissue is less consolidated than in the latter case. This condition, therefore, of the capillaries of the affected parts permits, as it were, every pulmonary cell to furnish the necessary means of growth to the tuberculous exudation which it contains, so that almost every individual cell may be regarded as an independent centre of action. As, however, all cells are not equally affected with tuberculous deposit, the rapidity of growth is likewise unequal amongst them. Hence, some cells have simply received an addition to the thickness of their walls, and are as yet engaged in an increased secretion from their epithelial surface; whilst others have shed their epithelium, and have lost by ulceration a portion of their basement structure. By the extension of this ulcerative action on the opposite surfaces of a cell-wall, the latter is shortly destroyed, when two cells thus become one. In the adjoining cells the same kind of action is going on—the same destruction of cell-walls is taking place, so that, by and by, not only two, three, or four cells, but many are broken into each other, and thus constitute a distinct cavern. This extends by the same growth and disintegration of tubercle in the adjoining cells, the lining membrane of which has now assumed an ulcerative action, which shortly effects the complete destruction of the cell-walls. In this way a considerable portion of the lobe of a lung is not long in undergoing complete disintegration.

In the acute form of pulmonary tuberculosis the formation of caverns is precisely the same as now described. The exudation, however, is of much greater superficial extent, involving simultaneously, or nearly so, the whole, or the greater part of the bronchio-pulmonary membrane of one or both lungs. In both the upper, middle, and lower lobes of the lung some lobules receive a greater amount of exudation than others. These consequently more quickly break down, and give rise to caverns of small but variable size throughout the lung. As a general rule, the formation of caverns in such cases takes place more early in the upper than in the lower lobes, but many instances occur in which this priority is not observed.

An inspection of the lungs at this period reveals caverns of various sizes in the interior of their substance, some of which communicate with the neighbouring bronchi; whilst others have as yet formed no such communication, and, therefore, constitute so many vomicæ. The interior of the minute bronchi, through which the cavities have discharged their contents, is of much deeper coloration than natural, more or less destitute of epithelium, and presents numerous irregular ulcerations of the basement structure, the surfaces of which are covered with exudation in the process of growth. This increased coloration of the minute tubes is likewise continued into the larger bronchi, in which, however, it is less vivid and less general in its superficial extent, with a partial desquamation of the epithelium of these tubes. Again, the lining membrane of the larynx and trachea is often found to be involved, and to present, in addition to hyperhæmia, epithelial and basement ulcerations, which, when situated upon the vocal chords, or in their immediate neighbourhood, produce the hoarseness so commonly present in all forms of pulmonary tuberculosis. Not unfrequently, too, the pleuræ have engaged in the disease, and present thickenings and partial adhesions formed by layers of lymph, which manifest their deficient vitality by their friability, and by the generally low degree of organisation which they present under the microscope. The appearances now described, offer a ready explanation of those compound physical signs of bronchitis, pneumonia, and pleurisy which are so frequently observed during the course of acute pulmonary tuberculosis.

The character of the sputum varies in different cases of the advanced stage of germination. It, however, admits of classification into—

1. The muco-purulent.
2. The purulent.

The former, in its physical appearance to the naked eye, consists of a thick, tenacious fluid, of irregular consistence, and of a light drab, white, yellowish-white, or slightly greenish-white colour. It is often interspersed with the semi-solid, cheesy-looking portions before described, which exist in variable numbers, and in size from that of a mere speck to that of a pin's head. Blood may likewise be present in variable quantity.

Microscopically examined, the fluid portion is seen to consist of mucous and pus-cells, plastic-cells, exudation masses and cells, epithelial and basement patches, bronchial casts, minute granular matter, and amorphous particles of the salts of lime, with, occasionally, one or other of the accidental constituents before-named; whilst the cheesy-looking portions are composed of the microscopic objects before described.

The persistence of epithelial and basement patches in this kind of sputum is a certain indication that the disease is progressing to a farther stage in its superficial extent; whilst the numbers and size of the exudation and other cells are a measure of the activity of the

disease. Hence, an examination of the sputum from time to time can better determine the real progress of the disease than the most delicate auscultatory examination. When, however, the two modes of examination are combined, which ought always to be the case, the diagnosis is rendered certain; whilst the prognosis, which is the legitimate deduction of knowledge obtained by such a combination, is far more likely to be realised than that which is based on auscultation or microscopic examination alone.

The purulent sputum, which, however, does not consist entirely of pus-cells, appears, in one form, in separate, isolated, nummular masses of a thick, creamy consistence, and of a yellowish-white, or faintly greenish-white colour. It is sufficiently tenacious to maintain, even on inversion of the vessel which contains it, the flattened and isolated form which it assumes immediately on being expectorated. Minute specks of cheesy-looking portions are occasionally entangled in its masses, which masses, on microscopic examination, are found to consist essentially of pus-cells, mingled with plastic and a few exudation-cells, and not unfrequently with epithelial and basement patches, together with here and there a bronchial cast. Such masses are common to the sputum during the existence of caverns in the lungs, but they are not diagnostic of caverns, inasmuch as they may likewise occur when epithelial and basement ulcerations of the bronchio-pulmonary membrane alone exist. The legitimate deduction, therefore, to be drawn from their presence in the sputum is, that there is *certain breach of structure* of some portion of the bronchio-pulmonary membrane, and that an excavation in the pulmonary tissue may likewise exist.

Another form of purulent sputum consists of a thick, creamy fluid, of a drab, yellowish-white, or greenish-white colour, of tolerably regular consistence, and frequently holding in suspension minute portions of the cheesy-looking substance already described. It is non-aërated, and but slightly viscid and tenacious, so that, on inclining the vessel which contains it, it quits the latter as a dense, heavy fluid, leaving but little adherent to the sides. On standing, it gradually separates into two portions—a lower, which is thick, dense, heavy, and of a dirty drab colour; and an upper portion, which is a thin, serous-looking fluid. The former consists essentially of pus-cells, and the minute cheesy portions before named; whilst the latter holds in suspension mucous corpuscles, epithelial and basement patches. Sometimes the pus-cells are of very diminutive growth, not measuring more than $\frac{1}{4000}$ths of an inch in diameter; at other times they are of full size, and are as perfectly developed as those arising from the most vitalised exudation. Sputum of this kind is diagnostic of the existence of a cavern when the *lungs alone* are the seat of disease. Its pus-cells are chiefly formed by the growth and subsequent degeneration of the tuberculous exudation existing in the walls of such caverns; and it derives its other structures from the diseased mucous surface over which

it has to pass in its expulsion from the lungs.—(Figs. 25 and 26, Plate I.)

The following table shows the composition of the sputum in fifteen cases of pulmonary tuberculosis, in which excavations existed in the lungs :—

IN 100 PARTS.

No.	Organic Matter.	Chlorides of Sodium and Potassium, Alkaline Sulphates and Phosphates.	Salts of Lime.	Water.
1	2·72	·25	·04	96·99
2	8·16	·50	·34	91·00
3	8·51	·96	·11	90·42
4	7·36	·95	·23	91·46
5	3·67	·45	·27	95·61
6	7·91	·55	·13	91·41
7	2·87	·35	·07	96·71
8	5·14	·43	·11	94·32
9	5·11	·56	·17	94·16
10	4·11	·78	·27	94·84
11	10·65	·90	·45	88·00
12	8·65	·48	·37	90·50
13	2·30	·35	·30	97·05
14	4·83	1·05	·35	93·77
15	5·07	·65	·23	94·05
	87·06	9·21	3·44	1400·29
Average	5·80	·61	·22	93·35

On comparing the above table with that of the composition of the sputum in the early stage of the germination of pulmonary tubercle, we find in the former a relative increase in the amount of organic matter, but a slight relative decrease in the quantity of alkaline and earthy salts. These facts, whilst they seem to confirm the views already expressed, in reference to the relative ratio of the alkaline salts to the degree and activity of cell-growth, at the same time indicate, by the excess of organic matter, the greater destruction of lung-tissue in the advanced than in the early stage of the germination of pulmonary tubercle. Viewed in comparison with the sputa of the inflammatory affections of the bronchio-pulmonary membrane, the above table assigns the same position, in the category of pulmonary diseases, to tuberculosis of those organs as was assigned to it by the method above named.

Reference to the following table will further show the constitution of the urine, at this period, in fifteen cases, the numbers of which correspond with those in Table II. of the sputum :—

TABLE II. OF URINE.

No.	Quantity in 24 hours.	Urea in	Uric Acid and Vesical Mucus in	Fixed Alkaline Salts in	Phos. of Lime and Mag. nesia in	Animal Extrac. tives and Am. Salts in	Water in	Albu. men in
	Fluid oz.	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000 grs.	1000grs.
1	30	3·100	1·980	5·900	1·000	13·960	970·060	4·000
2	22	6·000	1·040	6·020	1·020	25·440	960·480	,,
3	50	3·320	1·260	4·500	2·240	27·600	961·080	,,
4	60	12·140	1·500	5·600	1·100	10·780	966·740	2·140
5	80	5·760	1·100	10·500	·640	6·580	975·420	,,
6	80	3·120	1·440	5·660	1·620	17·600	970·560	,,
7	90	8·600	1·000	3·760	1·940	16·700	968·000	,,
8	60	20·160	4·200	6·400	2·900	5·440	960·900	,,
9	70	16·320	3·200	2·900	2·300	4·500	962·280	8·500
10	50	7·100	·700	7·800	2·200	18·380	963·620	,,
11	38	2·300	·300	6·800	2·300	17·800	970·200	,,
12	40	2·560	2·140	8·600	1·000	16·880	968·820	,,
13	30	1·500	·630	3·300	2·600	19·220	972·200	,,
14	30	12·480	3·460	8·800	2·100	23·040	950·120	,,
15	28	12·280	1·700	11·500	4·500	2·020	968·000	,,
	758	116·740	26·000	98·540	29·460	226·140	14488·220	14·640
Average	50·53	7·782	1·733	6·569	1·964	15·076	965·881	·976

According to the above table, the average quantities of urinary constituents excreted by the kidneys in twenty-four hours, were the following :—

Urea.	Uric Acid and Vesical Mucus.	Fixed Alkaline Salts.	Phosphate of Lime and Magnesia.	Animal Extractives and Ammoniacal Salts.
grs. 179·606	grs. 39·938	grs. 151·670	grs. 45·248	grs. 347·900

It will be seen from a comparison of the above table with that showing the average of Table I. of the urine, that the advanced stage of pulmonary tuberculosis is accompanied by a further reduction in the quantity of urea, that there is also a relative diminution in the quantity of uric acid, which is, nevertheless, somewhat considerably above the healthy average—and that the ratio of the salts of lime is again considerably increased. A further comparison likewise shows another important fact, namely, that the liability to albuminuria is in the proportion of 1 to 7·75 of all cases taken indiscriminately after the commencement of the germination of pulmonary tubercle, and of 3 in the advanced, to 1 in the early part of this stage.

Nature and Special Pathology of Pulmonary Tubercle.

In the preceding sections, the progress of pulmonary tubercle has been traced from its first deposition in the pulmonary organs, to its final breaking up and removal from the system. It has thus been shown that the phases through which it has to pass, differ, in no respect, from those attendant on the deposition and subsequent removal of a simple inflammatory exudation. The same localization, the same process of cell-growth, and the same method of removal, characterise the two exudations. The only *physical* difference observed, is in the relative intensity of the localising causes, the amount of exudation, and the degree of the rapidity of its growth. In this respect, the inflammatory affections of the bronchio-pulmonary membrane are, as a general rule, more intense, and the exudation to which they give rise, is of more rapid growth than in pulmonary tuberculosis; whilst the amount of deposit, considered in reference to the relative extent of lung which is affected, is generally greater in the latter than in the former diseases. To those pathologists who maintain the existence of a diagnostic "tubercle-corpuscle," the above-named physical distinctions between the inflammatory and tuberculous affections of the bronchio-pulmonary membrane, will not be sufficient. Let it, however, be shown on what tangible ground this objection (*i.e.*, the existence of "tubercle-corpuscles") rests.

If we take a portion of grey, semi-transparent tubercle, press it between two slips of glass, and, after having added a drop of distilled water, submit it to a power of 250 or 300 diameters linear, we find it to consist of a multitude of molecules and granules, often interspersed with granular cells,[1] usually of irregular shape, and varying in size from $\frac{1}{4000}$th to $\frac{1}{2500}$th of an inch in their longest diameter. With these bodies are associated others having no such granular appearance, which are perfectly round, measure from $\frac{1}{8000}$th to $\frac{1}{4000}$th of an inch in diameter, and which appear to be cells in an early stage of growth. To these may be added the occasional presence of epithelial patches and a few elongated masses of coagulated fibrine. The whole is more or less held together by a semi-transparent, connecting medium, which forms the undefined, unorganized portion of the exudation. On adding a drop of acetic acid, the molecular matter is either in part dissolved, or at least rendered more transparent; whilst the cell-structures also undergo the latter change; but are otherwise unaffected. Liquor potassa dissolves the cell-structures, and, at the same time, causes the molecular and granular matter almost entirely to disappear; whilst ether and alcohol exert but a slight effect in rendering the whole permanently lighter.

[1] In the mere thickening of the basement membrane, the tuberculous deposit presents no trace of cell-growth. This condition is only observed after germination.

If, in like manner, we examine a portion of yellow tubercle from the pulmonary cells, precisely similar structures are observed; but the degree of cell-growth is somewhat advanced. If, again, we test, by the same method, a portion of tubercle which has assumed more or less of a creamy consistence, the same structures are still found; the only difference being, that the granular cells may approach somewhat nearer, in their appearance, to what is called a pus-cell; whilst most of the non-granular cells have reached a further development, and constitute cells more or less round or oval, with well-defined margins, and varying in size from $\frac{1}{3000}$th to $\frac{1}{1500}$th of an inch in diameter. What now is the characteristic of an inflammatory exudation on the bronchio-pulmonary membrane, as deduced from a microscopic examination of the sputa? The answer is, that in addition to epithelial patches, and numerous well-developed mucus-corpuscles, the sputa contain granular and non-granular cells, molecules and granules, together with masses of exudation, which exhibit cells in different degrees of growth, and which are held together by the as yet unorganised portion of the exudation. With these are likewise associated elongated portions of coagulated fibrine, forming bronchial casts. Hence, there is no difference whatever between a tuberculous and a simple inflammatory exudation, so far as the variety of cell-structures is concerned. There is, however, at the present stage of the comparison, a difference between the tuberculous and non-tuberculous granular cell. But this difference refers only to the granular cell of a sthenic inflammatory exudation, which is round, whilst that of a tuberculous exudation is said to be, as a general rule, of irregular shape. In many instances, such is undoubtedly the case; but in others of manifestly tuberculous origin, the cells are as perfectly formed, and as round as in the most sthenic inflammatory exudation; whilst in exudations of an asthenic inflammatory character, they are frequently as irregular in shape, as those which are present in tubercle.

The comparison, therefore, plainly shows that there is *no diagnostic cell* of tubercle. It were, consequently, a misnomer to designate any cell by the epithet, "tubercle-corpuscle." When, however, an exudation consists essentially of irregularly shaped cells, mingled with numerous molecules and granules,[1] we may safely pronounce it to be of deficient vitality; but collateral circumstances must assist in determining the question of its tuberculous or non-tuberculous origin. It may or may not be tuberculous in its nature; the *microscope alone* cannot determine the question; a full investigation of the case, in all its bearings, is absolutely necessary for a correct interpretation of the physical appearances of the exudation.

Whilst the above facts negative the term, "tubercle-corpuscle," the microscopy of pulmonary tubercle, in every phasis in which it

[1] A large proportion of molecular and granular matter in an exudation is a tolerably good indication of its tuberculous nature.

is known to exist, demonstrates that the epithets, grey, yellow, hard, soft, encysted, scattered, infiltrated, as applied to tubercle, are but indicative of the age, extent, and particular condition of localization of the exudation, not of any variety of structure. Tubercle, whether it exists in the form of hard, grey, semi-transparent, miliary granulations, or in crude, yellow masses, or in the more diffused form of infiltration of the bronchio-pulmonary membrane, is, as has been already observed, one and the same in structure. Whatever difference exists between it and an inflammatory exudation, is essentially one of inherent vitality. Experience teaches that this difference of vitality may be either *acquired*, or the result of *hereditary transmission*. In the latter case, every tissue of the body must, from its first formation, be imbued with this hereditary taint; but examined in the ordinary process of nutrition, there is no absolute difference in structure between it and the tissues of a body not affected by hereditary predisposition. Thus the fibre of a muscle, of a nerve, or the parenchyma of any internal organ, is one and the same, in point of physical organisation, in both those predisposed and those not predisposed to tubercle. In the former, the tissues are built up and maintained, in a relative ratio of waste and repair, by a blastema imbued with this hereditary taint, precisely in the same way as they are, in the latter, by a blastema which reaches the standard of perfect health. In both cases, so long as the general balance of function is maintained between the different organs, there is no deviation in the nutrition of the body—the health is one and uniform throughout. But if, by any external or other circumstance, the balance of function is destroyed in either case, a local predisposition may be induced, and a local exudation, on a continuance of this predisposition, is certain to follow. This exudation is, in the one case, endowed with average vitality; in the other, it is, in this respect, deficient in proportion to the hereditary taint. When such taint exists in the hereditary constitution of the system, it does so, in a variety of systems, in such multitudinous shades of depth, as to account for the oft observed fact, that of two families predisposed to tuberculosis, and existing under similar conditions—with respect to food, climate, and other circumstances—the one family loses few, if any, whilst the other loses nearly all its members. The hereditary taint, once implanted in the system, can never afterwards be effaced, although it may exist in such minute proportion, as to offer no impediment to the growth and general nutrition of the body. Yet every particle of that body is imbued, each in its proportionate degree, with the hereditary taint, which is propagated from old to new particles of the system in the ratio of the removal of the tissues, precisely in the same way as the hereditary cause of insanity, cancer, and gout, is maintained in the body. Were this not the case, it would be impossible to transmit the hereditary predisposition to tubercle from parent to offspring. That it is transmitted, the concurrent testimony of all authors on the

subject testifies; and yet both microscopical and chemical research has failed to detect any difference of structure or composition between the germ which transmits, and that which does not transmit this hereditary taint to the offspring. The same truism holds good with respect to the structures of a body predisposed, and of one not thus predisposed, to tubercle. Chemistry, in the hands of various pathologists, has arrived at some results, as to the composition of the blood in tuberculous subjects; but these results are themselves so far contradictory of each other, and, at most, show so slight a deviation from the average healthy constitution of that fluid, as to afford no tangible or trustworthy data for legitimate deductions. The great weight of authority, therefore, is clearly in favour of a *vital* rather than of a *structural* or *chemical* peculiarity of tubercle. When this peculiarity of vitality is not of hereditary transmission, it is readily acquired by a persistence of those conditions, which, whatever may be their exciting causes, induce a deficient, depraved, and faulty nutrition of the body generally. Amongst these conditions, a faulty state of the digestive organs is pre-eminent; and although this condition exists in every *acquired* case of pulmonary tuberculosis, yet it is not to be regarded as the *fons et origo mali*. On the contrary, it is but the effect of a cause, which latter may resolve itself into depression of the nervous system, and the consequent lack of that supply of nervous energy to the different organs, which is essential for the due performance of their functions. Again, this depression of the nervous system is but the effect of a cause, which may receive its appropriate expression in an over-action of the brain and nerves, in an impoverishment of the blood, with respect to the elements of nutrition, or in a depraved condition of that fluid, arising from a partial action only of any one or more functions of the body. Nevertheless, in the *acquired* predisposition to tubercle, the most tangible cause is manifestly referrible to disorder of those organs which are engaged in the work of primary digestion. In, perhaps, no single instance, could this acquired predisposition to tubercle, take place without its being preceded by an aberration of function in the digestive apparatus. Hence, this aberration is an *essential* precedent to the *acquired* tuberculous predisposition, which corresponds to the term, "tuberculous cachexia" of Sir James Clarke. In the hereditary tuberculous predisposition, however, disorder of the digestive organs, although it not unfrequently precedes and accompanies the localization of tubercle, is not an essential precedent to such an occurrence. Hence, a group of symptoms, known by the epithet "dyspepsia," is not to be expected, as a *sine quâ non* to tuberculous deposits, in those who inherit the hereditary predisposition. The localization of tubercle in such cases, takes place under similar conditions to those which attend the localization of simple inflammatory exudations, and the disordered digestive function is generally but the counterpart of that which is observed in sudden and severe inflammations of internal organs. If

dyspepsia precedes the localization of tubercle, in any case of hereditary predisposition, it is rather to be regarded as the *effect* than as the *cause* of the deficient vitality of the blood-plasma. It may, in such cases, add to the intensity of the hereditary predisposition, but it does not create it. Where the tuberculous predisposition is inherited from birth, all exudations are, in their inherent vitality, tuberculous in proportion to the constitutional taint; and they are capable of passing through all the phases of organization and growth, which are observed in exudations of a healthy character. But, owing to the comparatively low degree of vitality with which they are endowed, they cannot sustain that "wear and tear" of which tissues formed from healthy vitalised plasma, are capable. Hence, a probable explanation of that physical and constitutional delicacy, which characterises the tuberculously predisposed.

Thus much may be said of the *acquired constitutional predisposition* to tubercle; but it must here be borne in mind, that such predisposition cannot have been fully and constitutionally acquired, until every tissue of the body has, in the process of waste and repair, been changed for those resulting from a less vitalised plasma. Then, indeed, the acquired tubercular predisposition becomes a constitutional peculiarity, which subsequently influences all exudations in the manner before described, and which is thenceforth transmissible from parent to offspring. In the transition-stage from that of perfect health to the acquired constitutional predisposition to tubercle, each fresh formation of blood is productive of an influx into the general current of the circulation, of a pabulum of nutrition, which is defective in its inherent vitality, and which first replaces blood of healthy vitality drawn off by the tissues, and then furnishes to those structures the elements of nutrition, by which the tissues themselves ultimately acquire an organisation defective in vitality. If, during this transition, a local exudation takes place, it is tuberculous in proportion to the deterioration in the quality of the blood. That fluid may as yet have received but a little addition of fresh blood defective in vitality, and hence the exudation approaches, in this respect, the character of healthy exudation; or the blood may have been so far changed, that three-fourths of it are already of defective vitality, in which case the exudation will observe a corresponding deflection from the standard of perfect health. When the whole mass of healthy blood has been replaced by blood of defective vitality, a local exudation becomes *entirely* and *absolutely* tuberculous in its character, although the structures in which such exudation takes place, may be composed of materials originally of perfect vitality. This is the case, when, after the deterioration of the whole mass of blood, there has not, as yet, been sufficient time for the ordinary molecular waste and repair of the tissues to effect the removal of the properly vitalised structures, and their replacement by others formed from the present imperfectly vitalised blood. As soon as this change has been perfected throughout the system,

the constitutional predisposition to the formation of tubercle has been acquired; but until the system has undergone this change, the formation of tubercle is of casual occurrence, and is as capricious as that of the digestive functions, on which it at present depends. So long, therefore, as tubercle is absolutely and entirely dependent on aberration of the digestive functions, it is an accidental product; but when it emerges from this dependence, and becomes linked with a constitutional predisposition, it is a necessary consequence. The conclusions to which these statements lead, may be thus expressed:—

1. That a constitutional predisposition to the formation of tubercle, implies a condition in which the blood and tissues are defective in their inherent vitality.
2. That this constitutional predisposition may be the result of hereditary transmission, or that it may be acquired by a long-continued functional aberration of the digestive organs, which thus influences the degree of vitality of the blood and tissues subsequently formed.
3. That the constitutional predisposition, once existing, ever afterwards affects the degree of vitality of the tissues, and of local exudations.
4. That local deposits of tubercle may take place without their being favoured by any constitutional predisposition; that, in this case, the formation of tubercle is the direct result of an imperfectly vitalised blood-plasma, arising out of a continued aberration of the functions of the digestive organs; and that its localization is dependent on the same conditions as those which determine the localization of simple inflammatory exudations.
5. That tuberculous blood is capable of nourishing the tissues in precisely the same way as in the ordinary process of nutrition, and that the localization of tubercle, like the exudation of simple inflammation, constitues an abnormal nutrition.

These views, by referring the peculiarity of tubercle to defective vitality of the blood-plasma, and by ascribing the latter condition to the influence of hereditary predisposition, as well as of those causes which determine the formation and the degree of vitality of the blood, dispose of the many theories which have been advanced, by different authors, respecting the mode in which tubercle is produced in the system. To one of these theories only it is, perhaps, here necessary to allude. The deservedly high reputation of Professor Bennett, his extensive medico-pathological knowledge, and his recently embodied investigations into the pathology of pulmonary tuberculosis, command the respect and attention of all who are engaged in similar pursuits. I shall, therefore discuss his theory of tubercle, with the view of ascertaining how far it is supported by fact. In his section on the "Nature of Tubercle,"

he introduces the subject by enunciating the fundamental facts that, for the healthy nutrition of the body, there must be a "proper admixture of mineral, albuminous, and oleaginous elements" in the food. He then refers to the theory of Dr Acherson, of Berlin, that the elementary molecules are formed of a particle of oil surrounded by a layer of albumen; and although he does not agree with Acherson that such molecules are developed directly into blood-globules and other tissues, yet he maintains that fat and albumen constitute essential elements of the nutritive chyme, that the former, divided into exceedingly minute particles by the latter, pass through the villi and form the milky fluid called chyle, that "the oil and albumen so derived from the food and so altered chemically and mechanically in the body, constitute the material from which blood is formed,"—and that "the presence and emulsionising of these elements in proper proportions, are absolutely necessary to supply and keep up the vital properties of the blood."

That fat and albumen form large constituents of the chyle, there cannot be the slightest doubt; but that the "elementary molecules are formed of a particle of oil surrounded by a layer of albumen" is by no means so certain. It is true that oil and albumen, when rubbed together, form a kind of emulsion, which consists essentially of globules; but this fact cannot be regarded as a direct proof that the albumen forms so many minute sacs, in which drops of oil are contained, since we find that the simple trituration of a drop or two of solution of potash, or of any alkali with oil, produces similar globules. Unless, therefore, these globules could be produced by the action of oil on albumen containing *no alkali*, there must remain the possibility, and, to me, the probability that their formation is rather due to the action of the alkali found in combination with albumen, than to the albumen considered absolutely as such. We know also that an alkali, added to oil, produces an emulsion-like fluid similar to the chyle, that the latter is not formed until the constituents of the food have become mixed with the alkaline secretions of the liver, pancreas, and glands of the bowels, and that both albumen and fat exist in the acid chyme of the stomach. If, therefore, the emulsionising of the chyle depended on the relative action of the albuminous and oily constituents of the food on each other, this action ought to take place in the stomach; but as such is not the case, as it only occurs when the contents of the stomach have become subjected to the alkaline secretions before named, and as we know that oil and an alkali do produce an emulsion-like fluid, there can, I think, be no doubt that the emulsionising of the chyle is entirely owing to the action of the alkalies of the bile and of the pancreatic and glandular secretions of the bowels, on the fat of the food.

The analyses of Dr Owen Rees show that the relative proportion of albumen to the fatty matter of chyle obtained from the lacteals, is, in 1000 parts, as 35·16 of the former to 36·01 of the latter.

Now, if these substances are united in the termini of the villi and lacteals to form, as stated by Dr Bennett, elementary molecules, we should naturally expect to find, in the contents of the thoracic duct and in the blood, either a corresponding proportion of these two substances, or a diminution in the relative quantity of albumen; because, on the assumption that these molecules consist each of an envelope containing a drop of oil, they would either pass into the blood as such, or the albuminous capsule of each must undergo solution before the particle of oil which it contains, could be subjected to the vital action of the mesenteric glands. But the assumed formation of such bodies in the chyle is itself a proof that there is nothing in the constitution of that fluid to effect their subsequent solution by chemical means; therefore their solution must be effected by a vital process, which is incompatible with the idea that the albumen would remain unchanged. A change in this substance would imply a diminution in its quantity; consequently, the amount of albumen in the blood would be less than that of the chyle at the commencement of the lacteals. Instead, however, of such being the case, the analyses of the contents of the thoracic duct by Rees, and of the blood by Denis, Lecanu, Lehmann, Simon, Rodier, Becquerel, and others, show that the quantity of albumen in each is equal to that of the chyle and its fatty matter taken together; whilst the fatty constituents of the blood are to those of the chyle as 1·3 to 36·01. These facts plainly show that, during the passage of the chyle along its appropriate vessels into the general current of the blood, the increase in the quantity of albumen is inversely as the reduction in the quantity of fatty matter. Is it then the office of the mesenteric glands to convert, by a vital process, the fatty matter of the chyle into albuminous compounds? The preceding facts favour this view.

From the foregoing observations, it also seems pretty certain that the molecular basis of the chyle is formed, in a great measure, by minute particles of fatty matter, *uncombined* with albumen; and that the emulsionising of this fluid is due to the action of the alkaline salts of the bile, pancreatic and glandular secretions of the bowels on such fatty basis. If the view already expressed relative to the function of the mesenteric glands is correct, the value of fatty substances, as remedial agents in the treatment of pulmonary tuberculosis, is at once apparent.

But to pass to the particular theory of the formation of tubercle propounded by Professor Bennett. He says that—"The peculiarity of phthisis, however, is, that an excess of acidity exists in the alimentary canal, whereby the albuminous constituents of the food are rendered easily soluble, whilst the alkaline secretions of the saliva and of the pancreatic juice are more than neutralised, and rendered incapable either of transforming the carbonaceous constituents of vegetable food into oil, or of so preparing fatty matters introduced into the system as will render them easily assimilable.

Hence an increased amount of albumen enters the blood, and has been found to exist there by all chemical analysts, while fat is largely supplied by the absorption of the adipose tissues of the body, causing the emaciation which characterises the disease."

With respect to that part of the above theory which refers the peculiarity of phthisis to excessive acidity of the alimentary canal, it may be stated, that such a condition is by no means invariably present; that, in those hereditarily predisposed to tuberculous deposits, such deposits very frequently take place without the slightest symptom of a dyspeptic character being present; and that were an excess of acidity of the alimentary canal an *essential* to the formation of tubercle, *all cases* in which acidity exists *ought* to lead to the deposit of tubercle. Instances, however, of excessive acidity of the alimentary canal, as evidenced by frequent and sour eructations, daily occur to the practical physician in those suffering from dyspepsia, gout, rheumatism, and other diseases, and yet there is no formation of tubercle. In phthisis, as in many other diseases, excessive acidity of the alimentary canal not unfrequently exists; but unless it could be shown that this condition is an *invariable precedent* and accompaniment of phthisis, the theory of its *essentiality* in the formation of tubercle must for ever remain untenable. But even granting the invariable existence of this acidity of the alimentary canal, what proof does Dr Bennett advance that the acid in question dissolves albumen? None; nor can he bring any such proof to the point at issue. He does not state what acid produces the alleged excess of acidity of the alimentary canal; but it is presumed, from the nature of things, that the acetic acid is the one intended. Acetic acid, however, does not dissolve albumen, but on the contrary, coagulates it—a fact announced by me in the first part of this work, but preceded, as I have since ascertained, by a similar statement from Lieberkuhn and Parkes. The objection which is thus offered to the theory of Dr Bennett, and which is manifestly fatal to such theory, is consequently supported by the authorities just named. I have, in Part I. of this work, shown that coagulated albumen boiled in pure acetic acid undergoes solution. Digested, however, for hours, at the temperature of the stomach, it experiences no change. The same may be observed of all other acids said to be discovered in the stomach either as the result of the decomposition of food, or as forming a constituent of the gastric juice. In excessive acidity, then, of the alimentary canal, the albuminous constituents of the food are thereby prevented from undergoing solution, and are consequently unable to undergo absorption into the lacteals. Instead, therefore, of the passage of an *excess* of albumen into the blood, a deficiency of it would enter that fluid, provided the acidity of the alimentary canal, as stated by Dr Bennett, really existed. The quantity, however, of albumen which enters the lacteal vessels, in cases of phthisis, is in proportion to the quantity of food taken, and to the activity of the digestive

organs; not to an excess of acidity in the alimentary canal. In phthisis, as in health, the food which is received into the stomach undergoes the same change, and is disposed of in the same manner. In both instances it is converted into blood by the same process; it is applied to the nutrition of the tissues in the same way; and it furnishes the same local exudations to causes which operate alike in both instances. Hence, the essentiality of tubercle is not in physical and chemical differences between it and the blastema poured out in the ordinary process of healthy nutrition or of simple inflammation, but in the *degree of inherent vitality* which it possesses, and which is inferior to that of health. In what way this inherent vitality is conferred on tubercle, as well as on all other exudations and tissual formations in the body, it must for ever remain impossible for us to say. It is an ultimate fact to which all our knowledge and reasoning on the subject lead us, but beyond which we are unable to go, and for which we cannot account.

In discussing still further the theory of tubercle, as laid down by Dr Bennett, it may be observed that he refers the formation of "tubercle-corpuscles" to a deficiency "in the necessary proportion of fatty matter" in the exudation, in consequence of which "elementary molecules are not formed so as to constitute nuclei capable of further development into cells; they, therefore, remain abortive, and constitute tubercle-cospuscles." Here again Dr Bennett brings forward no proof to show that there is a relative deficiency of fat as compared with the albuminous portion of a tuberculous exudation; whilst the analyses of Dr Glover demonstrate that in tuberculosis the fats of the blood are *not* deficient, and that "tubercle itself often contains a considerable quantity of fat." The fact that tuberculous blood is not deficient in fatty matters, offers a pretty strong proof that the theory of Dr Bennett is, in this respect, too, untenable; because it could not be expected that the fats of the blood would be *maintained* at the standard of health by absorption of adipose tissue, and by the disintegration and metamorphosis of albuminous structures, if those introduced by primary digestion were *invariably* deficient in tuberculosis.

In answer to Dr Glover on this point, Dr Bennett observes that—"The molecular basis of the chyle must pass in large quantity into the blood after each meal, where it may frequently be detected in the serum, constituting chylous or fatty blood;" and again, in the same paragraph, that "It cannot be an objection to the views advanced, therefore, that because the small amount of fat found by the chemist in blood, has undergone no diminution, that therefore the fatty molecules introduced by the chyle have no connection with the nutritive and morphological changes in the economy."

In reference to the former part of the above quotation, it may be remarked, that the condition of the blood (chylous) here mentioned by Dr Bennett is not that which is generally observed, and cannot, therefore, be advanced as the slightest indication of the real amount

of fatty matter which enters the blood by primary digestion during health. The molecular basis of the chyle is dissolved by ether, which thus shows it to consist of fat. Gerber and others have shown that this fat is in maximum quantity in those lacteals which pass from the intestines to the mesenteric glands, from which glands to the thoracic duct it exists in medium quantity; whilst in the last-mentioned vessel, but little trace of it is observed. It is therefore certain, that the molecular basis of the chyle does not, at any time, in perfect health, pass in "large quantity" into the blood, but that it undergoes a metamorphosis as it flows towards the thoracic duct; and it is probable that this metamorphosis is intimately connected with the increasing amount of the protein compounds of the chyle from its point of absorption, at the commencement of the lacteals, to its delivery into the general current of the blood. Hence, Dr Glover's argument against the theory that "tubercle-corpuscles" result from a "deficiency in the necessary proportion of fatty matter in the exudation," remains in full force, and Dr Bennett's theory must necessarily be considered as at present unsubstantiated by fact.

Again, in reply to the latter part of the above quotation, it may be urged that, were fat a "primary and evolving element,"[1] as considered by Dr Bennett, it would not be diminished in quantity as it flows along the lacteals; that nature would not thus all but destroy the very resources whence the nuclei of the tissues are to be drawn; that it would exist in greater proportion in the blood; that the quantity found in that fluid is manifestly insufficient to form with albumen the necessary nuclei for the proper nutrition of the tissues; that it would form a constituent part of healthy muscular fibre, and of other structures having a cellular basis of formation; and that, when found as a constituent part of such tissues, it would not be, as it invariably is, associated with disease. It seems, therefore, more consonant with facts to regard fat, absolutely considered as such, as taking no part in the formation and nutrition of tissues, than to look upon it as a "primary and evolving element" of such tissues.

That "tubercle often contains a considerable quantity of fat" is certain—not only from Dr Glover's analyses, but also from my own chemical examinations of tuberculous sputa. This fact, however, offers no argument against the theory of Dr Bennett; because the source of fat in tubercle is, as he observes, "the albuminous compound, which, like muscle, fibrinous exudation, and blood, may be transformed into oily granules by a chemical process not yet accurately determined. Flesh, by exposure to a running stream of water, is, as is well known, converted into adipocere." In the first part of this work, which was published prior to the treatise

[1] By "primary and evolving element," I suppose Dr Bennett to mean an element which enters directly into the formation of tissues.

of Dr Bennett on Pulmonary Tuberculosis, a similar view was expressed relative to the source of fat in pus. It was there observed (page 30), that the formation of "pus-cells is due to the action of oxygen on the structures of the plastic corpuscles, by which they undergo an *adipocerous* degeneration, similar to the well-known effect produced on dead muscular tissue when exposed to moisture, and to a very partial contact with air. Hence the origin of the fat which invariably forms a chemical constituent of pus; and hence also the colour of the latter fluid, as a consequence of the saponification of such fat by the alkalies present in the exudation." That the views here expressed, relative to the source of fat in tuberculous exudations are correct, the following experiments sufficiently demonstrate:—

A hundred grains of sputum, from a case of pulmonary tuberculosis, in which a large cavern existed in the right lung, were evaporated to dryness two days after being expectorated, the dry residue was triturated in a mortar, and was then digested in a certain quantity of æther in a well-stoppered bottle. After the mixture had stood several hours, during which it was frequently shaken, the fluid portion was carefully poured into a capsule, the weight of which had been previously determined, and in which the ethereal solution was allowed to evaporate—first in the open air, and afterwards under an air-pump over chloride of calcium. Having in this way removed every trace of moisture, the capsule was weighed, and was found to have gained exactly half a grain. The residue of the sputum, insoluble in ether, was next boiled in alcohol; the fluid portion was then decanted into a carefully weighed capsule, and subsequently evaporated by the aid of a gentle heat. The last traces of moisture were then removed by chloride of calcium in the manner above described, and the capsule was again weighed, when it was found to have gained 1·40 grains.

The quantity of sputum from which the above 100 grains had been taken, was now exposed to the air for twelve days, at the end of which time a part of it was evaporated to dryness, and 11·90 grains of it—a quantity equal to the dry residue of the 100 grains evaporated in the first experiment—were subjected to the action of ether and alcohol, in precisely the same manner as before. The fatty matter thus removed, was found to represent respectively ·65 and 3·60 grains. It was, therefore, evident, that by twelve days' exposure to the air, the albuminous constituents of the sputum had undergone fatty degeneration to the extent of 2·35 grains for every 11·90 grains of solid residue.

This, and other facts of the same nature, add a further confirmation of the view previously expressed, as to the origin of the pus-cell, whilst they, at the same time, demonstrate that the non-granular exudation-cell undergoes a similar degeneration, by which it is converted into a fat-cell.

From the facts now adduced, it appears that the peculiarity of

tubercle consists in its deficient inherent vitality; that the mode in which it is deposited, is precisely similar to that of a simple inflammatory exudation; and that its localization is preceded and attended by the same pathological condition of the blood-vessels, which precedes and attends an inflammatory exudation.

The indications of treatment, in pulmonary tuberculosis, must have reference to the particular stage of the disease. Three stages have been recognised, namely, 1st, That of local predisposition; 2d, that of deposition; 3d, That of germination of the tubercle.

In the first of these stages, or that of local predisposition, in which the blood-vessels are more or less engorged with blood, the indication is:—

To restore the natural diameter of the affected capillaries, and thus to prevent exudation.

It is seldom that the physician is sufficiently early in attendance to recognise this stage. In the most chronic forms of pulmonary tuberculosis, such is very rarely indeed the case; but in the acute form, as well as in those cases of intermediate character between the acute and chronic, this stage is now and then recognised. When recognised, the activity of the treatment must be commensurate with the extent of the local predisposition, and the suddenness and urgency of the symptoms. General bleeding is seldom if ever required, but local bleeding is, in many instances, extremely beneficial. The importance of this method of treatment is well illustrated by Dr Stokes, in his talented work on Diseases of the Chest. After the application of leeches, extensive counter-irritation of the chest should be practised, with the view of determining from the engorged lungs to the surface of the body. A quick effect must be produced; therefore dry cupping, mustard poultices, liquor ammonia, or hot turpentine, may be first applied, after which blisters, followed by the croton-oil liniment, or tartar-emetic ointment, will be used with advantage. A brisk purgative, exhibited contemporaneously with the adoption of the above remedies, will, in many such instances, be necessary; and in all cases, the action of the skin should be promoted by very small doses ($\frac{1}{16}$th of a grain) of tartar emetic, and by warm clothing. Perfect quietude of the voice, and a moderate temperature (60° F.), will also contribute to the end desired. After the local predisposition to tubercle has been removed, the patient should observe a plain yet substantial diet, avoid undue exposure to cold and fatigue, and always endeavour to maintain a brisk circulation through the skin, by a proper quantity of clothing. Whenever derangement of the digestive organs is present, the efforts of the physician must be directed to its removal; and for this purpose, a variety of remedies is recommended, amongst which, the preparations of soda, bismuth, the mineral and hydrocyanic acids, the salts of iron and of silver, either exhibited alone, or in combination with light vegetable tonics, are worthy of notice.

During the exhibition of such remedies, an occasional mild mercurial, coupled with a gentle saline aperient, will be found extremely beneficial in rousing the liver to a healthy action.

In the second stage of the disease, namely, that of deposition, the indications are:—

1. To prevent further deposit.
2. To promote the absorption of that which has already taken place.
3. To treat special symptoms.

To fulfil the first indication, extensive counter-irritation to the chest should be practised—in acute pulmonary tuberculosis, by a succession of blisters; in the chronic, by the tartar emetic ointment or croton oil liniment. In those cases which are of intermediate character between acute and chronic, the tartar emetic ointment, freely applied to the chest, produces pustules, which are a long time in healing, and which frequently exert a powerful influence in preventing further exudation into the tissues of the lung. It is important to bear in mind, that for any counter-irritant to be effective, it must be extensively employed. It will not be sufficient to rub with it a single region of the chest; on the contrary, the whole surface of the chest ought to be invaded by successive applications, which should begin with those regions in which the exudation has taken place, and which ought then to pass to the more distant points from the seat of disease. For this purpose, one-eighth of the whole surface of the chest may be rubbed at once, and when the eruption has appeared in sufficient quantity, a second portion of the chest should be treated in like manner; and thus the eruption may be excited successively on every part of it. By the time the eruption appears on the last part of the chest rubbed, that on the first part will be sufficiently well to permit a repetition of the application, which must be either made or withheld, according to the impression which has been already produced on the disease. In those cases in which the deposit is of very limited extent, in which it is taking place very slowly, and which seems to depend exclusively on imperfect action of the digestive organs, the extent of counter-irritation may be more circumscribed, or it may sometimes be confined to sponging, night and morning, the whole surface of the chest with tepid vinegar and salt, which should be immediately followed by brisk friction, to be continued until a temporary erythema is produced.

The use of setons and issues is attended by little or no advantage, inasmuch as they produce a less amount of irritation on the surface than exists within the chest, and thus, by exciting little or no derivation of blood from the lungs, they permit the process of exudation to continue. I have consequently ceased to use them in any case of pulmonary tuberculosis. As the pathological condition of the affected pulmonary capillaries is, in this stage, one of exhaustion

and consequent relaxation, the local abstraction of blood is, as a general rule, to be avoided. When, however, a local predisposition suddenly arises in the neighbourhood of a tuberculous deposit, and when, in consequence, a further exudation would take place into pulmonary tissue which has not as yet been invaded by the disease, the cautious abstraction of blood, either by cupping or by leeches, is to be recommended; but particular care must be observed not to resort too frequently to this method of depletion, lest the constitutional powers be thereby enfeebled, and permanent injury be done, as the cost of, perhaps, a temporary benefit derived from the local abstraction in question. In further accomplishment of the object, to prevent further exudation in this stage of the disease, warm clothing, quietude of the voice, and the constant observance of an equable temperature, are conditions which merit particular attention.

The second indication of the treatment of the stage of deposition —namely, to promote the absorption of the exudation which has already taken place—will be best fulfilled by endeavouring, in addition to medicinal means, to improve the constitutional powers, and thus to enable the exudation to undergo those changes which are necessary for its removal from the system. Amongst remedies for this purpose the cod-liver oil stands pre-eminent. The success of this agent during the last ten years, has been so great, as to justify the high encomiums passed upon it by Professor Bennett, to whom the profession is greatly indebted for its introduction into this country as a remedy of surpassing value in the disease now under consideration. Its action on the system combines that of a food and of a medicine. Its chemical composition, according to the analysis of Dr De Jongh, shows it to consist of an organic substance, which he names gaduine, glycerine, butyric, acetic, oleic, margaric, fellinic, bilifellinic, chelinic, phosphoric, and sulphuric acids, bilifulvine, phosphorus, iodine, bromine, chlorine, lime, soda, and magnesia. These constituents show that the cod-liver oil is not only a food, but that it is also one of the most powerful medicines which could possibly be exhibited. If the views previously expressed, as to the metamorphosis of fatty matters into protein compounds by the action of the mesenteric glands, are correct, we at once see why the cod-liver oil is superior to any other oil or fatty matter yet employed in the treatment of pulmonary tuberculosis. By its containing in its composition the elements of the bile, and by their being associated with phosphorus, iodine, bromine, and chlorine, the function of the liver is excited by the former, whilst by the latter powerful agents the organic-nervous, and mesenteric glandular systems are roused to an unusual degree of activity. Hence, the digestive function is increased, the mesenteric glands are better enabled to elaborate the fatty elements of the oil into protein compounds, and thus an increased nutrition of the body necessarily takes place. But cod-liver oil *is not admissible in every case of phthisis*. In acute pulmonary tuberculosis, as well as in the more

chronic form, when there is any degree of sympathetic fever present, it is positively injurious, inasmuch as it aggravates all the symptoms, both local and general. Experience had determined this fact before chemical analysis pointed out, in the presence of phosphorus, iodine, and bromine, the powerfully stimulating properties with which the oil is endowed. In the chronic forms of phthisis, in which there is considerable emaciation of the body, unattended by a hot and dry skin, red tongue, and thirst, there is no remedy of equal value; whilst in cases in which considerable sympathetic fever exists, there are few, if any, remedies whose exhibition hurries on the disease more rapidly. In acute pulmonary tuberculosis I have never found the system tolerate its use. The stomach generally rejects it, despite the form in which it may be administered; and if retained in such cases, it not unfrequently increases the diarrhœa which may be present, or excites it, if absent. The golden rule for its exhibition involves, in my opinion, the following conditions:—*emaciation, little or no thirst, cool skin, and no disposition to sympathetic fever*. These conditions observed, the oil may be administered notwithstanding the existence of diarrhœa and a furred tongue, the former of which it not unfrequently arrests. It may be given at first in doses of one or two teaspoonfuls twice a day; and as the stomach becomes more accustomed to it, the quantity may be increased to one, two, or even three tablespoonfuls repeated three times a day. Beyond this last mentioned quantity it is scarcely ever necessary to go. It is best tolerated *immediately* after a *meal*, with which it mingles in the stomach, and is in consequence gradually submitted to the action of that organ. Should bilious vomiting supervene on its continued use for some time, it is an evidence that the oil is too powerfully stimulating the liver, in which case its further use should be suspended until the liver has been unloaded by a few grains of blue pill and a saline aperient, after which it may be resumed in smaller doses than before. At first the pale coloured oil may be given; and if it be found to agree, then the darker variety, which is said by Dr De Jongh to possess greater medicinal virtues than the former, may be administered. To mask the unpleasant taste of the oil, it may be taken in spearmint, peppermint, or dill-water, or in any bitter infusion, as in that of quassia, gentian, calumba, orange peel, or hop. Cold weak brandy-and-water is a vehicle in which it is often exhibited with the least offence to the stomach. When taken in teaspoonful doses, it may be made into a mixture with a few drops of liquor potassæ, mucilage, and a light bitter infusion, of which an ounce may be given two or three times a day. If the appetite diminish after its use for a short time, and if frequent eructations, in which the taste of the oil is present, take place at a long interval after its exhibition, its further use should be suspended for several days, during which a mild saline aperient, if necessary, and the alkalies, together with magnesia and bismuth, either exhibited alone or in combination with any light bitter, will be of great service. After

the dyspeptic symptoms have been removed, the exhibition of the dilute nitric acid in a bitter infusion will be often found of advantage previously to the resumption of the oil. With the use of the oil, fats of various kinds are to be recommended. They are, however, for the reasons before named, inferior to the oil in a medicinal point of view. As a simple food, they are in no wise inferior to it; but, owing to their containing neither iodine, bromine, chlorine, nor phosphorus, they are less stimulating to the mesenteric glands, and are therefore less likely to undergo that complete elaboration which the oil itself in all probability undergoes. As substitutes for the cod-liver oil, others of vegetable origin have been tried in pulmonary tuberculosis, and report speaks favourably of that obtained from the cocoa-nut; but with me it is a question whether or not any oil, derived from other than a marine source, will in the end be found to have that efficiency which all concede to the cod-liver oil. In addition to the use of the last mentioned remedy, the absorption of the tuberculous deposit should be further favoured by a diet consisting of a due admixture of animal and vegetable food. In acute pulmonary tuberculosis, however, it is seldom that the red meats are either desired, or tolerated when taken. It is, therefore, to the use of milk, farinacea, and the different white meats, that such cases must in a great measure be restricted. But when there is no contra-indication to the use of a generous diet, the absorption of the tuberculous deposit will be best favoured by plain yet substantial foods, and by allowing, in many instances, a proper use of ale, porter, or wine. An opposite diet would, under such circumstances, be productive of incalculable mischief, and would, instead of retarding the progress of the disease, hasten its march to a fatal termination. In the further treatment of the stage now under consideration, the exhibition of other medicines is needful to those suffering from acute phthisis; but a residence on the coast is by far the most important remedy in chronic cases. In the former, the nitrate and other preparations of potash, those of soda, the acetate of ammonia, exhibited in some mucilaginous fluid, tend to allay sympathetic fever, and to promote the expectoration of viscid sputa. The cough is thus rendered less frequent and troublesome, and the ease and comfort of the patient are thereby promoted. In chronic cases, the patient should be removed as soon as possible to the coast, where he ought to remain until he has recovered his full flesh and strength. The greater the disposition in the tuberculous deposit to germinate—which is indicated by the quantity of sputa expectorated—the lighter and drier ought the air breathed by the patient to be. This rule will be found a sufficient guide to the selection of a particular climate;[1] nevertheless, when this has been determined, care must be taken that the residence of the patient be some short distance from the water's edge, in order to avoid the heavy fogs which sometimes

[1] On the subject of climate, consult the excellent treatise of Sir James Clarke.

prevail, and that it be sheltered from the easterly and north-easterly winds. If residence on the coast become irksome, a voyage of longer or shorter duration, according to the exigency of the case, may be recommended; but in thus expatriating a patient, even for a short time, his feelings must be consulted, and must give the "Yes" or "No." Unless, during an absence of this kind, the mind is perfectly satisfied with the change, the patient will not improve. A longing for home prevents, to a certain extent, the mind from enjoying the sights and reminiscences of the places visited, impairs the appetite and digestive functions, and in this way defeats the very object for the attainment of which the patient left his home. In no case ought a phthisical patient to travel alone. A pleasant and judicious companion, by acts of personal kindness, and by little attentions manifested in a variety of ways, tends to allay that unsettled feeling which often takes possession of a mind at a distance from home and alone. When a visit to the coast or a voyage is impracticable, and in some instances in which these means are adopted, the use of the preparations of potash, particularly the iodide of that salt, and also the different salts of iron, especially the proto-ioduret, in combination with some light vegetable bitter, is of service in promoting the general health, and in favouring the absorption of the tuberculous deposit.

Amongst special symptoms at this stage of the disease, cough is one of the most troublesome. In both acute and chronic pulmonary tuberculosis, it is best treated by regulation of diet, an equable temperature, counter-irritants to the chest, warm clothing, and quietude of the voice. Opiates exhibited with the view of allaying it, sometimes procure a *very temporary* relief at the expense of producing considerable disturbance in the digestive organs, indicated by sickness and vomiting, loss of appetite, headache, and manifest prostration of strength. They should consequently be given as seldom as possible; but when exhibited, they will be found to be of the most service when the skin is cool, the tongue clean, and when little or no thirst exists. When opiates are inadmissible, henbane, conium, or prussic acid may occasionally be administered, either alone or in some mucilaginous fluid.

Diarrhœa is another symptom which is occasionally troublesome during this stage. It depends on vitiated secretions of the stomach, liver, and bowels, or on a congested condition of the gastro-intestinal membrane, analogous to the congestion of the bronchio-pulmonary membrane, which immediately precedes the deposit of tubercle. The former condition may be treated by antacids, mild astringents, demulcents, sedatives, and a milk and farinaceous diet. When the biliary secretion is at fault, the preparations of soda are sometimes efficacious in correcting it. When acidity is the cause of diarrhœa, chalk or magnesia, coupled with a few grains of rhubarb, a few drops of tincture of opium, in peppermint water, will be found beneficial in the first instance, after which tormentilla, logwood, Gallic

acid, the nitrates of silver and of bismuth, and the acetate of lead, may, if necessary, be exhibited. When the diarrhœa depends on a congested condition of the gastro-intestinal membrane, counter-irritants to the abdomen may be used with advantage.

Hæmoptysis, another symptom which sometimes requires special attention during this stage, is best treated by the erect posture, cool air, perfect quietude, the frequent swallowing of ice, cold acidulated drinks, ipecacuanha, acetate of lead and opium, and the tinctures of secale and matico. Dry cupping will also assist in arresting the hæmorrhage, which may afterwards be treated, with the view to prevent a recurrence, by counter-irritants to the chest. Hæmorrhage at this stage, is a matter of less immediate danger than when excavations exist in the pulmonary tissue. In the former, it is the result of congestion of the bronchio-pulmonary membrane, which is relieved by its occurrence; in the latter, it generally arises from the laying open of a vessel by ulceration, or by its sudden laceration during a fit of coughing. In the one case, it may be regarded with little apprehension for the time being, inasmuch as it is certain to subside so soon as the congested membrane has relieved itself of its excess of blood; but in the other case, it is fraught with the greatest danger, since the vessel laid open usually permits a great quantity of blood to escape, by which suffocation is immediately threatened, whilst it rarely or never closes so effectually, as to prevent a fatal recurrence of the hæmorrhage.

Vomiting is a symptom which not unfrequently harasses the patient at this stage. When accompanied by tenderness over the pit of the stomach; by a tongue red over its whole dorsum, or red at the tip and edges; with a whitish or brownish-white fur in the centre, as well as by thirst, a blister to the pit of the stomach, together with the use of magnesia and prussic acid, or, if there is no diarrhœa, the effervescent mixtures coupled with prussic acid, the trisnitrate of bismuth, and a farinaceous diet, will be the best remedies. If, on the other hand, the tongue is large, loose, flabby, and covered with a dirty, yellowish-white, slimy fur, and if the vomits indicate the presence of bile, the sickness should be first combated by two or three grains of blue pill, with three or four grains of the extract of dandelion, and a draught, consisting of magnesia, rhubarb, and the sulphate of soda, after which benefit will be derived from the remedies previously named, or from the use of the medicinal naptha, in doses of eight or ten drops every three or four hours.

For sweating, general prostration, and other symptoms arising from debility, the manifest remedy is, to improve the constitutional powers by a good diet, and the cod-liver oil, aided by such other medicinal means as may seem necessary.

In the stage of germination, the indications of treatment are:—

1. To prevent the formation of caverns.

2. To heal pulmonary excavations where they already exist.
3. To treat special symptoms.

All these indications are to be fulfilled in much the same way, and by the same means, as those which have been recommended in the previous stage, namely, by good, generous diet, the use of the cod-liver oil, of counter-irritants to the chest, and of other remedies calculated to improve the general health, and to remove unpleasant symptoms.

END OF PART SECOND.

ON

CONGESTION OF THE HEART,

AND ITS

LOCAL CONSEQUENCES.

BY

WILLIAM JENNER, M.D.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL, AND PROFESSOR OF PATHOLOGICAL ANATOMY AT UNIVERSITY COLLEGE.

[*From Volume XLIII of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

J. E. ADLARD, BARTHOLOMEW CLOSE.

1860.

ON

CONGESTION OF THE HEART,

AND ITS

LOCAL CONSEQUENCES.

BY

WILLIAM JENNER, M.D.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL, AND PROFESSOR OF PATHOLOGICAL ANATOMY AT UNIVERSITY COLLEGE.

Received March 8th.—Read June 26th, 1860.

The principal object of this paper is to call the attention of the Fellows of the Society to the occurrence of congestion of the muscular tissue of the heart; to the most common and direct consequences of that congestion, viz., induration, toughening, and thickening of the walls of the heart; and to the influence which those changes of texture exercise (as predisposing causes) on the development of permanent dilatation of the heart.

The expression, congestion of the heart, has been hitherto used to signify extreme distension of the cavities of the heart with blood; few pathologists have even mentioned congestion of the substance of the heart, and none have, I believe, so much as adverted to its most important consequences.[1] When the right cavities of the heart are over-

[1] Bertin notices the dilatation of the veins of the heart which accompanies engorgement with blood of the right auricle: "Il (le sang) s'accumule donc dans leur cavité, distend leur parois, et les engorge jusqu'à leurs dernières extrémités." He observes also that rupture of the distended veins may occur. ('Traité des Mal. du Cœur,' 1824, p. 41.)

distended with blood from an impediment existing to its onward flow, there is not only engorgement with blood of the venæ cavæ and of the veins opening into them, but also of the coronary sinus, veins, and branches. Now, it is a pathological law, that mechanically induced congestion, if long continued, slowly formed, and intermitting altogether or in degree, has for effect induration, and also (when the fibrin exuded is not of the powerfully contractile variety) permanent increase in bulk of the organ or tissue.

Thus, when the inferior vena cava is unable to pour its blood freely into the right auricle, the liver and kidneys are at first enlarged and softened from over-distension of their vessels and effusion of serosity into their structures. If the impediment to the onward flow of the blood be quickly removed, those organs soon recover their normal size and consistence; but, if the impediment continues for any length of time, then the organs in question are found to be indurated, toughened, and (except under the conditions previously specified) permanently enlarged. The induration, toughness, and enlargement, are due, chiefly at least, to an interstitial exudation of lymph, which is found after death, either amorphous, or in the form of granules, or more or less organized into fibrous tissue. It may be that, in particular cases, there is also some true hypertrophy of the proper structure of the congested part.

When the contractile coats of a hollow viscus are indurated, toughened, and thickened by an interstitial exudation of lymph, their normal contractile power is diminished. The consequence is, that although the resistance to over-

Rokitansky has the following remarks on the subject at page 267 of the second volume of the last edition of his 'Path. Anat. § 7: Krankheiten der Textur.—Hyperämie:' "Ein Strotzen der Gefässe des Herzens, zumal seiner Venen und kleine Blutaustretungen in Form von Hirsekorn—Linsengrossen Ecchymosen unter dem Pericardium in der Nähe des Sulcus transversalis, an den Verhöfen und an den Ursprungstücken der Arterienstämme kommen bei Stenosen des Herzens und bei Asphyxien Neugeborner und Erwachsener häufig vor."

distension of the walls of the viscus is greater than natural, yet, if the distending force be sufficient to overcome that resistance, and be repeatedly applied at short intervals over a length of time, permanent and extreme dilatation of the viscus will be produced.

What is true in these respects of other organs and tissues is true of the walls and cavities of the heart. When there is long-continued obstruction to the passage of the blood out of the right ventricle, and consequently impediment to the free entrance of the blood from the coronary sinus into the right auricle, congestion of the walls of the heart follows. If the impediment to the exit of the blood from the sinus be slowly produced, be moderate in degree and permanent, or be frequently repeated, then induration, toughening, and thickening of the walls of the heart will ensue, and permanent dilatation of its cavities be the final result; the over-distension of the walls of the cavities being the immediate cause of the dilatation—the induration, toughening, and thickening being the cause of the permanence of the dilatation.

The peculiarities of the indurated and toughened walls of the heart are as follows:

When divided across, the outer wall does not fall inwards, the form of the cavity being still retained, even though, as is sometimes the case, the walls are (in consequence of the extreme dilatation of the cavity) thinner than natural; the cut surface of the muscular tissue has a very smooth, compact, homogeneous appearance; the columnæ carneæ stand firmly out; the tissue is harder than natural, and singularly tough. This tough, leather-like quality is one of the most marked characters. The colour of the indurated, toughened tissue may be paler or darker than natural.[1]

The microscopical appearances of the indurated and toughened tissue are—

1st. The striæ of the muscular fibres are generally

[1] Laennec described several of these characters as proper to the dilated and hypertrophied heart. De l'Auscult. Méd., vol. ii, p. 514.

indistinctly seen, though here and there they are as perfect as in health.

2d. The muscular fibres are more firmly united to each other than in the healthy heart.

3d. Both between and within the muscular fibres are innumerable molecular granules, chiefly protein.

4th. Lymph which has a granular form exhibits in the heart, as elsewhere, a tendency to undergo fatty degeneration, and the muscular fibres of the heart, damaged by the congestion and the presence in and among them of the lymph, are prone to the same change; the consequence is, that it is common to find here and there a considerable number of particles of free olein as well as muscular fibres which have undergone fatty degeneration.[1]

5th. In some cases the cellular or connective tissue seems to be increased in quantity.

When the exit of the blood from the right auricle is suddenly or greatly impeded, it is not unusual to find small crimson spots studding the external surface of the heart, and less commonly the internal surface. These spots are evidently extravasations of blood, the result of capillary hæmorrhage from mechanical obstruction to the onward flow of the blood.[2] Under like conditions there is often found a considerable excess of serosity in the pericardium, and œdema of the loose cellular tissue at the base of the ventricles; the latter especially, if the patient is much emaciated.

When the congestion has been extreme and long continued, the coronary sinus is found to be more capacious than natural.

In studying the effects of congestion on tissues and organs, it is necessary to separate not only slight from extreme congestion, but also continuous from intermitting

[1] When the fatty metamorphosis of the muscular tissue and lymph is extensive and extreme, softening of the walls of the heart may ultimately occur; the parietes of the heart being first indurated, and subsequently softened, in consequence of the indurated tissue undergoing fatty degeneration. See Rokitansky, in previous note.

congestion, suddenly formed from gradually formed congestion, and congestion of organs the action of which is normal or increased from congestion of organs the functions of which are less actively performed than in health.

It is the very gradually developed, long-continued, intermitting congestion of organs the functions of which are over-actively performed notwithstanding their congestion, which is especially followed by induration, toughness, and hypertrophy.

The causes which lead mechanically to an accumulation of blood in the right cavities of the heart have been attentively studied, and are well known. They are referable either to disease of the arteries of the heart itself, or to some cause interfering with the free passage of the blood through the vessels of the lungs.

The following cases are sufficient to *illustrate* the main facts adverted to in the foregoing remarks; viz., the most common causes of mechanical impediment to the passage of the blood through the right side of the heart; the relation between those impediments, engorgement of the right cavities of the heart, and congestion of the walls of the heart; the result of that congestion of the cardiac parietes, viz., induration and toughness, especially affecting the parts most in action; and the influence of induration and toughening of the walls of the heart in the production of permanent dilatation of its cavities.

Case.—A woman, æt. 50, suffered during life from general anasarca, symptoms of extreme congestion of the lungs, liver, kidneys, &c. After death, the walls of the heart were found greatly thickened, and the cavities dilated. When the heart was opened the divided walls still retained their rounded form; they did not fall inwards; the substance of the organ was remarkably tough and hard; the mitral orifice was much constricted; the left auricle was scarcely larger than natural, the right was very capacious; the foramen ovale was so patent as to admit the points of three fingers; the right auriculo-ventricular orifice was very

large (five inches and a quarter in circumference); the coronary sinus and veins were considerably dilated. The orifice of the aorta seemed of normal size, it measured two inches and five eighths in circumference; the orifice of the pulmonary artery was so much dilated as to measure four inches and a half in circumference. There was a large amount of subendocardial and subpericardial extravasation of blood in very thin layers, varying in extent, at some places forming mere specks, at others large patches.

The kidneys were hard, tough, and granular. The liver was remarkably tough and granular, and fibrous at the margin.

The lungs were the seat of pulmonary apoplexy.

When examined by the aid of the microscope, every fibre of the heart was found to be studded, both within and without, with minute granules—the majority protein, a few olein. The fibrous tissue seemed to be more abundant than natural. The cross-markings of the fibres were less distinct than are those of the fibres of a healthy heart.

Remarks.—The primary disease in this woman was evidently narrowing of the left auriculo-ventricular orifice. The blood, thus prevented from readily passing into the left ventricle, must have exerted abnormal pressure on the septum auricularum; as a consequence, it is probable that, an obliquely patent foramen ovale, such as is so often found when no admixture of the blood occurs, became dilated. The pressure on the two sides of the septum being unequal, the blood must have passed from left to right auricle, thus accounting for the difference in the relative size of the two auricles. The large size of the pulmonary artery was, doubtless, due to the impediment to the escape of blood from the pulmonary veins, and to its passage through the lungs.

These changes, as well as the condition of the liver and kidneys, point to the long continuance of the impediment to the circulation; the large size of the veins of the heart, and the extravasation of blood under the visceral pericardium and under the endocardium, show the degree to which the substance of the heart must have been congested during life; the toughness and hardness of the tissue of the walls of the heart were singularly well marked.

Case.—J. S—, æt. 35, a man of intemperate habits, by trade a carpenter, was admitted into University College Hospital, November 14th, 1860.

On September 6th he had an epileptic fit. On the 16th he was seen by Dr. Coghlan, of Notting Hill, who found, in addition to much cerebral disturbance, a loud, soft, blowing, systolic cardiac murmur, having its point of greatest intensity at the apex; the heart's impulse abrupt and irregular in force and frequency; the pulse at the wrist so rapid, weak, and irregular, that it could not be counted. No anasarca.

Against the advice of his medical attendant the man resumed work, and when again seen he was suffering from extreme dyspnœa and extensive dropsy.

On admission into the hospital, about six weeks after the supposed commencement of his illness, the most prominent symptoms were extreme dyspnœa, anasarca, and ascites.

All the superficial veins were distended with blood. The jugular and subclavian veins became distended to the utmost when the man coughed; they pulsated synchronously with the heart's beat; a strong thrill was perceptible by the finger placed over the point of junction of the right internal jugular and subclavian veins. On slight pressure both the pulsation and the thrill ceased.

Physical signs indicated the existence of hypertrophy and dilatation of the heart, and regurgitation of blood through the right and left auriculo-ventricular orifices. The urine contained a considerable quantity of albumen. The congestion of the various organs and tissues diminished very greatly during the first few weeks of the man's stay in the hospital; so much so that the anasarca almost disappeared; there was scarcely a trace of albumen in the urine; the pulse fell to 96, and was more regular in force and frequency, and the

man could lie on his back, and walk about the ward without difficulty.

For a fortnight before the man's death, on January 4th, 1858, however, the distension of the venous system was even greater than on his admission.

The larger veins were abruptly dilated at intervals, indicating the situation of their valves. There was distinct pulsation of cardiac rhythm in the veins of the arm; pressure on a vein stopped the pulsation on the distal, but not on the proximal side of the point of pressure; the pulsation, therefore, was not transmitted through the capillaries. The thrill in the veins at the root of the neck was very perceptible to touch, it was synchronous with each beat of the heart. A loud systolic murmur, evidently generated in the veins, was audible at the spot where the thrill was to be felt. There was neither thrill nor murmur in the carotid or subclavian arteries.

At two points, where the bulging of the brachial vein attained the size of half a broad bean, the pulsation of the vein was perceptible to touch as well as eye.

There was orthopnœa, extreme anasarca, and ascites.

After death about eight ounces of reddish serosity was found in either pleura, and ten ounces in the pericardium.

The heart was much larger than natural. The right auriculo-ventricular opening measured six and a quarter inches in circumference. The left auriculo-ventricular opening was so much contracted from old disease of the mitral valve, that even the points of two fingers passed through it with difficulty. All the cavities of the heart were much dilated; that of the left ventricle, however, less so than the others.

The walls of the heart were thicker and much harder and tougher than natural. When cut across they did not fall inwards, but the cavities still retained their rounded form unchanged.

The coronary sinus was very capacious; the right internal jugular vein was enormously dilated; the valves at its orifice were perfect, as were the valves of the veins of the arms.

There was no disease of the aorta itself, and only adhesion to a trifling extent of two adjacent aortic sigmoid valves to each other.

The aortic orifice measured three inches in circumference. The pulmonary artery measured three inches and a half in circumference.

The liver was small, but singularly tough; it was strongly granular; the tissues of the portal canals were especially thick and tough; the acute margin of the organ was reduced to fibro-cellular tissue. The kidneys were exceedingly hard and tough, and finely granular. The lungs were the seat of pulmonary apoplexy and congestive pneumonia.

Remarks.—The starting point of the disease of the heart in this case evidently was at the mitral orifice; to this all the other diseased states of the organ were mechanically referable. The disease of the mitral orifice was doubtless, notwithstanding the history, of long standing.

How extreme must have been the impediment to the passage of the blood through the right side of the heart, was shown by the remarkable fulness of the superficial veins. The distension of the right jugular vein was so great that the valves at its opening into the subclavian vein no longer sufficed to close the aperture, and regurgitation was permitted; in the same way as regurgitation was permitted through the dilated right auriculo-ventricular orifice from insufficiency of its healthily sized valves to close the abnormally large aperture. The thrill felt at the root of the neck, and the systolic murmur heard at the same point, were evidently due to the same cause, viz., to the flow of blood past the margin of the valves into the dilated jugular vein.

That the same cause which had led to the distension of the visible veins, viz., the impediment to the escape of blood from the right side of the heart, had also led to distension of the veins of the heart, was shown by the dilatation of the coronary sinus and the amount of serosity in the

pericardium. The condition of the liver and kidneys indicated that they were the seat of the exudation of contractile lymph, favoured by, if not the direct result of, their mechanical congestion. The induration and the toughness of the heart were so great that the attention was at once arrested by them.

Case.—T. B—, æt. 9. This boy had from a very early period of his life been the subject of well-marked cyanosis, and some time before death suffered from a considerable amount of anasarca.

After death, in the *pericardium* was found rather more than two ounces of transparent serosity, and its tissue was more opaque and thicker than natural. Here and there beneath the visceral pericardium were crimson spots due to extravasation of blood; there was a good deal of serosity in the loose cellular tissue at the base of the heart, *i. e.* between the auricles and ventricles.

The *right auricle* and *venæ cavæ* were filled with recent clots and fluid blood. In the appendix of the auricle was an old clot, and a clot also of some age was interlaced among the columnæ carneæ of the *right ventricle*.

The *left auricle* contained much fluid blood, and in its appendix was an old clot.

The *left ventricle* was almost filled with old clot; at first sight it looked as if a fungous growth was sprouting upwards from the apex. A recently formed clot extended from the old clot into the aorta.

The right and left sides of the heart, both auricles and ventricles, were greatly dilated, and their walls thickened. The *valves* were quite healthy.

Dimensions of orifices—aortic, $1\frac{1}{8}$ths inch; pulmonary artery, $2\frac{1}{4}$; left auriculo-ventricular, $3\frac{1}{2}$th; right auriculo-ventricular, $4\frac{1}{4}$th. The weight of the heart, with the old clot in the left ventricle, $7\frac{3}{4}$ ounces.

The *coronary sinus* was of very large size.

The liver, spleen, and kidneys were all remarkable for their hardness and toughness.

As far as could be ascertained this boy had been the subject of cyanosis from his earliest infancy. The long duration and the extreme degree of the impediment to the circulation through the lungs and right side of the heart were shown by the large size of the pulmonary artery and tricuspid orifices and of the coronary sinus, as well as by the state of the liver and kidneys. That there was no great cardiac impediment to the circulation at the time of birth was proved by the closure of the foramen ovale. The sub-pericardial hæmorrhage, and the œdema of the cellular tissue at the base of the heart, indicated a sudden extreme increase in the obstacle to the circulation.

Remarks.—It is probable that the primary affection was endocarditis of the apex of the left ventricle occurring very soon after or shortly before birth; that coagulation of blood on the roughened surface followed, and that to the clot so formed the impediment to the circulation was due. The enlargement, hardness, and toughness of the abdominal viscera evidently resulted from their mechanical congestion. Can we doubt that the extreme induration and toughness of the walls of the heart were due to the same cause?

Case.—J. R—, æt. 47, a cabman, of intemperate habits, was admitted into University College Hospital, November 17th, 1857, suffering from extensive and extreme hypertrophous emphysema of both lungs, capillary bronchitis, and hypertrophy and dilatation of the heart, more especially affecting its right side.

The man stated that he had been for many years subject to winter cough, and that he had previously had five or six attacks of bronchitis, the last about a year ago; that his breath was always short, and his face and lips dusky purple in colour; that a year since he first observed that his legs were swollen, and shortly after that his abdomen was larger than natural; that he vomited a little blood two years ago, had often coughed up mucus streaked with blood, and had suffered repeatedly from epistaxis.

He dated his present illness from the 9th of November, when he had rigors, followed by heat of surface, thirst, increase of cough, and dyspnœa, and was obliged to take to his bed.

When I saw him after he entered the hospital he was suffering from extreme orthopnœa. The whole surface was livid. The vessels of the conjunctivæ were dilated and filled with blood; the eyeballs were prominent, the lower lip swollen and everted. The large veins of the neck, which during inspiration were scarcely perceptible, were distended during expiration, but did not pulsate. The heart's sounds were not audible, though its pulsations were perceptible below the ensiform cartilage. There were present all the physical signs of extreme, extensive hypertrophous emphysema. There was some anasarca. The pulse was 132, very small and weak; the respirations were 38 in the minute. The urine contained a large quantity of albumen, and some small waxy casts.

On the 18th of December the colour of the man's face, trunk, and extremities was darker, *i. e.* he was more cyanosed, than I ever saw the skin of a patient who was not the subject of congenital heart disease.

The lividity varied in degree during the succeeding fortnight. On the 28th the veins of the neck were constantly much distended, and pulsated; there was a large quantity of fluid in the peritoneal cavity; the anasarca had increased in amount; there was a systolic murmur audible at the base of the sternum, *i. e.* just above the ensiform cartilage.

On the 30th my notes were—Cyanotic symptoms more marked than at any time since admission. Heart's beat lower. Left lobe of the liver considerably depressed by the manifestly over-distended right side of the heart and the emphysematous lungs. Veins of neck extremely full, knotted, pulsating.

The man died on the 31st.

On examination after death there was found extreme hypertrophous emphysema of both lungs; great hypertrophy and dilatation of the right side of the heart, moderate hypertrophy and dilatation of the left side. The walls of the heart were indurated and very tough; this change affected the right side infinitely more perfectly than the left. The muscular tissue of the right side of the heart was very compact, and the cut surface particularly smooth and homogeneous in appearance. The mitral orifice measured 4½ inches in circumference; the tricuspid, 5½ inches; the aortic, 3 inches; the pulmonary artery, 3½ inches. The coronary sinus admitted the little finger with facility; it was evidently much dilated. There was general anasarca, and a considerable amount of fluid in the peritoneal cavity.

The liver was enlarged, it weighed fifty-two ounces. It was uniformly and finely granular; it was hard and very tough. The kidneys were large and irregularly contracted, so as to be coarsely, unevenly granular on the surface. They weighed six ounces each.

Remarks.—This case affords a good example of the most common causes of over-distension of the right cavities of the heart, and therefore of congestion of the walls of the heart, and, as a consequence, of induration, toughening, and permanent dilatation of the heart; viz., repeated attacks of bronchitis and hypertrophous emphysema. The congestion of the whole venous system showed the great impediment that existed to the flow of blood through the right side of the heart; and the extent to which the capillary system had suffered dilatation proved the length of time that the impediment had existed. The anasarca and the large size of the coronary sinus supported the same inferences.

The liver and kidneys were all enlarged, hard, and tough—changes doubtless chiefly due to their long-continued mechanically produced congestion. The fluid in the peritoneum and the albumen in the urine were also, probably, due to the obstacle to the return of blood from the liver and kidneys. The induration of the heart was very decided; the specific gravity of the most markedly indurated part of the right ventricle was taken for me by Mr. Russell, in Professor Williamson's laboratory; it was 1·053.

The very trifling evidence of old pulmonary collapse found after death, and the enormous extent and degree of the emphysema, conjoined with the fact that, during life, there was very forcible protrusion of the intercostal spaces above the level of the ribs, and equivalent prominence of all the soft parts of the thoracic parietes in violent expiration, told strongly in favour of the expiratory theory of emphysema laid before the Society by myself in 1857.

Case.—Robert W—, aged two years, was a healthy child till the commencement of 1858, when, his mother said, he suffered from bronchitis. During that attack she noted that his feet were swollen. All œdema disappeared in the course of the ensuing spring, but he never recovered his previous state of health.

The child was admitted into the Hospital for Sick Children on January 30th, 1859—his mother stating that his present illness was of only three days' duration.

There were present the physical signs of general but imperfect solidification of both lungs; strongly marked pulsation in the jugular and subclavian veins; a little lividity of the lips; œdema of legs, hands, and face. The child was seized with convulsions on the 1st of February, and died.

After death the whole of both lungs were found to be loaded with gray granulations and yellow tubercles; and here and there, in addition to much collapse, were nodules of lung solid from pneumonia.

About two drachms of serosity were found in the pericardium; the veins on the surface of the heart were filled with blood.

The pulmonary veins, the venæ cavæ, both auricles, the right ventricle, the pulmonary artery, and the aorta contained a large quantity of dark, loosely coagulated blood. The walls of the right ventricle were only slightly thicker, but were much tougher and harder than natural. The walls of the left ventricle were much less tough and hard than were those of the right ventricle, and its cavity was not dilated.

Examined by the aid of the microscope, the muscular

fibres of both ventricles were found to be studded within and without by protein-granules. A few of the muscular fibres were in a state of fatty degeneration. The most completely degenerated fibres were found in the muscular band which passes from the right wall to the septum ventriculorum.

The spleen, liver, and kidneys, were all loaded with blood, and tougher than natural.

Remarks.—Here the primary impediment to the circulation of the blood was seated in the lungs. Bronchitis, pulmonary collapse, emphysema, and an enormous accumulation of tubercles in every part of the lungs, were the causes of the obstacle; the degree and duration of the impediment were indicated by the dilatation of the right auriculo-ventricular orifice, by the state of the spleen, of the liver, of the kidneys, and of the veins, and by the anasarca.

The texture of the heart was damaged in the same mode and from the same cause as was the texture of the liver, spleen, and kidneys, *i. e.* from mechanically produced venous congestion.

Case.—E. B—, aged eight years and nine months, was admitted into the Hospital for Sick Children on September 10th, 1859. In November, 1856, she suffered from severe rheumatic fever, and from that time had been obliged to cease her occupation, viz., that of a tight-rope dancer. When she came under my care she was suffering from extreme anasarca, ascites, great fulness and pulsation of the veins of the neck and upper extremities, and orthopnœa. The chest was in form the type of that which accompanies great hypertrophy and dilatation of the heart in a child. The physical signs indicated very plainly the lesions found after death.

She died October 3d, 1859; the body was examined twelve hours after death. The sternum being removed, the tissues of the mediastinum were found to be much more vascular than natural, and, in common with the cellular tissue at the root of the neck, were infiltrated with serosity,

and remarkably tough. There were about four ounces of transparent serosity in the pericardium. Numerous crimson spots, due to sub-pericardial extravasation of blood, were present at the base of the heart, especially about the root of the pulmonary artery; the loose cellular tissue between the ventricles and auricles contained much serosity. Both auricles contained very large coagula; that in the right auricle was continuous with a clot in the coronary sinus. The latter clot, when removed from the sinus, measured three inches in length. The veins on the surface of the heart were gorged with dark-coloured blood. The walls of the heart were greatly hypertrophied, especially those of the right ventricle; they were remarkably hard, tough, and dark in colour. The tissue of the walls was more compact, its cut surface smoother, its edges sharper, and its aspect more transparent than natural. All the cavities of the heart, excepting that of the left ventricle, were extremely capacious. The left auriculo-ventricular orifice was greatly reduced in size, from disease of the mitral valve; the right measured $3\frac{3}{4}$ths inches in circumference; the aortic orifice, $1\frac{3}{4}$ths inch; the pulmonary artery, $2\frac{1}{4}$ inches.

The liver, spleen, and kidneys were all loaded with blood, and very tough.

Examined by the aid of the microscope, the muscular fibres of the heart were found to be paler, tougher, and more closely united to each other than natural; they were covered with very fine molecular granules; their transverse striæ were very imperfect. There appeared to be an excess of fibrous tissue in the walls of the heart.

Remarks.—The primary disease in this case was evidently constriction of the mitral orifice, and the duration of the disease three years. The toughness, &c., of the cellular tissue, the state of the liver, spleen, and kidneys, the dilatation of the pulmonary artery, the serosity in the pericardium, the fluid in the cellular tissue at the base of the heart, the dilatation and engorgement with blood of the coronary sinus and the veins on the surface of the heart,

and the sub-pericardial extravasation of blood, were all evidently due to mechanical impediment to the passage of the blood through the left auriculo-ventricular orifice.

The change in the texture of the heart was, as in the preceding, the result of the same cause that produced the change in the texture of the liver, spleen, and kidneys; and the change was of the same nature; only, in the case of the heart, the call on the organ to act was more powerful than in health, and the impediment to the onward flow of the blood necessarily induced over-distension of the cavities behind the impediment.

ON THE

INTESTINAL DISCHARGES

IN

CHOLERA.

BY

E. A. PARKES, M.D.

ASSISTANT-PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

(From the London Journal of Medicine.)

London:
RICHARDS, PRINTER, 100, ST. MARTIN'S LANE.
1849.

ON THE INTESTINAL DISCHARGES IN CHOLERA.

By E. A. PARKES, M.D., Assistant-Physician to University College Hospital.

THE chemical and microscopic examinations of the discharges from the alimentary mucous membrane, present certain difficulties, and are liable to certain fallacies, which do not occur in the investigation of the constitution of the urine. Whatever abnormal ingredients may exist in the urine, must have been derived from the blood, and have passed, with more or less change imposed upon them, through the secreting textures of the kidney or bladder. But the excretion from the intestinal mucous membrane is, in health, mixed with a mass of extraneous matter, such as the debris of food, the excrementitious portions of certain secretions, etc.; and, in disease, with the abnormal discharges induced by medicines, or even with the medicines themselves. In the case of severe Cholera, however, many of these possible difficulties and errors are avoided. The fæculent contents of the intestines are, at an early period of the disease, expelled; no solid food is taken, or, if taken, is ever tolerated by the stomach; the bile, and probably the pancreatic fluid, is retained; and there is no fear of urine mixing with the stools, for the kidneys are inactive. The Cholera dejections are therefore the products of the mucous membrane alone, with the possible addition of certain medicines, or of fluids, which, having remained on the stomach, and then passed into the intestines, have been discharged nearly unaltered.[1] In the following observations, I believe even these sources of error have been guarded against. In most of the cases I was aware of the treatment which had been employed; and if any fluid that I obtained, was found to contain an obviously extraneous and foreign ingredient, I did not make use of it.

The stools in Cholera, it need hardly be said, consist of two very distinct portions: of a colourless fluid, which is turbid, or, more usually, nearly clear, or perfectly transparent; and of a sediment, which is white, and of different physical characters, according to the period of the case. In several cases I have made analyses of the fluid, and have also made, or caused to be made, microscopic examinations of the sediment. In the following paper I shall first detail the chemical analyses of the supernatant fluid; and subsequently, the microscopic appearances of the cholera-masses in the same cases.

I. CHEMICAL EXAMINATION OF THE FLUID.

Two of these analyses were made by Dr. Garrod, the remainder by myself. I have shortened all details as much as possible; and have not included negative experiments, unless of an important kind. Although it has not been possible to analyze, as I desired, all the stools passed during the course of a single case, it will be seen that my observations embrace all periods of the illness in different cases, and indicate, with tolerable accuracy, the probable course of events in individual patients.

[1] This seems to be the case with milk, which often is not rejected by the stomach, and then passes through the bowels so unchanged as not only to give the stool a milky appearance, and to present under the microscope multitudes of oil globules, but actually, after standing for some time, to form a kind of cream.

Observation 1. *First or second true rice-water stool of the algide stage, from a middle-aged man, who subsequently died.* Analyzed by Dr. Garrod.—The stool separated into two parts, on standing: *specific gravity* of the entire fluid, 1009.16 at 56° Fahr.; *specific gravity* of the clear fluid, 1009.40 at 53° Fahr.; or if the temperature be taken into account, about 1009.0; *reaction*, slightly alkaline, but well marked; *odour* peculiar, slightly fæculent.

Heated with a small quantity of *nitric acid*, the fluid acquired a pink tint, and a precipitate fell; a peculiar odour was developed;[1] there was no effervescence. Strong nitric acid destroyed the pink tint. The alcoholic extract gave the tint with nitric acid; showing, in this way also, that it could not be dependent on uric acid. On drying the watery extract, however, and exposing it to the vapour of ammonia, a pink colour again became evident, most probably from the formation of murexide. No urea could be detected. With *hydrochloric acid*, the purple tint, from the presence of a protein compound, was produced. *Acetic acid* gave a slight precipitate. There was none of the organic principle of the bile.

Composition.—Water		985·35
Solids		14·65
		1000·00
Albumen	2·40	
Other undetermined matters, including principle coloured by nitric acid, trace of uric acid, etc.	1·27	
Salts, phosphates, and chlorides,—the latter in excess	10·98	
	14·65	

Dr. Garrod remarks, that "the ash has nearly the same composition as that of the serum of the blood, the chlorides being greater in amount than the phosphates."

Observation 2.—*Stool passed by a man aged about* 50, *thirteen hours after the commencement of the attack, and six before death. Algide symptoms intense.*—The stool separated, on standing, into two parts. The supernatant fluid was perfectly watery and limpid, passing readily through fine filtering paper. *Reaction*, markedly alkaline; *odour*, peculiar, and faint,—perhaps slightly fæculent; *specific gravity*, 1009.5, at 49° Fahr.

The liquid became hazy by heat, and the peculiar odour was rendered more intense. A drop of *nitric acid* increased the turbidity; a few more drops considerably increased it, and produced a very faint, but decided violet, tint; a further addition of acid destroyed this colour, and gave a light yellow tinge. A tolerably copious precipitate fell. When acidulated with *acetic acid*, the yellow prussiate of potash, and the bichloride of mercury, both gave precipitates. The ash of the fluid was perfectly white; it effervesced with nitric acid; the solution of the salts was strongly alkaline. There was no urea, nor uric acid, in the fluid.

[1] Dr. Garrod compared this odour to that of butyric acid.

Composition.—Water		989·13
Solids		10·87
		1000·00
Coagulable organic matter,—probably albumen	1·18	
Incoagulable organic matter, and a little phosphate of lime	0·55	
Soluble Salts, viz.: Chlorides of sodium and potassium	5·188	
——— ——— Phosphate of soda	1·059	
Other soluble salts, of which sulphate and carbonate of soda formed a part	2·893	
	9·14	
	10·87	

Observation 3.—*Last stool passed by a woman, aged* 28, *in the deep algide stage, six hours before death; cramps had been severe, but had nearly ceased.*—The whole stool was only ʒx in quantity. It separated into two parts: the fluid part was perfectly clear and watery. It was filtered through fine paper. *Reaction*, alkaline; *odour*, peculiar,—not at all fæculent; the *specific gravity* was not taken.

Boiling produced no change of colour. A drop of *nitric acid* produced a little decided effervescence; but no violet tint. A few more drops gave a flocculent precipitate; but there was no change of colour. A very peculiar odour was given off; and a gas was evidently disengaged, as it was now difficult to boil the liquid, on account of its occasional violent ebullition. The odour was certainly different from that of butyric acid, with which it was at the time compared. It was less sharp, and had more of a burnt odour. By adding nitric acid in excess, and boiling for some time, the odour disappeared, and the precipitate more completely separated itself. There was no uric acid. The ash of the fluid effervesced with nitric acid.

Composition.—Water		990·294
Solids		9·706
		1000·000
Organic matter (of which a large portion was albumen) and insoluble salts	2·186	
Soluble salts, viz.: Chlorides, phosphates, and sulphates	7·520	
	9·706	

Observation 4.—*One of the last stools passed by a middle-aged man, in the deep algide stage, who subsequently died.* It had the usual physical appearance, and was alkaline.

Dr. Garrod was kind enough to examine it very carefully for urea, and uric acid, but could not detect the presence of either. The organic compound which gives the red or violet tint with heat, and nitric acid, was also absent. The stool contained, as usual, salts, and coagulable organic matter, but the quantities of these were not determined.

Observation 5.—*Either the* 6*th or* 7*th stool passed by a woman, aged* 23. *There were severe cramps at the time. The algide symptoms were well marked, but the patient ultimately recovered.*—The stool consisted of two parts, as usual. The fluid was thin, watery, and colourless, but not perfectly transparent. It was not filtered, but drawn off with a pipette.

Reaction, strongly alkaline; *odour*, peculiar, and very slightly fæculent; *specific gravity*, 1008.3, at 60° Fah.

With *heat*, a slight haziness occurred. A very small quantity of *nitric acid* added to the hot fluid, gave a very beautiful violet tint, which boiling for several minutes did not destroy. The addition of more nitric acid at once destroyed it, and the liquid assumed a slightly yellow tinge; a flocculent precipitate fell. The peculiar odour was given out as usual. The ash, but not the fluid, effervesced with nitric acid.

Composition.—Water		989·17
Solids		10·83
		1000·00
Coagulable organic matter	0·27	
Incoagulable organic matter and insoluble salts	2·23	
Soluble Salts, viz.: Chloride of sodium	4·013	
——— ——— ——— potassium	0·791	
——— ——— ——— phosphate of soda	0·326	
——— ——— ——— sulphate of soda	0·457	
Other soluble salts	2·713	
	8·83	
	10·83	

Observation 6. *A stool passed by a girl, aged* 11, *who had been much purged, and had vomited, and had had severe cramps. At the time she passed this stool, the pulse was imperceptible, or nearly so; the eyes were sunken, the surface was very cold, but there was comparatively little cyanotic tinge of the skin, and respiration was easy; six hours afterwards, reaction commenced, and she recovered.*

The fluid was thin, colourless, and slightly opaque; it was filtered through coarse paper.

Reaction, faintly alkaline; *odour*, peculiar, not at all fæculent; *specific gravity*, 1005.8 at 65° Fah. By heat the fluid became hazy. It was not treated with nitric acid. The ash effervesced with nitric acid.

Composition.—Water		991·053
Solids		8·947
		1000·000
Organic matter, of which an undetermined quantity was albumen	3·12	
Soluble Salts, viz.: Chlorides, phosphates, sulphates, carbonates	5·827	
	8·947	

Observation 7. *The last stool passed by a boy, aged* 10, *seven hours before death. There had been from eight to twelve previous stools during the illness. The algide symptoms were intense, but there were no cramps at any time.*[1]—The stool measured ʒxij—it was like gruel; there was a very incomplete separation of the flaky substance from the fluid, until gentle heat was applied, when the flaky substance fell, and left a perfectly clear and limpid supernatant fluid. It was deemed advisable to estimate the solids without separating the suspended solid matter.

Reaction, alkaline; *specific gravity*, 1010.27, at 52° Fah. The fluid would not pass through the coarsest filtering paper, and very slowly through muslin.

Nitric acid and heat produced a yellowish or fawn colour, there was no red tint; the peculiar odour, rather sharper than usual, was given

[1] Cramps are often absent in children; but in boys of this age they are generally present.

out. When the flaky substance was separated, the thin fluid gave a flocculent precipitate with nitric acid. The residue had a brownish colour. The phosphate of lime was in unusual quantity, and may have been derived from the unseparated flaky substance which caused the quantity of organic matter to appear large.

Composition.—Water	982·68
Solids	17·32
	1000·00
Organic matter, a portion of which was albumen	8·962
Soluble Salts: Chlorides, phosphates, sulphates	7·258
Phosphate of lime	1·100
	17·320

Observation 8. *A stool passed by a boy, aged ten, who had presented all the algide symptoms in moderate intensity. Reaction was just commencing, and in a few hours afterwards, consecutive fever was fully established.*[1]

The stool did not separate readily into two portions; when it did so, the supernatant fluid was subalbid, and turbid. The liquid was strained.

Reaction, alkaline; *odour*, peculiar, not at all faeculent; *specific gravity*, 1014, at 56° Fah.

The fluid, when heated, gave out a peculiar odour. *Nitric acid*, in small quantity, produced a yellowish tint, and a more peculiar and characteristic odour was evolved, but not so well marked as in some of the stools. An excess of nitric acid was added, and the liquid was kept boiling for six hours, when a distinct, but very small, precipitate fell, which was in too small a quantity to weigh.

Composition.—Water	972·82
Solids	27·18
	1000·00
Organic matter, of which only a small portion was albumen, and insol. salts	20·84
Soluble Salts, viz.: Chlorides of sodium and potassium	2·938
——— Phosphate of soda	1·8
——— Sulphate of soda	·3951
Other soluble salts, including carbonates	1·2069
	6·34
	27·18

Observation 9. *A well marked case in a woman, aged 27; the symptoms were, however, mild, though very characteristic; she ultimately recovered. First stool during the period of reaction which had a faeculent smell; at the time it was passed, the woman still presented many of the algide symptoms, though the pulse was stronger, and the surface and breath were regaining warmth.*

The stool was of a yellowish-white, or subalbid colour, and separated into two portions on standing; the upper portion was whitish-yellow,

[1] This patient did not pass urine for fifty-three hours; he then passed about ten ounces of clear yellow urine; after standing for twenty four hours, there was no sediment. The specific gravity was 1.016, at a temperature of 56°. By heat and nitric acid the fluid acquired a light red tinge, and about one third of its bulk of albumen was thrown down. Evaporated to one-sixth, and treated with nitric acid, it exhibited, after twenty four hours, no nitrate of urea. It was not tested with Pettenköfer's test for choleic acid.

thick, and turbid; the sediment was greenish-yellow, granular and dense, and with a decided faeculent smell.[1]

Fluid filtered through coarse paper. *Reaction*, faintly alkaline: *specific gravity*, 1008.91, Temp. 55° Fah.

When boiled for ten minutes, the colour changed, and became of a light red, or fawn tint—the liquid continued alkaline—a peculiar odour was given out, which was not faeculent—there was no ammonia evolved. A few drops of nitric acid added to the hot liquid, gave a dark red, or brown colour; a copious precipitate fell, and the usual odour was given out. After boiling for an hour and a half, the red colour was unchanged. An excess of nitric acid, however, at once destroyed the colour, and the liquid became of a light yellow tint. It appeared as if a portion of the precipitate was re-dissolved. The ash effervesced with nitric acid; the solution of the salts was strongly alkaline.

Hydrochloric acid, boiled with the liquid, gave a slight, but very distinct purple tinge, and a precipitate fell; a great excess of acid neither destroyed the colour, nor re-dissolved the precipitate. Sulphuric acid gave a reddish tint, and a flocculent precipitate. Uric acid was not tested for; but after some days keeping, the fluid had acquired a smell very much like stale urine.

Composition.—Water	983·38
Solids	16·62
	1000·00
Coagulable organic matter not redissolved by excess of nitric acid	1·48
Incoagulable organic matters	6·055
Soluble and insoluble salts, the former consisting of chlorides, phosphates, sulphates, and carbonates	9·085
	16·62

Observation 10. *A boy, aged* 15, *attacked with Cholera. Subsequent reaction and febrile symptoms; then recurrence of rice water purging. The stool was taken at this time. Ultimate recovery.*

The stool separated into two parts by rest. The fluid part was watery, but turbid, and of a very light brown colour; it had no faeculent smell. The sediment was greyish-white, and was divided into two strata; the upper whiter, more flocculent, and less granular, than the lower. The fluid portion was filtered through coarse paper.

Reaction, markedly alkaline; *specific gravity*, 1017.83, at 45° Fah.

Heated with *nitric acid*, it effervesced slightly; a red tinge appeared, which became very much deeper after a few minutes boiling. An ex-

[1] This woman passed urine, for the first time, thirteen hours after the fair commencement of reaction. It was about five ounces in quantity, faintly acid, and of a yellow colour. Specific gravity, 1.018. It was highly albuminous, and threw down, with heat and nitric acid, a copious precipitate, which occupied nearly one-half of the bulk of the fluid. At the same time a very deep red colour was struck by the acid, exceedingly like the colour given by nitric acid to the stool in typhoid fever, only not quite so dark. A portion evaporated to an eighth of its bulk, and nitric acid added, gave no crystals of nitrate of urea after thirty-six hours standing.

A white flocculent precipitate subsided from this urine after a few hours standing. As I had not time to examine this, my friend, Mr. Clover, examined it, and found the following objects:—

1. Scaly and columnar epithelium.
2. Corpuscles like mucous or exudation corpuscles.
3. Vibriones.
4. Pus corpuscles?

There were no crystalline objects.

cess of nitric acid being added, and the boiling continued, the colour was partially, but not entirely destroyed. A tolerably copious precipitate fell. Uric acid was not detected.

Composition.—Water	981·79
Solids	18·21
	1000·00
Organic matter coagulated by heat and nitric acid, albumen (?)	·855
Incoagulable organic matter and salts . . .	17·355
	18·210

Owing to an accident, the soluble salts were lost after they had been dissolved out from the incinerated mass, and were being evaporated to be weighed. They appeared to be in their usual quantity, and assuming this to be 8 parts in 1000, it leaves 9.355 for the incoagulable organic matter.

Observation 11.—*A middle-aged man, after rallying from the cold stage of well-marked Cholera, continued to suffer from diarrhœa. Seven days after reaction, he had a return of what was considered rice water purging. He ultimately recovered.*

The fluid separated into two portions; the sediment was white and flocculent.

Reaction, faintly alkaline; smell peculiar, not at all faeculent; there was not sufficient fluid to take the specific gravity. Heat and nitric acid gave no violet, or red colour, nor at first was there any precipitate; but after some time, a small flocculent precipitate appeared. The peculiar odour of cholera stools was given out.

Composition.—Water	991·53
Solids	8·47
	1000·00
Incoagulable and coagulable organic matter (the latter in small quantity) and insoluble salts	4·589
Soluble salts	3.881
	8·470

Observation 12.—*Fluid taken from the small intestines after death.* After filtration, it was distinctly alkaline; it coagulated by heat alone, and also by heat and nitric acid, which produced no red colour. It contained chlorides and phosphates in abundance. After some days, crystals of triple phosphate formed in it.

Observation 13.—My friend, Mr. J. E. D. Rodgers, has been kind enough to furnish me with the following account of his examination of a specimen of *intestinal fluid taken after death.*

The fluid was alkaline before evaporation, but after this process it yielded a strongly acid solution.[1] After incineration, the ash yielded a strongly alkaline solution, which contained carbonates. "Analysis indicated an immense quantity of alkaline chlorides, alkaline and earthy phosphates, and albumen."

In addition to the analyses given above, I have examined several other cases, and have always found, that the thin fluid was alkaline,

[1] Mr. Rodgers suspected this acid to be the butyric, but was not quite certain on that point.

and contained an abundance of alkaline chlorides, phosphates, and sulphates, and a certain proportion of albumen. In one or two cases, there has been a trace of iron, possibly from the presence of blood particles, which form an occasional, but very infrequent, constituent of the Cholera stools.

These observations correspond with those of O'Shaughnessy,[1] Vogel, Wittstock,[2] Buchanan,[3] and many others, made during the epidemic of 1832. I shall now proceed to draw such inferences as may safely be done from so small a number of cases.

1. There is no doubt that it is incorrect to speak of the Cholera fluid as the serum of the blood; the fluid is derived from the serum, but is not composed of all its ingredients; it consists of its water, and of its salts, with a very small proportion of its organic elements. If other organic elements of the serum are poured out, they do not remain fluid. The ash effervesces like the ash of the serum, with nitric acid.

2. The Cholera fluid, in its purest form, consists of little else than water, salts, and coagulable organic matter, which is probably albumen. The incoagulable organic matter, or extractive, which is probably an excretion from the intestinal mucous membrane, is reduced to its lowest proportion, (in one case, only .55 in 1000 parts), and possibly, in some cases, may be altogether absent.

3. This soluble incoagulable organic matter, or extractive, which exists in great quantity in healthy fæces, soon disappears from the cholera fluid. It appears to be restored when reaction commences. It raises the specific gravity of the fluid and the proportion of the solids in 1000 parts.

The following observation proves its existence in the mildest form of Cholera, or what may be called, as the algide symptoms were not present, choleroid diarrhœa:[4]—

Observation 14.—*Stool from a middle-aged woman, who had had serous diarrhœa for two or three days. Vomiting then came on; twelve hours after its commencement, she had cramps in the extremities.* The stool was passed a few hours after the commencement of cramps; at this time there was languor, and the pulse was weak; but there were no other signs of collapse; the skin was warm, and the eyes were not sunken; she speedily recovered. Cholera was ravaging the neighbourhood; and a daughter of this woman was at the time labouring under its developed form.

The stool separated, as usual, into a fluid and sediment.

The fluid was slightly brownish in tint, and semitransparent; the sediment was brownish and flocculent.

Reaction, distinctly but not strongly alkaline. *Odour* peculiar, not at all faeculent. There was not enough to take the specific gravity.

No effect was produced by heat. A small quantity of nitric acid did

[1] Report on the Chemical Pathology of the Malignant Cholera. Highley, London, 1832.
[2] Simon's Chemistry by Dr. Day, vol. ii, p. 382, and Hoefle, Chemie und Mikroscop am Krankenbette. Erlangen. 1848, pp. 87-8.
[3] Observations on the Malignant Cholera, 1832, p. 11.
[4] In another less advanced case of premonitory diarrhœa, the extractive was 3·846, the albumen ·466, the phosphate of lime ·548, and the soluble salts as much as 9·04, in the 1000 parts. The sediment was white and flocculent; and was found, under the microscope, to consist entirely of undigested food, chiefly vegetables.

not produce any red or violet tinge; an excess gave a slight yellow tinge, and a precipitate, which had previously formed, separated itself completely. An odour similar to that from other cholera stools was at the same time evolved. The solution of the salts was strongly alkaline.

Composition.—Water	986·9
Solids	13·1
	1000·0
Coagulable organic matter, albumen	0·29
Incoagulable organic matter and insoluble salts	6·82
Soluble salts, viz.; Chlorides, Phosphates, Sulphates	5·99
	13·10

The following tabular view, from which the seventh Observation is excluded, will show more conspicuously the relation of the extractive matter, as well as of the albumen and salts, to the cholera fluid:—

Period of disease in which the stool was passed.	Specific gravity.	Albumen in 1000 parts.	Extractive in 1000 parts.	Sol. salts in 1000 parts.	Total of solids in 1000 parts
Diarrhœal period	1012·9	0·466	3·846	9·04	13·9
Diarrhœal period	—	0·29	6·82	5·99	13·1
Early algide stage	1009	2·4	1·27	10·98	14·65
Developed & intense algide stage	1009·5	1·18	0·45	9·14	10·87
Developed & intense algide stage	—	2·186		7·52	9·706
Developed and moderate ditto	1008·3	0·27	2·23	8·33	10·83
Developed and moderate ditto	1005·8	3·2		5·827	8·947
Commencement of reaction	1014·9	20·84		6.34	27·18
Commencement of reaction	1008·91	1·48	6·055	9·085	16·62
Relapse	1017·83	0·855	17·355		18·21
Relapse	—	not weighable	4·589	3·881	8·47

4. Although the extractive is thus affected by the intensity of the disease, it does not appear that the salts and albumen are equally so. The analyses are too few to allow me to speak with certainty; but it seems probable that, both in the premonitory period, and in the stage immediately following the algide period, when the pulse is again felt and warmth is returning to the surface, the proportion of effused salts and perhaps of the albumen, may be as great in any particular stool as in the depth of the algide stage. The number of stools, of course, differs in the respective periods.

5. The albumen and the salts do not seem to bear a very constant proportion to each other; but the salts hardly ever seem to be thrown out, without carrying with them a portion of albumen, however small.[1]

[1] In the liquid stools of typhoid fever (by which term I mean the disease described by Louis), the quantity of albumen in solution, is, as in Cholera, very small; and is never found unless soluble salts, similar to those of the blood-serum, are also present. In one case it was 0·63, while the soluble salts were 7·24, in 1000 parts. In a second case it was 0·457, and the salts were 5·164, in 1000 parts. It would even appear as if some portion of the albumen of the serum were so much more intimately combined with the salts than the remaining larger mass, that it is alone effused when the salts are poured out.

The greatest amount of the albumen is very trifling; and I have never seen any other case in which, from the bulk of the precipitate, I was led to infer that it very greatly exceeded in amount the quantity stated in the above analyses.

6. Boehm remarks,[1] that the discharges probably do not all come from the mucous membrane; but that some of the fluid may be derived from the immense quantity of water drank by the patient. This may be the case sometimes, but there is no indication of such a dilution in the above analyses; the proportion between the salts and the water is tolerably well preserved, and renders it likely that these constituents were both derived from the same source.

7. As in the deep algide stage the excretion of the extractive is arrested, so also, in great measure, is that of the earthy phosphates, which are thrown out so largely by the healthy intestinal surface. In some cases there has hardly been a trace of the phosphate of lime; in others this has existed in diminished quantity. In no case has it nearly equalled the healthy standard, or approached to the excessive increase which occurs in the stools of typhoid fever. In the cholera stools, the crystals of triple phosphate, although sometimes seen, are infrequent.

8. Nitric acid does not, as Simon supposed, produce any change of colour in the true cholera fluid. When the violet tint does occur, the stool has generally a fæculent smell. The colour is clearly not owing to uric acid, for it is destroyed by an excess of nitric acid, though not, in my experiments, by boiling. Moreover, as Dr. Garrod pointed out, it must be owing to an organic substance soluble in alcohol. It is then either to be attributed to the presence of bile, or to some substance excreted by the intestinal mucous membrane. There is no evidence of bile beyond this colour, and as there is a remarkable retention of this product in cholera, as in dysentery, I am disposed to think the last supposition the most probable one. The same organic matter, if I may speak from the similarity of the remarkable tint produced by the acid, was passing off by the urine in one of the cases of reaction. As uric acid appears to be usually excreted in very small quantity in the first urine passed after cholera,[2] the colour was probably not attributable to it, and it was indeed dissimilar in shade.[3]

[1] Die Kranke Darmschleimhaut in der Asiatischen Cholera, etc., von Dr. Ludwig Boehm. Berlin, 1838, p. 15.

[2] Dr. Letheby has lately made two analyses of urine passed during reaction, in which the proportion both of urea and uric acid is low, while that of extractive (kreatine, kreatinine, and other organic matters) is very high.—(*Medical Gazette* for December.)

[3] The same colour, only of a very much deeper shade, is given by the fluid part of the stools in the "fièvre typhoide," and I have also seen it several times in the urine of persons labouring under obscure affections of the digestive organs, not connected, as far as I could make out, with the liver. It is important to remember, as bearing on the question of the nature of fever consecutive to Cholera, that urea and uric acid and kreatine are not the only excretions which may be retained in the blood; it is very possible that the peculiar organic compounds, usually excreted by the intestinal mucous membrane, may be also retained, in consequence of the deep injury this membrane has undergone. In other diseases, I have been led to infer, although it is exceedingly difficult to prove the supposition, that the interruption to the secreting function of the intestinal mucous membrane produces serious disorders in other organs, which may be connected with it in function, or may be called upon to supply the deficiency. I have formerly argued that certain diseases of the liver, of the pan-

9. Although the inference of Wittstock, drawn from the colour given by nitric acid, that the cholera fluid contained uric acid, is erroneous, because the colour cannot be always obtained, and when obtained, cannot be owing to uric acid, it appears, from Dr. Garrod's first analysis, that a trace of this acid may exist, as it does in healthy serum; Dr. Garrod, in his second analysis, could not detect any, and I have carefully looked for it without success.

10. In many cases, Pettenköfer's test for the organic principle of bile has been used, but no trace of this substance has been found.

II. MICROSCOPIC EXAMINATION OF THE SEDIMENT IN THE STOOLS, THE SO-CALLED "CHOLERA MASSES."

Many stools receive the name of "rice-water", which have no claim to this appellation. The sediment in the true rice-water stool is perfectly white, in flocks or little shreds, and is exceedingly light. Sometimes, the entire stool seems not to have a greater specific gravity than the fluid separated from the sediment. This lightness may be illustrated by the following fact:—The whole flocculent substance from a stool which measured more than a pint in quantity, and which was well washed and dried, in order that it might be submitted to ultimate analysis, weighed only four grains.

The most accurate microscopic examination of the cholera masses are those made by Boehm in 1832, and described and figured in the work to which I have already referred. His descriptions, however, apply almost entirely to the fluid contained in the intestines after death, and he does not seem to have bestowed equal attention on the sediments of the stools. As the proper comprehension of Boehm's admirable researches is essential to the discussion on this subject, I shall, as briefly as clearness will permit, detail his opinions.

In the first chapter, Boehm describes minutely the changes in the epithelium of the intestinal mucous membrane in Cholera. In the second chapter, he describes the microscopic character of the cholera masses. After saying that, in all languages of Asia and Europe, the epithets flocculent, creamy, like groats-broth, like rice-water, etc., have been applied to the intestinal fluid, he goes on to say that the various products, however different to the naked eye, described under these terms, may be all referred to changes in the epithelial coat. The thin fluid, however, he decides to be undoubtedly a secretion from the blood, which is poured in large quantity through the diseased intestinal membrane. The varieties of the intestinal contents depend on the varying proportions of this secretion and the cast-off epithelial particles, and on changes in the latter. He then enumerates the several varieties. The fluid was like *milk* when the quantity of secretion was great, and the epithelial particles were all separated from each other; it was *purulent* or *creamy* when the epithelium, separated into its finest elements, was scantily diluted with the secretion; it was *flocculent* when, with a profuse secretion, the epithelial particles were not separated, but cohered together, and under the microscope presented flat lamellæ if they came from the smooth mucous coat, or crooked cylinder-like masses, or tubes, domes, or glove-like cases, according as, more or less perfectly cohering, they were shed from the apices, bases, or entire surfaces of the villi, or from the interior of the follicles; it was like *rice-water* when the flocculi, in smaller quantity, swam in a slightly turbid secretion; it was like *oatmeal-broth*, when the larger epithelium masses were of various colours, white or greyish green, mixed intimately with each other, assuming, from the scanty secretion mixed with them, a pulpy consistence. When the cholera masses were adherent, this was owing to the partial separation of large patches of epithelium, the further mixing of which with the general contents of the intestine was prevented by death. Boehm then goes on to describe the *mucous* intestinal contents (which, he says, are rare as long as Cholera is acute), the *bilious* contents seen at the end of the slighter cases, and the *bloody* contents seen in some of the severest cases. With these last observations I shall not concern myself at present. Boehm makes one very important observation, viz., that while in the severest cases of Cholera epithelial particles, in immense quantities and with the sharpest outlines, are found on the iliac side of the ilio-colic valve, on the other side of the valve their number at once diminishes in an extraordinary manner, and sometimes they are not seen at all. He accounts for this by supposing that the epithelial coat of the small intestine is soluble in the fluid poured into the large intestine. He says also, that he yet seldomer found pyramidal epithelium particles in the discharges, although there was a quantity of flocculi, which, at the end of the chapter, he describes as composed of semitransparent long strings of an elastic consistence, so that, after being pressed between plates of glass, they recovered their form. These flocks often enclosed little crystals of a rhomboidal form, which, as they also occurred in the cholera fluid, seemed to imply that these elastic shreds might arise from coagulation of some material, which perhaps, he says, owed its origin to solution of epithelial fragments.[1]

In some respects, it would be difficult to describe more accurately the contents of the intestines after death; in no other disease are such beautiful specimens of epithelium seen, and their several conditions cannot be better described than they are in Boehm's work. But the account of the flocks of the stools is meagre and unsatisfactory; and Boehm appears to me to have even overlooked some elements in the intestinal contents.[2]

creas, and less frequently of the kidneys, follow large and irreparable destruction of the mucous membrane of the colon in tropical dysentery. Some affection of these organs is not uncommon after mild attacks of Cholera; it may be, possibly, from a similar cause.

[1] Op. cit. pp. 10 to 35.

[2] I have not been fortunate enough to meet with any detailed observations made during the present epidemic in St. Petersburg or in Berlin, on the intestinal discharges. From some very interesting debates, however, in the *Gesellschaft für wissenschaftliche Medicin* of Berlin, reported in *Die Medicinische Reform* for September, October, and November, it is apparent that Boehm's opinion of the extreme importance of the casting off of the epithelium is considered erroneous. This is not only maintained by Virchow and the partisans of the opinion which regards Cholera as a general blood disease, but also by Leubuscher and others, who consider all symptoms as sequences of the disease of the intestinal mucous membrane, and who, it might have been supposed, would have attached much importance to changes in the epithelial coat. Thus Leubuscher says that he cannot consider, with Boehm, the essence of Cholera to be repre-

Before proceeding to this point, I shall subjoin a few microscopic observations on the stools. I was anxious to have these made by independent observers, so that no hypothesis of my own might be supposed to have biassed me. The names of the gentlemen who so kindly complied with my request, is an ample guarantee for the extreme accuracy of the observations. The numbers refer to the corresponding analyses of the fluid of the same stool.

Observation 1. *Early algide stage.*—Examined by Dr. Jenner, with a power of 400 diameters.

"1. Some closely aggregated amorphous, very fine granular matter, rendered somewhat more transparent by acetic acid.

"2. Numerous minute granules, the largest transparent and yellow, surrounded by a dark border, the smallest dark and opaque; acetic acid rendered some of the granules more transparent; liquor potassæ dissolved a few, and rendered others more transparent; boiled in ether for seven minutes, they appeared undiminished in number and unaltered in appearance.

"3. Organic corpuscles, similar to those described subsequently (Observation 6, No. 3), but much fewer in number, and generally smaller; many of them imperfectly formed, the capsule enveloping them being indistinct, so that they looked like a simple aggregation of black and yellow granules.

"4. A few semitransparent, peculiarly smooth granules, unaffected by cold liquor potassæ or acetic acid.

"5. No epithelium particles, no pus globules, nor blood corpuscles, either red or white."

Observation 2.—*Developed and intense algide stage.* Examined by Professor Ellis.

"The white, flaky substance, suspended in the rice-water stool, consists of a clear transparent basis, which contains a granular material and cells.

"*Granules.*—The granular material is seen on the surface of the mass, and is collected here and there into patches of uncertain size and irregular form; the molecules composing it are very minute.

"*Cells.*—The cells are strewed thickly through the mass; and, speaking generally, they may be said to be rather larger than the white corpuscles of the blood. In many parts they seem to be arranged in lines, but this appearance may be due to only a certain number coming into focus at the same time.

"*Form of cells.*—Some of the cells are roundish in form, though none are circular; and many (perhaps an equal number) are oval or elongated. The lengthened cells are about the size of the others in their short diameter.

"*Contents of cells.*—Both sets of cells have finely granular contents, and a dark defined circle round them.

sented by this throwing off of epithelium; this is only a secondary process; "die Abstossung ist immer nur ein secondärer Prozess."—(Op. cit. Nov. 3, p. 124.)

It may not be uninteresting to mention that, in these debates, are many important details of the "diphtheritic inflammations" of various organs, which occur in the consecutive fever.

"*Size.*—The statement respecting size must not be taken too literally, for many cells are larger than the size stated, and many are smaller.

"*Action of acetic acid*, equal parts of acid and water. Acetic acid causes the mass to shrink and become more transparent. The cells become very indistinct, being in some cases scarcely recognizable, even where the material is thin. But where the mass is thick, the granular material comes more distinctly into view. Of course the fibriform appearance is lost; the acid seems to gelatinize the mass.

"*Nature.* The material resembles exudation mass more than any other substance."

Observation 3.—*Developed and intense algide stage.* Examined by Professor Sharpey.

"1. No characteristic epithelium particles to be seen.

"2. Slimy flakes, containing, scattered about, irregular-shaped particles, and small molecules.

"3. A few white finely-granular corpuscles; some round, others of a more or less irregular shape; in size a little smaller than the pale corpuscles of the blood."

There may have been other matters; but Dr. Sharpey had time to make only a cursory examination.

Observation 4.—*Developed and intense algide stage.* Examined by Mr. Hillman; Mr. Quekett, of the College of Surgeons, was kind enough to confirm the observation, and to make sketches of the objects, which I possess.

"1. Squamous epithelial particles; none of the columnar form.

"2. Nucleated and granular bodies, resembling exudation corpuscles.

"3. A small number of blood corpuscles.

"4. Irregularly-shaped flattened cells, with single large central nuclei; rather larger in size than the exudation corpuscle.

"5. Membranous shreds, resembling basement membrane."

Observation 5.—*Developed and moderate algide stage.* Examined by Mr. Clover. Unfortunately it could not be examined till four or five days after it was passed. There was a quantity of granular matter, and scaly and columnar epithelium.

Observation 6.—*Developed and moderate algide stage* Examined by Dr. Jenner.

"1. Colourless, semitransparent, amorphous, membranous flakes; dissolved or rendered transparent by acetic acid or liquor potassæ. Boiling ether has no effect upon them (albumen?)."

"2. Semitransparent fibres, lying in close apposition; perhaps united by No. 1, or may be only the same matter arranged in striæ. They were seen most distinctly at the parts of the field in which the tissue had accumulated in the thickest masses. Liquor potassæ dissolved them, or rendered them very transparent (fibrine?).

"3. Organic corpuscles; round, oval, irregularly oval, or pointed at one end, and rounded at the other; more or less spherical, finely-granular on the surface, and containing, in their interior, minute granules, varying in number from six or eight, to sufficient to fill the entire corpuscle.

Some of these contained granules were mere black points; others were transparent and yellow, with dark margins. In size, the round corpuscles varied from 1-4000 to 4-5000 of an inch in diameter. The oval corpuscles were about 1-4000 in breadth and 4-5000 in length. They had both a faintly-yellow tint. Boiled in ether for seven minutes, they were unchanged.

"4. Granules similar to those described in observation 1, No. 2.

"5. One large granular body, semiopaque and yellow; 1-1000 of an inch in diameter; unaffected by acetic acid.

"6. Vibriones in great numbers. Two or three oval transparent bodies placed end to end. When the stool was kept, these fungi increased in numbers."

Observation 7.—*Intense algide stage. No cramps throughout illness.* Examined by myself. Power of microscope, 400 diameters.

1. Amorphous finely-granular matter.
2. A few molecules.
3. No epithelium or blood corpuscles.

In this case the flaky matter did not separate from the thin fluid till gentle heat was applied; it then formed a confused sediment, which had not the usual flocculent appearance. Mr. Bowman saw this stool, and confirmed the above observation.

Observation 8.—*Commencement of reaction.* Examined by myself. Power of microscope, 400 diameters.

1. A large quantity of transparent gelatinous matter, completely dissolved by acetic acid.
2. In the meshes of this matter, many dark or yellowish granules, unaffected, or nearly so, by acetic acid.
3. In some few cases, these granules were aggregated together, and formed little masses; whether then surrounded by a cell-wall could not be determined.
4. One or two large cells, containing two or three central granules.
5. No epithelium. After a long examination, one or two particles were seen, which looked like debris.

Observation 9.—*Commencement of reaction.* Examined by myself, immediately after it was passed.

There were a few epithelial scales, and granular and molecular matter, but hardly any of the peculiar corpuscles formerly described.

Observation 10.—*Relapse.* Examined by Mr. Quekett and Mr. Hillman.

"1. Small masses of biliary resin.

"2. Granular matter, similar to that seen in mucous corpuscles, and such as is obtainable by squeezing the follicles of the intestinal canal.

"3. Portion of muscular tissue (adventitious).

"4. No epithelium—no exudation corpuscles—no blood."

Observation 11.—*Relapse.* Examined by myself.

To the naked eye, the sediment appeared partly finely-granular and partly flocculent,—not at all unlike some true Cholera masses. It consisted entirely of shreds of striped muscular fibre (adventitious), of particles, apparently of undigested food, and of molecular matter. No dark yellow granules were seen; neither was there any appearance of epithelium, nor of blood.

Observation 12.—*Fluid from intestine.* Examined by myself.

1. Epithelium particles very numerous.
2. Organic corpuscles, about the same size as the pale corpuscles of the blood, finely granular on the surface, and containing from six to twelve dark yellow or black granules. Acetic acid caused no appearance of nuclei.
3. A few red blood corpuscles, very much altered and irregular in outline.
4. A few cells, like immature epithelial particles, small and irregular.
5. Granules of the same size as those described in No. 2, but free.

After several days' keeping, the fluid was again examined, when the same elements were seen, with the addition of a few crystals of triple phosphate.

Observation 13.—*Fluid from Intestine.* Examined by Mr. Rodgers.

An immense quantity of epithelium was seen. A dried specimen exhibited a large quantity of crystals of alkaline chlorides, a few crystals of phosphate of ammonia and magnesia, and a little phosphate of lime.

Observation 14.—*Choleroid diarrhœa.* Examined by Mr. Clover and myself.

Epithelial scales, granular matter, and shreds of vegetable fibre and cells derived from food, were observed.

In addition to the above examinations, I have made some others, which agree completely with them, but which, to avoid repetition, I shall not here insert.

These observations, made by independent observers, have a remarkable uniformity among themselves, but differ completely from Boehm's descriptions. In the true Cholera masses, there are the fibres to which he alludes; but there exist also corpuscles, peculiar granules, and an amorphous flaky matter, which he has not noticed in the stools, nor in the contents taken from the small intestine after death. In both these situations, these elements exist in abundance, though mixed, in the intestine, with an immense quantity of epithelium, which may possibly, in some cases, obscure them, or cause them to be overlooked.

I do not think any one who had ever seen these corpuscles, could for a moment suppose them to be any modification of broken down epithelium, or to be immature and imperfectly-formed epithelial nuclei. They are different in size, and in all their physical appearances; and, moreover, it must be remembered, that cylinder epithelium is confessedly scarcely ever seen in the cholera stools; and that if it were so seen, and could by any change or breaking down form either the corpuscles or the granules, we ought to be able to find all grades and phases of the change. Neither can it be considered probable that the epithelium of the small intestines is soluble in the fluid of the large, and that then, by deposition, it forms these corpuscles, or the fibres which Boehm describes. In the fluid of the small intestines, the epithelial particles often preserve their sharp and definite outline for days, although they occasionally do seem to break up. It is by no means probable that the fluid effused into the cæcum, which is doubtless of the same nature as that from the whole canal, can possess any such solvent power.

If not epithelium, can these be mucous corpuscles and granules

thrown off, as it were, by an intense *catarrh* of the mucous coat?[1] In reply to this, it may be argued: 1, that these bodies are much smaller than the large corpuscles with the central granules, seen in mucus; the granules are of a different kind, are differently arranged in the interior of the cell, and are differently acted on by acetic acid. 2. It is not likely that mucus would be secreted in such enormous quantity, while the usual excreting functions of the mucous membrane are evidently totally abolished. 3. If these corpuscles are mucous, why should they not appear through the whole course of the disease, instead of being confined to the deep algide period? They are not seen in the premonitory diarrhœa, nor after the algide stage; and disappear when the pulse and the warmth of the surface are returning. Now, it seems unlikely, that if they are formed by an intense irritation of the mucous coat, they should so rapidly disappear. I do not wish, however, positively to contend that these corpuscles are not mucous, since I have not compared them, as I hope soon to do, with the intestinal mucous corpuscles in other diseases.

But whatever be the nature of these corpuscles, it seems to me impossible to consider the fibres and gelatinous-looking flakes, mucus. If they are so considered, everything which appears on the surface of a mucous membrane may be called mucus, without any regard to the nature of the action which has been exerted upon the transuding blood fluid, by the textures through which it passes. These fibres and flakes, in all respects, chemical and microscopical, resemble those seen in the so-called inflammatory exudation matter, and it is in the highest degree probable that they owe their origin to effused blood-plasma, which assumes with great rapidity a low, ill-defined, and non-progressive organization. This opinion is rendered still more probable by the fact, that other elements of the blood are effused in the cholera fluid, and that the plasma itself is certainly poured into the solitary and Peyerian glands, (distending their capsules to a great extent,) and perhaps into the entire mucous membrane.

Boehm alludes very briefly to the possibility of this occurrence. He writes:—

"In previous observations, carried on to determine the nature of the flocculent substance, that opinion was most likely, which considered that together with the serous element of the blood, the so-named albuminous, or plastic material, transuded in a dissolved form into the intestinal canal, and then separated in the form of flocculi. In this case, however, the flocculi would naturally exhibit no organized structure and arrangement."[2]

Boehm certainly appears to have been a little biassed by his observations on the great desquamation of epithelium, when he did not proceed to *prove* that this unorganized plastic matter was not present, an observation it was certainly incumbent upon him to make.

With regard even to the separation of the epithelium, although, from the facility with which this structure is shed, even during certain ordinary healthy processes, it does appear probable *a priori*, that it would be

[1] This would probably be the opinion of Andral, who has already (*Gazette Médicale*, No. 33, 1847) called this substance "modified mucus."

[2] Op. Cit., p. 10. From the size of Boehm's plates, it appears probable that his microscope was not of sufficient power to enable him to see the corpuscles.

largely thrown off in Cholera, there is absolutely no proof that it is so thrown off, until after the death of the patient. The stools contain none, or a quantity not more considerable than is present in common diarrhœa, if even so much.[1] If the epithelium is separated, during life, what could prevent its passing off? It cannot surely be separated and yet retained. Is it then so broken up that it cannot be recognized? This is impossible. Boehm himself accounts for this difficulty, by assuming that the epithelium is dissolved in the fluid of the large intestine; but this is, in the first place, unlikely; and secondly, is not indicated by analysis. Besides, if during the whole course of Cholera, epithelium is thrown off, how is it that we find it after death in such great abundance?[2] Is it not most likely that the epithelium does not really separate in any quantity during life; but that after death, being continually immersed in the fluid which fills the intestines, and being possibly, in some measure, rudely shaken by the previous rapid transit of the Cholera fluid, it may separate itself more or less completely from the basement membrane, and divide itself more or less minutely into its smallest elements, according to the length of time after death, the violence used in opening the intestine, and other obvious circumstances?[3] It is quite certain that the several varieties of the stools, such as milky, chalky, &c., are given, not by epithelium, but by very small granules. In the milky stools, there is often a complete "molecular base."

It appears to me the most simple, and the most likely view to consider all the flocculent matter of the true Cholera stool, cells, dark yellow granules, fibres, flakes, and amorphous matter, to be but modifications of the same substance, viz., fibrine. The granules appear to form, by aggregation, little masses, round which a cell wall forms in the way which, as pointed out by Henle, occurs in certain inflammatory exudations, or perhaps cells may form from the liquid plasma, as in the low organization taking place in the follicular deposit of typhoid fever.

Whether this be the case or not, it may be considered probable that the microscopic forms now referred to, co-exist in their greatest perfection with the purest type of the Cholera fluid.[4] They are effused while the disease is at its acme. Immediately after the algide stage, in the first stool of reaction, they become indistinct, and finally disappear; the effusion of fibrine into the intestinal canal appears to have stopped; the sediment of the stools consists now only of amorphous matter, a few perhaps of the peculiar granules, and a little epithelium. It may be mistaken, however, if judged only by the eye, for a true Cholera mass, although it does not contain the characteristic elements.

[1] It is probable, however, that in the period of reaction especially, there is a great tendency to desquamation, and that one form of consecutive fever is complicated with this.

[2] In order to account for the great quantity of flocculent matter found after death, and the small quantity passed in the stool, I formerly, believing the matter to be of the same kind in both, suggested that the thinner portions only might pass off; but this explanation is unnecessary, if the opinions advanced above are correct.

[3] The epithelium of the gall-bladder, in other diseases besides Cholera, separates in this way after death, particularly where the bile is watery.

[4] I must state, however, that I have met with two or three stools, passed during the cold stage, in which the flocculent matter appeared granular and amorphous. But these were all mild cases, and there were foreign matters in the stool which obscured the view, and I may mention that in these cases it was singular that there was very little lividity of the skin.

Then gradually the quantity of Cholera fluid lessens, and finally, bile pigment appears, and the stools lose altogether the Choleraic character.

Want of space compels me to dismiss these most important topics, with this indication merely of the chief points of interest. I hope on a future occasion, to be able to adduce additional facts in corroboration, or modification of my views, and to enter more fully into the question of the relation of the individual elements of the Cholera dejections, to the general phenomena of the disease.

AN INQUIRY INTO THE BEARING OF THE EARLIEST CASES OF CHOLERA, WHICH OCCURRED IN LONDON DURING THE PRESENT EPIDEMIC,

ON THE STRICT THEORY OF CONTAGION.

FROM NO. VII OF THE

British and Foreign Medico-Chirurgical Review.

BY

EDMUND A. PARKES, M.D.,

ASSISTANT-PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

LONDON:

PRINTED BY C. AND J. ADLARD, BARTHOLOMEW CLOSE.

1849.

AN INQUIRY,

&c.

[The following Inquiry originated in a request made to Dr. Parkes, by the General Board of Health, that he should examine into the evidence which might be derived for or against the doctrine of Contagion, by an analysis of the early cases of cholera in London. It is here published, with the cases abridged, in the belief that the evidence brought forward in it will be acceptable to the profession, and will be found to have an important bearing on the great question at issue.]

I propose to give in this Report the chief facts which I have been able to collect, regarding the early cases of cholera in London; in order that some decision may be formed as to the mode in which these cases originated, whether from a poison emanating from the bodies of other persons labouring under the same affection, from a poison introduced in any other method, or from a poison actually generated in London itself.*

It is universally and truly considered, that the inquiry into the origin of the first cases of an epidemic disease, in any locality, is a necessary preliminary to all other inquiries respecting the origin of future cases. At that period of the epidemic, the question is reduced into as simple elements as we can ever hope to find it in; and the influence of essential antecedents is less obscured than at a later date, by the presence of accidental and unnecessary circumstances.

In order that the terms which I am about to use may be correctly defined, and that it may be clearly understood in what method I am about to investigate this subject, and in what light I regard the general aspect of the great question of the nature of contagious and epidemic diseases, I shall commence with a condensed statement of what I consider to be the most prominent and correct opinions at present entertained by medical men, respecting the diffusion and mode of propagation of those diseases, which are generally allowed to arise from specific and uninterchangeable poisons, and which are capable, under certain conditions, of becoming so prevalent as to be entitled to the appellation of *epidemic*. This statement will be, in fact, a general definition; and when completed, the bearing of the subsequent argument will be at once apparent.

All the opinions of the day, however widely different they appear, may, I believe, be comprised under two separate creeds: that of the strict, and that of the modified contagious theory.

* I assume that cholera, like other epidemic and contagious diseases, must result from the action of a specific agent, rather than from any temporary combination of atmospheric influences. I believe that satisfactory reasons may be given in support of this assumption. Into these I do not wish now to enter; but as far as cholera is concerned, I may refer to the second Volume of the 'British and Foreign Medico-Chirurgical Review' (p. 93), in which this question is shortly discussed.

The *strict contagious theory* I take to be that which refers epidemic diseases to the action of specific poisons, which (it alleges) multiply themselves only during their passage through the animal body. All other reputed modes of increase this doctrine considers to be doubtful or untrue; and it looks upon the external circumstances which surround the animal frame as influencing the efficient cause or poison of the epidemic, only so far as they render the body a more or less fit recipient for its action. As it concludes that the body is the only source from which a fresh supply of the specific agent can be evolved, it deems it necessary that the person, to be infected, should come within the influence of the particles of poison (imparted by contact, diffused in the air, or adhering to clothes) which have been emitted from the breath, surface of body, or excretions of an individual already suffering from the disease, or from the corpse of one who has already died of it. Nor can it be said that this view is otherwise than philosophical; that is to say, it is rested on a foundation of undeniable truth, and its inferences are not obviously inconsistent with the premises it lays down. It sprang naturally, indeed, from a recognition of the great truths, that each epidemic disease originates from a cause which is peculiar to itself, and which is not interchangeable with the cause of another epidemic disease; that, however two or more such diseases may be temporarily combined, they are yet fundamentally distinct; that they are governed by separate laws, and display attributes which manifestly prove their non-identity. And it so happened that when this opinion first took solid root in medical literature, the epidemic diseases which were chiefly witnessed in France, England, and even in Italy, did really spring from poisons whose most potent, and apparently whose only source, was in the very bodies, whether of men or of animals, which were suffering from their effects. The poison grew at the expense of that it tainted. At least, this was eminently the case with smallpox, and with measles and scarlet fever, which were sometimes distinguished, sometimes confounded together. It was also generally presumed to be the case with the Levant plague, which then had received very little study, except in the countries in which it was comparatively an infrequent and transient visitor.

And as in some of these cases the poison was actually tangible, could be procured in substance (though not in a state of absolute isolation), could be carried on the point of a lancet, and transferred from one body to another, it was not really a greater assumption than was warranted by the facts then known, to infer that the more volatile or intangible poisons, which were not inoculable, and the argument for whose existence was indeed founded only on analogical reasoning, did in reality multiply only in the same manner as did those poisons whose existence was demonstrable otherwise than by their effects on the human system alone.

During the last sixty years, however, the study of several diseases imperfectly known to the older physicians has added so many new facts to our knowledge of the several specific epidemic diseases, that the strict contagious theory has been insensibly undergoing alteration, until in the present day it bids fair to become merged in a higher generalization.

The extension of commerce and the military occupation of colonies have made us better acquainted with the several forms of fever, which in the West Indies, and on certain parts of the African coast, have an endemic and a local origin, but which also possess the power, under certain circumstances, of increasing themselves in the human body. The birthplaces of the Oriental plague, Egypt and Syria, have been traversed by the military and civil surgeons of France and England; and the progress of science and of national intercourse has enabled us to note more fully the returns, and to interpret more correctly the attendant phenomena, of that catarrhal fever, which, under the name of influenza, la grippe, &c., so frequently pursues its evanescent course over the greater portion of the world. The more accurate investigation of the present day has, particularly in the last twenty years, opened up fresh points of view, under which the several

forms of fever prevalent in this country might be studied. While the different affections which were formerly described (under the name of continued fever) rather as varieties of a single entity, than as separate and uninterchangeable diseases, are now considered with great probability to be the products, not of a single poison, but of two or more, the influence of these several poisons has been studied, not simply as by the older physicians during their action on isolated patients, but with reference to classes of individuals. Thus it has been discovered that the circumstances under which these classes are placed, exercise an astonishing influence in aiding or counteracting the entrance of the specific agent into the body, and in favouring or nullifying the vigour of the action it there exerts.

The tendency indeed of all these observations has been to show—1st, that the effect of the human body, as a single and uniform element in producing changes in the morbid poisons, and in thereby assisting their diffusion, is by no means so great in these instances as it is in the cases of smallpox and scarlatina; 2d, that it is most probable that these poisons are in the first instance derived from sources foreign and extrinsic to the human frame, and are subsequently more or less frequently propagated and multiplied by means which are also foreign and extrinsic to it; and 3d, that the degree in which they multiply in the human body, and are in this way propagated, varies in the case of each particular poison, and according also to its alliance with other poisons, or to the state of the system through which it passes.

The evidence on which this opinion is founded cannot, of course, be here discussed; but it may be stated that all the later researches on plague, yellow fever, and in a less degree on the fevers of this country, have tended to strengthen this position, both in respect to the amount and to the precision of argument by which it is supported.

To assimilate these observations, and to accommodate their dogma to the new truths which were inconsistent with the rigor of its previous enunciation, the advocates of the strict contagious theory have introduced various changes, and have modified their modes of expression according to the genius and temperament of each individual advocate.

Such advocates advance the following considerations in reply to the statement above given:

In the case of tropical fevers, they seek to break the link which has been made between marsh and contagious yellow fevers, by considering the latter a peculiar disease, and by placing the specific poison which springs from marshes in a distinct category of morbid agents. They deny the validity of the evidence by which the contagious yellow fever (for such undoubtedly exists) is supposed to be traced up from fevers arising from endemic sources, and which evidence, if true, would necessarily place the poisons of all these affections in the same class.

While the influence of epidemic constitutions of the atmosphere, and of external local circumstances, over the fevers of this country, as over the Oriental plague, is admitted, these conditions are presumed simply to augment the susceptibility of the subject, but to have no direct influence on the virus itself. They act on the soil which is to receive the plant, but they do not heighten the inherent force which is normal to the seed. To explain certain special cases, which seem strongly opposed to such a view, and which point to the development *de novo* of the specific virus, the advocates of the strict contagious theory have attributed an extraordinary durability to the poisons of fever and of plague, and a power of lurking within the frame unaltered until the accessory causes have prostrated the force which at first successfully resisted them.

Influenza, again, which is a disease manifestly arising from a special virus, and which attacks so many individuals at once, and is propagated with such rapidity that we can hardly suppose this to occur through the influence of the human body, has not yet been fairly considered by the strict contagious theory. If it

is not allowed to modify this theory, it must be shut out altogether from this class of morbid poisons, although in many respects it is an apt representative of the class.*

The strict contagious theory was considered to derive its strongest proofs from the fact, that those persons only suffered who came in contact or were in proximity with the sick. Lately, however, a section of this party has questioned the possibility of setting bounds to the diffusion-distance which a poison can traverse before its powers are destroyed. Whilst the older contagionists never assigned a greater distance than a few yards, this new opinion (which still looks on the human body as the *fons et origo mali*) doubts whether the poison may not float for an indefinite distance through the atmosphere, the condition of which may favour or impede its volatility and rapidity of diffusion. Consequently as regards this party, the evidence which is considered decisive against contagion is not decisive against them, or at least they do not consider it so; although it may be demanded how (independent of inoculation) they could prove that a single morbid poison was ever reproduced in its passage through the body, unless by the evidence derived from the fact that those about the sick are more liable to the disease than those who are not?

Thus, at the present time, two great opinions seem to divide the medical world, and to be contending for the mastery. Each opinion has the same foundation—each looks on epidemic diseases as produced by specific and unintercheangeable agents—each admits that some of these agents evidently propagate themselves chiefly, or possibly solely, by means of their action on the body. After this, their paths divide. What they both assume to be true of certain specific agents, the strict contagionist affirms to be true of all of them; the modified contagionist affirms it to be true of certain of them, only in a limited and conditional sense.

The latter party, in seeking to determine the mode of non-contagious propagation, considers with the greatest attention the media external to the human body, which surround the particles of any special poison. It regards the humidity and temperature of the air, its purity, its electrical condition, the weight of its column, and the movement of its masses. It questions what may be the exhalations from the soil, from decaying substances, from all the various natural or artificial peculiarities which vary the surface of the ground. In all these circumstances it sees a twofold action—an action upon a virus introduced among them—an action upon a human frame submitted to them. It seeks to determine what relative assemblage of these conditions is most favorable to the spread of a poison; it believes that, under its favouring conditions, some poisons introduced *ab externo* may augment by reproducing themselves. It goes even farther than this; in certain cases, it sees, in an intense concentration of these several circumstances, a development *de novo* of that specific poison, which is proper to that particular assemblage of conditions. On the banks of the Ganges it witnesses the cholera poison spring into existence; in the swamps of Batavia, the malignant intermittent passing into remittent; on the river marshes of Western Africa, the deadly remittent, from which springs the epidemic yellow fever; among the effluvia emitted from our great cities, or among the miserable hovels of a starving nation, the fever-poisons of this country; among the Fellahs of Egypt, with their peculiar rites of sepulture, and with their pernicious customs of social life, the poison of bubo-plague. It attaches, also, much importance to the consideration of the occasional, and even periodical, augmentations in the

* If Influenza arises from any peculiar allotropic condition of the oxygen of the atmosphere produced by electrical vicissitudes, and does not originate from any agent allied to the ordinary contagious agents, it may be dismissed from consideration; but, at present, the production of ozone during influenza epidemics, the capability of ozone to produce the peculiar symptoms, and the other questions which would prove a connexion between the two, are all matters of the merest speculation. On the other hand, the general laws of influenza, the peculiarities of its course, and even its occasional irregularities, render its separation from the poisons of cholera, of some forms of marsh fever, and perhaps of pertussis, almost impossible.

vigour of the specific poisons, believing that these are partly dependent on influences exerted on these poisons by "epidemic constitutions" of the atmosphere.

While the followers of this creed admit that some or all of these specific poisons may be truly contagious—that is, may be reproduced by the human system—they are disposed to limit this mode of propagation, and to admit it no farther than it can be proved by actual evidence. They refuse the argument from analogy, that because smallpox is manifestly augmented by the body, any other disease is necessarily subjected to the same influence. In the case of several poisons, they advance evidence to prove that this mode of increase is exceptional, and comparatively unimportant.

Finally, in conformity with their creed, the supporters of this opinion seek to arrest the progress of epidemic diseases by destroying the external local conditions, which, with perhaps some general atmospheric conditions, are assumed to be the necessary pabula for the poison, and by removing the internal states of the system which predispose to the reception of the cause.

The strict contagious theory, on the other hand, considers these opinions as overstrained; it looks primarily to the human body as the focus for the poison, and to the external conditions as simply predisposing and accessory causes. It questions the doctrine of origin *de novo*, and contenting itself with the fact that the poisons exist, doubts whether it is possible to trace them to their origins. It avoids, indeed, this question as beyond its reach. It separates such poisons as have a determined local origin from the true contagions, and denies that they can ever become epidemic. It places its great reliance, not on sanitary preventive measures, though for the reasons above given it does not deny the wisdom of these, but on isolation and quarantine. By preventing proximity and exposure, it deems that the poison must become extinct, if it meets with no animal frame in the structure of which it may increase. Consequently, to push this view to its legitimate conclusion, a perfect system of quarantines would secure to a nation complete immunity from all epidemic diseases, except those which were previously located within it. To be perfectly consistent with itself, this theory must restrict the action of the so-called "epidemic constitution" to a simple effect on the recipient of the poison.

It will be seen at once that these two opinions are in one sense opposed to each other, in another are even compatible. The former includes the latter, or might do so; for it admits the human body as a possible multiplying source, in the case of all the poisons. It only demands that the proportion of cases which increase in this way, shall be proved from evidence similar to that which proves the production in cases from other sources. And according to the peculiar mode of regarding evidence in each individual mind, the supporters of this opinion are divided into various subsections, who all more or less incline, but none of whom actually reach (except perhaps as regards the case of influenza), the extreme of non-contagion. If in some cases (as in influenza) the evidence of the increase of a poison in the body is so defective as to cause doubts whether it ever occurs, still it is admitted that such negative evidence is always to be received with the reservation, that peculiar conditions may yet remain to be discovered or noted, which may really have the effect of temporarily increasing the influence of this particular source of the poison.—The strict contagious theory, on the other hand, is not capable of this expansion and amalgamation. It repels, and indeed necessarily and completely excludes, everything which is not comprised within its definition, or is not capable of being added to that definition without impairing its fundamental tenet. It cannot, in fact, abandon its doctrine that the body is the only multiplying agent of each poison, without entirely surrendering the character which stamps its individuality. Immediately that a partisan of this opinion allows that the impure and fetid atmosphere which hovers over a fever-locality, can generate a single particle of fever-poison, or can cause a particle thrown off from the body to generate another like itself,

he has undermined his theory; and the question between the relative propagating powers of the body and the surrounding media, becomes merely a question of degree.

Such are, I believe, when stated with the utmost brevity and simplicity, and divested of all qualification not absolutely essential, the two great opinions of the day respecting the causes of epidemic diseases. There can be no doubt that in a scientific and a commercial point of view, the debate is one of great importance, and it seems probable that it is now gradually approaching a final solution.

There is one disease to which I have avoided making any but the faintest allusion, but the investigation of which has contributed essentially to the reception of the modified contagionist view. Asiatic Cholera belongs manifestly to the most strongly marked order of epidemic diseases. Its symptoms are remarkably precise and recognisable, and their course and mode of evolution are determinate; its poison propagates itself at intervals over countries which it afterwards abandons for a series of years. There is, therefore, in these countries nothing of the endemic or the local about it.* It sweeps, like an atmospheric wave, over vast tracts of the earth, or possibly itself brings or induces the epidemic constitution which permits its poison to be continually generated. There is little doubt but that it nosologically belongs to the same order of diseases as smallpox or typhus—viz. that it is produced by a definite epidemic poison entering the person *ab externo*.

As, therefore, it is a disease evidently not indigenous to this country, as it comes to us from without, and merely borrows force from the elements of increase it finds here, whatever these may be, it affords a most convenient opportunity for applying the two theories above described, and for observing which best interprets its phenomena. And as every point which bears on Asiatic Cholera bears also on the general subject of contagion, it is of the greatest importance to render all evidence as correct as it is possible to make it.

In the following Inquiry, I do not intend to enter on the general subject. I am simply desirous of placing on record the evidence respecting the first cases of cholera in London, and to inquire particularly into the possibility of these cases having arisen from contact or proximity with persons already diseased.

The strict contagionist theory has always considered that strong evidence in its favour was to be obtained from a study of the early cases. It claims to be always able to point out the channel of introduction, and to trace the first steps of the malady; afterwards, when the sick have become numerous, it considers that persons may be exposed to emanations, and may sicken, without being aware that they have been so exposed. At this late period, therefore, the proofs of contact or proximity cannot be obtained; and negative evidence loses its value.

The first twenty-five or thirty cases are then, in this point of view, most important. Did they arise together, or near each other? Were they exposed to sources of contagion, from which the other inhabitants of the district were exempt? Can each successive case be traced to a prior case, until the patients are too numerous to be followed up?

If these questions can be answered in the affirmative, it must be conceded that the strict contagionists have carried their point; if they are not so answered, then the observer has to seek for the cause of the early cases in other directions.

It is necessary in examining evidence on this point to adopt two precautions.

1. Every reputed case of the disease must be known.

* I assume here, that common English cholera is an entirely different disease from Asiatic cholera. This point is not fully ascertained; but at present the weight of the argument is against the identity. But, even if we admitted that every year sporadic cases of true cholera occurred in London, still the bearing of the argument in the text would be unaffected. The epidemic has certainly on two occasions travelled regularly to us, and has not arisen into activity among us independently of an external and peculiar influence.

2. Every reputed case must be inquired into, and its exact nature determined; the loose accounts of by-standers and non-professional persons not being received as credible evidence.

With regard to the first of these precautions, there is little doubt that the cases I am about to detail comprise all, or nearly all, the first cases in London. Almost all the early cases in London were reported to the General Board of Health, and were immediately investigated by means of their agents; and if any early case was not so reported, and proved fatal (which in the case of cholera it would probably do), it would be found in the Registrar-General's returns.

As to the second precaution, I have to state that the General Board, as already said, having, by means of competent agents, inquired into all the cases, no loose hearsay evidence will have even to be discussed. I have myself carefully examined the records of all these cases; in many instances I saw the patients themselves; and any case which seems doubtful will be left out of the argument, unless its admission would seriously modify a conclusion come to in its absence, in which case I shall discuss the point both with and without it.

It has been stated in some of the medical journals, that several cases, very much resembling Asiatic cholera, occurred in London during the summer and early autumn months of 1848. The only decided case of this kind which I can find, however, is one reported by Mr. Haden, in the 'Medical Gazette' for October 13, 1848, to which journal I must refer for its details.

Case I. This case, at the time of its occurrence (July), was considered by Mr. Haden to bear so strong a resemblance to Asiatic cholera, that he felt inclined to believe it to be an advanced case of the approaching epidemic of that disease, and not to be simply a case of the common English or bilious cholera. If it was, however, a severe case of common sporadic cholera, it becomes a question by what symptoms it can be distinguished from the epidemic disease. The assemblage and concatenation of symptoms were altogether similar to those of Indian cholera. The evacuations were like conjee water, and without bile; there was the usual failure in the circulation, with its accompanying symptoms; there were the copious sweats and the suppression of urine; there were the usual periods of the disease, vomiting and purging, soon attended by cramps, a subsequent superadded cold stage, and a period of reaction. It is clear that had this case occurred at a time when epidemic cholera was prevalent in the neighbourhood, it would unhesitatingly have been considered a marked example of the disease.

As, however, I am anxious to avoid all possible fallacies, I shall put aside this case, or rather shall not use it in the present inquiry. Whatever its nature, it cannot be attributed to any poison brought near the patient by an infected person. There had been no case of cholera in the neighbourhood, and no one subsequently suffered.

Case II. The second case was reported to the Board of Health by Mr. Howell, of Wandsworth. The patient, a man aged 41, a coalwhipper by trade, living at Spencer's court, Waterside, Wandsworth, was attacked at 11 p.m., July 31, with violent purging and vomiting, and severe cramps; these symptoms were speedily succeeded by blueness, lividity, and coldness of the surface and tongue. He remained in the cold stage for forty-eight hours, and then passed through slight consecutive fever.

Although the symptoms above given are well marked, and are detailed by a practitioner, who it is believed was well acquainted with the symptoms in the epidemic of 1832, still, as cases similar to this one are said by some practitioners to be of annual occurrence in London, and in other parts of the country, it will be advisable to omit this also from the present inquiry. It may be mentioned, however, that in this very locality a considerable number of cases of undoubted Asiatic cholera occurred during the last month of 1848, and the first month of the present year.

Case III. The third reputed case in London, with which I am acquainted,

occurred in the practice of Mr. Fairbrother, of Southwark, on the 16th of September. A man, named John Dean, aged 53, resident at No. 5, Lion street, New Kent road, died after a few hours' illness. An inquest was held, as there was a suspicion of poisoning; and Mr. Fairbrother gave it as his opinion that the patient died from Asiatic cholera.

Although I could not undertake to say positively what this case may have been, and although Mr. Fairbrother, who saw the case, is more likely to be correct than any one who had not that advantage, it does appear to me that some doubts may be entertained whether it was really Asiatic cholera. Without, however, deciding on this point, more particularly as I am not aware whether a post-mortem examination was made, it will be advisable not to use this case. It was not supposed to be traceable in any way to contagion, and therefore its omission is no prejudice to the contagionist argument, but rather the reverse.

Case IV. The fourth case of cholera which occurred in London, was the following.

The Elbe steamer left Hamburgh on the 22d of September, and arrived in the river on the 25th. A seaman, named John Harnold, left the vessel, and went to live at No. 8, New lane, Gainsford street, Horsleydown. On the 28th of September he was seized with symptoms of cholera, and died in a few hours. It is stated in a letter to the General Board of Health, from Mr. Russell, who attended the patient, that all the characteristic symptoms of Asiatic cholera were present. Mr. Bowie, who inquired on behalf of the Board into the particulars of the case, corroborated this statement. This may then be considered as an undoubted case of cholera.

It becomes a question of some interest to determine whether this man became infected at Hamburgh, where cholera was raging at the date of the sailing of the vessel, or in Horsleydown itself. Some support is given to the first opinion by the fact that another probable case of cholera had occurred on board the steamer at sea.

Mr. Bowie's Report gives the following particulars of this case:

"On the voyage from Hamburgh to London, and about forty miles from Lowestoft, the second engineer, who had been long in ill health, died of an attack very much resembling cholera. Immediately after death, the body was placed in a box which was closely nailed down. All the clothes and bedding were thrown into the sea. When the vessel arrived at Gravesend, she was detained about six hours, during which time the quarantine doctor came on board, and granted permission for the body to be conveyed to London to be delivered to the friends, which was done at St. Katharine's Docks by the first engineer."

It may be questioned, however, whether the statement of the captain, "that the disease very much resembled cholera," is of much value, particularly when it is remembered that this man had been for some time in bad health. But if this were a case of cholera, it would be important, as proving satisfactorily that the crew of the Elbe had been exposed at Hamburgh to the influence of the choleraic poison. The *certainty* thus afforded us that John Harnold was exposed in that locality to the action of the cause, would necessarily increase the probability that he was there infected. It may be said also that the time between the departure from Hamburgh, and the occurrence of the disease in London (six days), was within the limits of the incubative period; though certainly it is rather a long time.

On the other hand, there is little doubt, from the facts to be presently adduced, that Harnold, when he entered London, entered a place where cholera was commencing to prevail; as cases, contemporaneous or nearly so with his own, occurred in two or three distant parts of the metropolis. And as it seems to be a peculiarity of this singular disease, that journeys from infected places increase the liability of the system to suffer from its cause, it may be doubted whether it is not as likely to be the fact, that this man became infected during the three days that he was on shore, before the disease appeared.

As far as the argument is concerned, however, it is of no importance which view is the most correct. If the disease was imported thus from Hamburgh, it did not spread in Horsleydown. Two days subsequently, indeed, Mr. Russell was sent for to a patient in the same house, who fancied he had cholera; but on examining into particulars, it turned out that the individual in question had been greatly alarmed at the death of the seaman, and was suffering more from the effects of fear than anything else. He was quite well in a few hours. No other person was taken ill in the house or immediate neighbourhood; although if the second case had not been inquired into, a vague story of communicated disease might have arisen in the neighbourhood.

Case V. Within 24 or 36 hours after the death of John Harnold, an undoubted case of cholera occurred in another part of London, in Lambeth, which is distant about two miles from Horsleydown. The patient was a man named John Murphy, aged 22, resident at No. 26, Lower Fore street, South Lambeth. He was a labourer in the adjoining gas-works, but out of employ, and badly fed. On the 30th of September he had been with a cousin to Kensal Green, on the Harrow road. He appeared in the morning to be perfectly well; but was first seriously attacked at 11½ p.m., and died at 8½ on following morning (Oct. 1).

The correctness of the account given to me by the friends of the patient, when, by order of the Board, I inquired into this case, was corroborated by Mr. Thomson, of Lambeth, who attended the patient, and who at once considered the case to be one of Asiatic cholera. After death this man became very stiff. When I inspected the house five days after his death, he was still unburied. I removed the lid of the coffin, and found that, though decomposition was far advanced, and the face was swollen and black, the cadaveric rigidity was yet well marked in the extremities. That this was a genuine case of Asiatic cholera, I have no hesitation in affirming.

At the same time that this case occurred in Lambeth, the first of a series of cases appeared in a small court in Chelsea, situated on the opposite side of the river from Lambeth, and still farther removed from Horsleydown. In the course of a few days, six cases had occurred in this narrow court, which is situated close to the river, and is one of the most wretched and dirty localities in Chelsea. These cases were attended by Mr. Keen, of Chelsea, and were also visited by numerous medical men, who were all satisfied that they were undoubted examples of Asiatic cholera.

Case VI. Richard Cook, aged 10. This case, the first of the series just referred to, was certainly very mild in character; yet the symptoms, and the occurrence of better-marked cases immediately afterwards in the same locality, seem sufficient to establish the fact of its being one of cholera.

Case VII. The next case occurred in a locality widely removed from any of those formerly described, viz. in a court leading out of Fleet street. The subject of it was Jane Langham, aged 27, who resided at No. 3, Harp court, Fleet street. She was visited by Mr. Digby, of Fleet street; who, when first summoned to the patient, considered her to be in the collapsed stage of Asiatic cholera; and his diagnosis was fully borne out by the general symptoms.

Case VIII. The next occurred in a part of the town several miles removed from any of the former localities.

Owen Jones, æt. 60, a convict on board the Hulk Justitia, lying off Woolwich, was admitted into the Convict Hospital-ship Unité, also lying off Woolwich, at 7 p.m., October 2d. It was ascertained that he had eaten his dinner as usual, and that soon afterwards he appeared chilly and indisposed. At tea-time he had spasms in the arms, and his shipmates becoming alarmed at this, and at the cold listless state into which he seemed to be falling, sent him to the hospital. When admitted he was evidently in the collapsed stage of cholera, and died after seven hours.

I was informed by Mr. Dabbs, R.N, on duty on board the Unité, that this

case was undoubtedly one of cholera. It was apparently a case of the most malignant type, in which fibrine is poured into the intestinal canal with very little of the water and soluble salts of the blood. The diagnosis of the case was also confirmed by the speedy occurrence of other cases of cholera, presenting symptoms very similar.

Case IX. The next case occurred in a house three doors removed from the one in which Case V had occurred.—James George, aged 40, residing at No. 29, Lower Fore street, Lambeth; a butcher's carter.

On Wednesday, October 4th, he got up at his usual time, viz. 4 a.m.; and immediately complained of pain in the bowels; he passed suddenly a very loose and offensive evacuation. Before 5 o'clock he had two or three more evacuations, but certainly not more than this; he did not vomit or complain of noise in the ears, giddiness, or faintness. His wife was so accustomed to see him affected with bowel complaint, that she paid little attention to his symptoms, and, after taking a dose of brandy and rhubarb, he went to his work in the city. On his way to Newgate Market, he was attacked in St. Paul's Churchyard with violent vomiting and purging; the latter symptom continued until his admission into St. Bartholomew's Hospital at half-past 8 a.m., where he died at 9 p.m., having been ill seventeen hours from the period of the first stool.

The physicians at St. Bartholomew's were unanimous in considering this an undoubted case of Asiatic cholera, and reported it as such through the treasurer of the hospital to the Secretary of State.

It should be mentioned that this man was not acquainted with Murphy (Case V), nor with any one in the house (No. 26) in which Murphy lived. If the two men had been at all in contact, during the time Murphy could be said to labour under the disease, it must have been in the evening of Saturday, Sept. 30, when Murphy was returning from Kensal Green, and may be supposed to have been already infected, although the symptoms had not declared themselves. But it is unlikely that a mere casual encounter in the street, if such occurred, could have infected the patient George. It is more probable that, if he derived the disease from Murphy, it must have been through the particles of poison floating over the neighbourhood. Even with this supposition, the period of incubation is rather prolonged (three to three and a half days). Such a supposition becomes probable in this case, only when its probability has been shown on other grounds.

Case X. A daughter, aged 6, of the above patient (James George) was attacked on the same day (Oct. 4), and nearly at the same hour, with diarrhœa. At half-past eight she was conveyed to St. Bartholomew's Hospital, and admitted under Dr. Barrows, who considered it to be a decided though mild attack of Asiatic cholera. She ultimately recovered.

Case XI. The eleventh case occurred in the same court, in Chelsea, in which the sixth case occurred, and in the person of the mother of that patient Susan Cook, aged 40, residing at No. 7, White Hart court, Duke street, Chelsea.

This patient was seen by Mr. Keen on the 4th October; at that time she presented symptoms similar to those of her son in an exalted degree. She continued to present nearly the same symptoms till the 6th, when she began to improve. She passed urine on the 7th, and fæculent motions on the 8th. She eventually recovered.

Case XII. The twelfth case occurred in Horsleydown. A man, aged 77, residing in Horsleydown, was attacked at 5 a.m., Oct. 5, and died at 8½ p.m., 15½ hours after the first stool. Dr. Greenwood, who reported the case to the Board, had no doubt that it was a pure case of Asiatic cholera; and this opinion was shared by Mr. Bowie, who made inquiries into the case.

This patient lived in Horsleydown, and consequently may casually have been in contact with John Harnold (Case IV), who died between six and seven days before. I am not aware at what distance his dwelling was situated from the

lodging of John Harnold, but I believe it was not in the immediate neighbourhood. The reporters do not say a word as to any possible connexion between this case and that of John Harnold.

Case XIII. The thirteenth case occurred in an altogether different locality, viz. on board the Dreadnought Hospital-ship, lying off Greenwich.

Robert Gordon, aged 16, a sailor, was admitted on board the Dreadnought, in the beginning of September, with the sequelæ of smallpox. A few days after his admission he was attacked with diarrhœa, and continued for about a fortnight to pass frequently rather copious frothy yellow stools. These, however, were gradually diminishing in number, and it was intended to discharge him from the hospital. On the morning of October 5, he was employed in assisting the nurses, and appeared as well as usual; but about the middle of the day the characteristic symptoms of Asiatic cholera presented themselves, and he died in about twenty-four hours.

It will be observed that this case occurred in a patient under treatment. It was not brought into the Dreadnought. By the kind permission of the officers of the Dreadnought, I took the opportunity of inspecting the admission-book, and learned that no sailor arriving in a ship from any port in or near which cholera was or had been prevalent had been admitted into the Dreadnought, for any complaint whatever, for some considerable time. The disease, therefore, could not have been brought on board by the clothes of some non-infected individual arriving from an infected ship.

Case XIV. John Healey, aged 25, a cousin of John Murphy (Case IV), and living in the same room with him, was taken ill at 8 a.m., Oct. 5. This man was also a labourer in the gas-works, and out of employ. He was a very temperate man, but for some months had lived very badly, being sometimes thirty-six hours without food. He had never been subject to bowel complaint. He was first attacked on the morning of October 5; I saw him the next morning at 9 a.m., and he then presented all the signs of a man about to rally from a mild attack of cholera; he subsequently passed into the stage of consecutive fever, and died on the sixth day with coma and a black tongue.

Case XV. Joseph Hill, aged 5, residing in White Hart court, Duke street, Chelsea, either next door or two doors removed from the house in which Cases VI and XI occurred. At 12, noon, Oct. 5, he was seized with vomiting, and when visited two hours subsequently, had passed into a state of deep collapse, cold and pulseless. He was not purged till a short time before death. He died at half-past 7 p.m. the same day.

Case XVI. On the same day, Oct. 5, two cases occurred in Spitalfields, and are reported by Mr. Hart in the 'Lancet' (Oct. 14, 1848, p. 419). The subject of the first was Mary Ann C., aged 11, residing at Wilson's place, Spitalfields. She was suddenly attacked shortly after 8 o'clock, a.m., Oct. 5, and after being in a state of collapse for some hours, reaction came on, and she rapidly recovered.

Case XVII. A sister of the above, aged 3 years, was attacked at the same time, and presented symptoms of a similar kind, but of less severity. On the 8th instant the mother of these two children (Case XXVIII) was attacked with undoubted cholera, and died in twenty-eight hours.

Case XVIII. William Cook, aged 40, the father of Richard Cook, (Case VI) and the husband of Susan Cook (Case XI), residing at No. 7, White Hart court, Duke street, Chelsea, a man of drunken habits, had diarrhœa on the 6th October, having had occasional looseness during the previous week. At 6 o'clock p.m., October 6th, he was seized with genuine Asiatic cholera. He died at 2 a.m., on the 8th, having been ill about thirty-six hours.

Case XIX. William Cook, aged 14, son of the above, residing in the same house, was seized at 6 p.m., October 6th, with vomiting and purging; and was seen at 8 a.m., October 7th, in the cold stage of Asiatic cholera. He died at 1½ p.m. On examination after death, the appearances usual in cholera were found.

Case XX. Elizabeth Morris, aged 14, residing at No. 7, White Hart court, Duke street, Chelsea, in the same house as the family of Cook. She was attacked with diarrhœa on the 6th of October. This continued all day; at night she began to vomit rice-water fluid, and had cramps in the legs. When first seen at 8 a.m., October 7th, she was in the cold stage. She died at 1 a.m., October 8th, having been ill between thirty-six and forty-eight hours.

Case XXI. John Rutherford, aged 47, convict on board the Hulk Justitia, lying off Woolwich; admitted into the Hospital-ship at midnight October 6th, in a state of decided collapse. He died at twenty minutes to 10, October 7th, having been ill thirteen hours.

Case XXII. ——— Rabett, aged 30, a sailor, had been for several months a patient on board the Dreadnought Hospital-ship, with dysentery and pleurisy. He was, however, convalescent, and was about to be discharged at the time he was attacked with cholera, the first symptoms of which showed themselves on Friday night, October 6th. On the 9th reaction seemed to commence, but the bladder remained empty; he became drowsy, and this drowsiness deepened into coma. He then presented the usual symptoms of that form of consecutive fever which is complicated with non-elimination of urea, and died about eight days from the period of attack.

Case XXIII. James Bigwood, aged 44, convict on board the Justitia Hulk, lying off Woolwich. Admitted at 3 a.m., October 8th, in the state of collapse; and died at 2½ p.m. on the same day.

Case XXIV. Louisa Hill, aged 10, sister of Joseph Hill (Case XV), residing in White Hart court, Duke street, Chelsea. Attacked in the morning of October 8th. She ultimately recovered.

Case XXV. James Paterson, middle-aged, a sailor, arrived from Shields on the 2d of October. Since then, had lived on board ship in the Pool. During October 7th, he suffered from watery diarrhœa, at 11 p.m. he had vomiting and cramps of moderate intensity. At 9 a.m., October 8th, when admitted into the Dreadnought, he presented all the characteristic symptoms of cholera, and died at 8 a.m., October 9th. It will be observed that this man had been five days in the Thames, and probably eight or ten days had elapsed since he left Shields, when the premonitory diarrhœa commenced. No other case had occurred on board the ship.

Case XXVI. A man aged 44, residing at No. 7, Prescott street, Clapham Common. Although this case occurred after some of those which follow it, I give it here, as it is an interesting case.

On Sunday, October 8th, this man was as well as usual, and his wife stated that he had no bowel complaint. The patient himself, however, stated to Mr. Greenwood, of Clapham, who attended him, that he had suffered for two or three days before his death from relaxation of the bowels. His wife was not cognizant of this, and states that if the relaxation had been great she must have known it. On Monday, October 9th, he was as well or even better than usual. He went to his work about 9 a.m., and was employed for an hour and a half in emptying and repairing a drain, some distance from the house. The drain was a small one running from a sink. Another man opened the drain, and the subject of this Report carried the contents in a pail to the place where they were to be thrown. He had been accustomed to empty privies and drains, and did not on this occasion complain of any particular effluvia. On this day he took for dinner beef and bread, and for tea bread and butter. He took no supper. He was not purged during Monday night. He was quite well on Tuesday morning. The wife does not think that the bowels were at all relaxed. He made a very hearty breakfast, and went to his work. He came in at 1 o'clock to dinner, having been affected since 9 o'clock with painless diarrhœa. Soon afterwards the symptoms of collapse appeared, and he died between 9 and 10 the same evening, having been ill about twelve hours.

This case was one undoubtedly of Asiatic cholera; the nature of the symptoms

and the rapidity of their evolution seem to prove this; and in addition, the patient was visited by several of the medical men in the neighbourhood, who were unanimous in their diagnosis.

It becomes a question whether any predisposing or accessory influence is to be attributed to the effluvia from the drain, which the patient was engaged in emptying, on the day previous to his death. There was a doubt as to whether the patient really had suffered from any diarrhœa which might be considered premonitory, previous to this occurrence. If so, the effect of the drain would have been purely accessory, if not absolutely negative. On the other hand, if there had been no premonitory diarrhœa, and if these effluvia had a more active influence, of what nature was it? Did they render the frame of the individual obnoxious to the effect of the already present cholera-poison? Did they permit to be generated *de novo*, under the influence of some general occult atmospheric condition, the specific poison, which then happened to meet with an individual susceptible to its influence? To this notion there are many objections, of which I will only adduce three: 1. Such a development *de novo* has yet to be proved. 2. The development in so short a time (90 minutes) is unlikely. 3. If the specific poison could be formed so readily from drains under the influence of a peculiar choleraic atmosphere, cholera ought at this time to have been much more prevalent than it was, as in London there must have been many localities affording similar effluvia in as great abundance and concentration as those to which this patient was exposed. It is of course understood that the effluvia of the drain *per se* were quite incompetent to produce cholera,—a disease springing from a specific poison.

Altogether it seems most probable that the drain had no special effect, or that if it had any effect it must have been simply as an assisting accessory cause, posterior in point of time to the reception of the true choleraic poison into the body. At the same time there are still great difficulties about this case, which will probably remain unexplained till our knowledge of cholera is much greater than it is at present. The patient resided near Clapham Common, in a part of the town by no means overcrowded, not perfectly drained it is true, but still not very deficient in this respect; in a house not densely occupied, scrupulously clean, and dry; he lived tolerably well, and had not been exposed to weather. There was no cholera in the neighbourhood; he had not, as far as could be learnt, been out of the neighbourhood or been in contact with any infected person. In this neighbourhood also, even up to this time (Feb. 1849), there has been no second case of cholera, or at least none is known to the Board of Health. And yet this man, unsurrounded by the specific poison, unencompassed with the accessory causes which give that poison local power, died of malignant cholera in one of its severer forms. At least I do not see how the evidence to this effect can be resisted. Was there then some extraordinary susceptibility, some intense predisposition to the disease, which caused this man to fall a victim to a degree of intensity of poison quite inadequate to affect any second person, not so intensely liable? To this question it is at present impossible to give an answer; since we do not know the exact nature of the predisposition, nor, of course, the amount and extent of its influence.

The following cases, into the details of which I need not enter, but which were all unquestionable, complete the number which I consider sufficient for the present inquiry.

Case XXVII. A sailor, named Rouse, was admitted from the Pool into the London Hospital, on the 8th Oct., with undoubted cholera. He passed through the cold stage, and died of consecutive fever.

Case XXVIII. Mother of Mary Ann C., Spitalfields, Oct. 8th.

Case XXIX. A man, named Thomas Baker, residing in Friendly place, South street, Long lane, Bermondsey, was attacked on the morning of Oct. 9th, and died at 2 a.m., Oct. 10th.

Case XXX. David Davison, sailor, from Hartlepool, taken ill in the Pool, at 4 o'clock a.m. Oct. 9th; died in the Dreadnought at half-past 5 p.m. Oct. 9th.

Case XXXI. A convict in the Justitia Hulk, Woolwich, admitted Oct. 10th; died Oct. 10th; seven hours and a half ill.

In addition to these cases, the following appear in the Registrar-General's Report. Cases of cholera are reported in August and September, but these were examples of common autumnal cholera—they did not exceed the average; and the Registrar-General states, in the Quarterly Report for the quarter ending September, 1848, that, "so far as the returns down to the end of September go, there is no trace of the epidemic of cholera in England." (p. 3.)

In the week ending September 30th, 4 cases of cholera were reported, the weekly average, as estimated from five years' returns, being 7.

In the week ending Oct. 7th, 13 cases of cholera were returned, the weekly average of five years for this week being only 1.

These 13 cases are thus stated in the Registrar-General's weekly Report:

"Fatal cases of cholera returned in the week ending Saturday 7th. (All are certified by medical attendant.)

"1. In Old street (subdistrict), St. Luke's, at No. 39, Rahere street, wife of a gentleman, æt. 59, 'disease of the bowels, simulating Asiatic cholera' (38 hours' duration).

"2. In south subdistrict, West London, F. 27 years, 'cholera' (13 hours' duration).

"3. In St. Bartholomew's Hospital, West London, M., about 40 years, 'Asiatic cholera.'

"4. In town (subdistrict), Bethnal Green, at No. 4, Cheshire street, a weaver, F., 21 years, 'enlargement of the heart' (12 months' duration), cholera spasmodic (12 hours).

"5. In Spitalfields, Whitechapel, M., 23 years, 'cholera' (12 hours' duration).

"6. In Whitechapel, north, a girl, 4 years, 'English sporadic cholera' (7 days' duration).

"7. In St. Paul (subdistrict), St. George's in the East, M., 38 years, 'cholera' (2 days' duration).

"8. In Mile End Old Town, Lower Stepney, M., 47 years, 'cholera' (36 hours' duration).

"9. In Lambeth Church, second part (subdistrict), at Orsett street, daughter of a chairmaker, 11 months, 'cholera' (3 days' duration), convulsions (1 hour).

"10. In Rotherhithe, a boy, 11 years, 'cholera' (19 hours' duration).

"11. In same subdistrict, F., 38 years, 'cholera' (19 hours' duration).

"12. In same subdistrict, a girl, 2 years, 'cholera' (2 days' duration).

"13. In Greenwich, west (subdistrict), F., 37 years, 'cholera' (4 days' duration)."

Of these cases, two (Nos. 2 and 3) appear among my list; eleven are distinct cases. It is quite uncertain how many of these were examples of Asiatic cholera; choosing, however, the severest cases, we may suppose four (viz. Nos. 5, 8, 10, 11) to have been of this type. Some of the others may have been so, but of course this is still more doubtful. We shall presently see that their omission will not seriously prejudice either argument. I may remark, that the cases I have given, or rather those of them which were fatal, are registered among the later weekly returns of the Registrar-General.

I believe that no one who examines the evidence for himself, will have any difficulty in admitting, that the range of cases now reported, commencing with that of John Harnold (Case IV), were all examples of true cholera.

Taking this for granted, the accompanying table will show the next point in the inquiry. The table is divided into four columns:—in the first is the date of attack; in the second, the name of the individual; in the third, the locality he inhabited; and in the fourth, the infected person, if any, with whom he had been in contact or proximity.

Date.	Name.	Locality.	In contact or not with a person labouring under cholera.
Sept. 28	John Harnold	Horsleydown	With the mate of his vessel, who was said to have died of cholera—case not certain; with cholera patients in Hamburgh?
Sept. 30	John Murphy	Lambeth	Not in contact with any cholera patient
	Richard Cook	Chelsea	Not in contact with any cholera patient
Oct. 1	Jane Langham	Harp court, Fleet street	Not in contact with any cholera patient
Oct. 2	Owen Jones	Justitia Hulk, Woolwich	Not in contact with any cholera patient
Oct. 4	James George	Lambeth	Not known to be in contact with any cholera patient
	Daughter of above	Lambeth	In contact with her father
	Susan Cook	Chelsea	In contact with Richard Cook
	An old man	Horsleydown	Not known to be in contact with any cholera patient
	Robert Gordon	Dreadnought, Greenwich	Not in contact with any cholera patient
Oct. 5	John Hally	Lambeth	In contact with John Murphy
	Mary Ann C.	Chelsea	Probably in contact with Richard and Susan Cook
	Sister of above	Spitalfields	Not in contact with any cholera patient
	William Cook	Chelsea	With her sister Susan Cook
	Son of above	Chelsea	In contact with his father and mother
Oct. 6	Elizabeth Morris	Chelsea	With the above
	John Rutherford	Justitia, Woolwich	With Owen Jones
Oct. 7	— Rabett	Dreadnought, Greenwich	With Robert Gordon
	James Bigwood	Justitia, Woolwich	With Owen Jones and John Rutherford
	Louisa Hill	Chelsea	With Joseph Hill
Oct. 8	James Paterson	Pool	Not known to be in contact with any cholera patient
	Rouse (a sailor)	Pool	Not known to be in contact with any cholera patient
	Mother of Mary Ann C.	Spitalfields	With her daughters
Oct. 9	James Baker	Bermondsey	Not known to be in contact with any cholera patient
	David Davison	Pool	Not known to be in contact with any cholera patient
	A convict	Justitia, Woolwich	With the convicts mentioned above
	A man, aged 44	Prescott street, Clapham	Not in contact with any cholera patient.

It thus appears, that, from the 28th September to the 10th October (twelve days), 28 cases of cholera occurred in ten different localities, situated, not in proximity, but in various and remote divisions of this immense city.

The inhabitants of these localities do not appear to have held any intercourse with each other; and in no single instance could it be discovered that the *first* person sick in any particular locality had been in contact or proximity with another individual previously diseased, in another locality.

The following considerations support this assertion:

1. Such contact was in no case alleged to have taken place by the patients themselves, nor by their relations. John Harnold had been in London only three days before his death, and it is not known how he was employed during those three days; but most probably he was at the docks. John Murphy, the second case, resided some miles away from Horsleydown or the docks; and, although I did not specially inquire whether he had been at the docks (being ignorant at the time of the case of John Harnold), I made general inquiries into his habits, and learnt that he remained usually at home, but, on the day preceding his death, had gone to Kensal Green, a district situated five or six miles on the Harrow road, in a contrary direction to Horsleydown or the docks. Jane Langham, again, lived near Fleet street, in a place far removed from either Lambeth or Horsleydown; and in her case it was proved, that she had not been out of the immediate neighbourhood for some time before her death. In respect to the cases at Chelsea, again, I begged Mr. Keen, who attended them, to inform me if any facts had come to his knowledge, which could lead to the suspicion that his first cases had been in contact or proximity with any diseased persons; Mr. Keen replied, that no such communication was traceable. What, indeed, except accident, could have brought John Murphy, the labourer in the gasworks at Lambeth, in contact with Harnold, the seaman of Horsleydown, or either of these with the wife of a coalporter in Fleet street, with a convict in the hulks at Woolwich, or with a sick man on board the Dreadnought Hospital-ship at Greenwich?

2. But if these several persons were in no way connected by trade, profession, or calling, if they were not exposed by proximity of habitation to the emanations emitted from the bodies of each, is it not possible that some extraordinary series of accidents may have brought them together; and that, without actual knowledge of the fact, John Murphy may have met John Harnold, Jane Langham, or the boy at Chelsea, and have infected, or have been infected by these persons?

To those who know the localities above named, and who are acquainted with this vast metropolis, this question will appear almost absurd. To those who are not possessed of this information, it may be mentioned that Horsleydown lies on the south side of the Thames, to the eastward of Bermondsey, and is situated close to the river, at a point nearly opposite to that where St Katharine's docks are placed. From Horsleydown to Lambeth, by the river, is a distance of several miles, as the Thames here makes a large curve. As the crow flies, however, Horsleydown is about two miles distant from Lambeth, or rather from Lower Fore street, where the second case of cholera occurred. The two spots are separated by the districts of Bermondsey and Southwark, which compose an immense city, crowded with inhabitants. The spot in Chelsea, in which the next case occurred, is on the other side of the river, and close to Battersea Bridge; it is, in a direct line, about two miles from Lower Fore street, Lambeth, and four from Horsleydown. Harp court, Fleet street, is in the city of London, about half way between Temple Bar and Ludgate Hill, and is separated from the Thames by the breadth of Fleet street, and the length of the lanes running from Fleet street to the river. As the crow flies, it is about two miles, or one mile and a half from both Horsleydown and Lambeth. It is about four miles from Chelsea. Woolwich is situated about eight miles from Horsleydown, ten from Lambeth and Fleet street, and twelve from Battersea Bridge. The

Dreadnought Hospital-ship lies off Greenwich, about three miles above the Justitia, and five from Horsleydown. Spitalfields, Clapham Common, and Bermondsey are also districts widely separated from each other. The spot in Bermondsey, in which the case occurred on the 9th of October (eleven days after John Harnold's case), was at the part of Bermondsey most remote from Horsleydown. If the four cases which occurred in the Registrar-General's weekly return, of the week ending October 7, are included, the following localities will have to be added, Stepney (one case) and Rotherhithe (two cases), both places being some considerable distance from the localities given above. The fourth case occurred in Spitalfields. The localities would thus be increased to twelve; the cases to thirty-two. As far as can be known, therefore, to leave out these four cases, and the two additional localities, is not likely to prejudice the strict contagious argument; whilst any addition to the number of localities would, *cæteris paribus*, be unfavorable to it.

Now as these twenty-eight were the only cases of cholera occurring in London within ten days (with the exception of the Registrar-General's four cases, which, if included, would even increase the number of affected localities), if we seek to derive the disease of each patient from contact or proximity with a previous case, we must have recourse to the most extraordinary suppositions. We must suppose that John Murphy had somewhere, in the densely crowded streets of London, brushed past John Harnold (who had been only three days in town, and was probably at the docks all day, rather than in the neighbourhood of Lambeth); that Jane Langham, in the same extraordinary way, also had a fatal rencontre with one of these two individuals; and that in a similar manner all the first cases in the several localities must have obtained the disease, by this accidental passing in the street of an individual whose residence was many miles away.

But the probability of such accidental rencounters in London will appear to every one to be unsustainable for a moment; and, in addition, is it possible that the rapid transit of individuals should in this way communicate cholera? If so, cholera is the most virulent of contagions, and nothing could prevent it from half depopulating entire London. But if this rapid transit did communicate it, it must be remembered that the only time John Murphy could have met John Harnold, or Richard Cook could have met either of these two, or Jane Langham any of the three, must have been before the symptoms were developed, even in the shape of premonitory diarrhœa, which symptom, indeed, did not occur in the first two cases, and commenced at night in the third. But I need not delay on this topic, as the next argument is absolutely conclusive against the opinion in question.

3d. In two instances, if not in more, it is quite certain that there *could* not have been even an accidental encounter between these patients and others labouring under cholera. In the hulk at Woolwich, the first case occurred on the 2d of October. Now the convicts at Woolwich may be said to be in a kind of quarantine; they work in the dockyard, are watched by the appointed officers, and are allowed no intercourse with other persons. The Justitia Hulk was lying about three miles below Greenwich, and far apart from any other vessel, except the convict hospital-ship. No merchant vessel, indeed, ever anchors at this point of the river; or if any vessel should anchor, it is merely on account of the tide, and the place of anchorage would be many hundred yards from the spot where the Justitia was moored, which was close in shore; so that if cholera had been raging in Woolwich, and had been prevailing in the vessels in the Thames above Woolwich, the origin of cholera in the Justitia would still, in all probability, not have been attributed to contagion. But there was no cholera in Woolwich, or in the merchant vessels in the Thames; and the only cases in London which were anterior in point of time to this at Woolwich, were those of John Harnold, at Horsleydown (seven or eight miles distant), John Murphy, at Lambeth (twelve or thirteen miles distant), Richard Cook, at Chelsea (thirteen or fourteen miles distant), and Jane Langham, in Fleet street,

(ten or twelve miles distant). The occurrence, then, of anything like contact or proximity between these individuals and Owen Jones, the convict at Woolwich, may be said to have been absolutely impossible. So again in the Dreadnought Hospital-ship, Robert Gordon was attacked on the 5th of October. The Dreadnought lies off Greenwich, and is three or four miles away from the Justitia, with which it holds no kind of communication. It is also many miles removed from Horsleydown, Lambeth, Chelsea, and Fleet street. Robert Gordon had been for a month on board before his seizure; he could not, therefore, have been in contact or proximity with any of the nine cases which occurred previous to his attack.

A question may be put here in respect to these two cases, which I shall have to put presently to all the cases. Although Owen Jones and Robert Gordon could not have been in contact or proximity with any of the known previous cases, may they not have been exposed to some other source of contagion?

To this it may be answered—

(*a*) That none such is known. On board the Justitia, as already said, the convicts were far removed from all other vessels, and were not allowed intercourse with any one. And on board the Dreadnought, as I have stated in a former part of this Report, no sailor arriving from any infected place, had been admitted with *any complaint whatever*, for some considerable time prior to the case of Robert Gordon. The Dreadnought also lies in a clear part of the river, and is several miles below the Pool where the merchant vessels lie.

(*b*) No other source of contagion can be indicated, except the cases above given, and unreported cases which may have occurred among the merchant vessels in the Pool. For these London cases are among the earliest in the kingdom, being only four or five days posterior to the cases at Hull; so that the number of possible cases is reduced to so small a number, that we can feel certain that nearly all chances of error arising from unreported cases are avoided.

It may therefore be concluded, with as much certainty as can ever be obtained from evidence of this kind, that—1, as there is no evidence of the persons mentioned above having been in contact or proximity with each other; —but on the contrary; 2, as such contact or proximity is, from the respective nature of the localities in which these cases occurred, and from the nature of the occupations in which the individuals were engaged, in the highest degree improbable; and, 3, as in two cases it is *absolutely* impossible that there should have been such contact or proximity;—therefore, the strict contagious theory, viz. that which supposes that an individual derives the epidemic disease from being in contact or proximity with a person already sick of the same malady, is not capable of explaining the mode of origin of the earliest cases of cholera in London.

The argument on which this conclusion is founded can be equally supported, if any of the cases now considered as cholera are denied to be of this nature. It is not worth while to go over the steps of the problem; but if the cases of Richard Cook, Jane Langham, or any others, are considered doubtful, and are left out, other cases become available, by which it is still more clearly proved that contact or proximity must have been impossible; and in addition, the period between the successive attacks becomes too great for the ordinary period of incubation, so that an additional argument arises against contagion.

We may ask here, whether any of these parties could have been exposed to any other "focus" of poison? I have already given reasons for believing that this could not have been the case with the cholera patients of the Justitia and the Dreadnought. There is no evidence of such latent or unknown source of contagion in the other cases. John Murphy had been for months resident in Lambeth; no other case had occurred at that time in Lambeth.* The family of Cook had been living in the same house in Chelsea in which they were attacked; no other person had had cholera in Chelsea since Mr. Haden's case, four months before. Mary Ann C. and her mother and sister lived in Spitalfields. No other case had occurred in Spitalfields at that time (unless the Registrar-General's case was anterior). James Baker was the first case in Bermondsey. The man in Prescott street, Clapham, was the first and only case in that locality, and had not been in contact with any diseased person. In fact it is needless to dilate on this point, as the chances are inconceivably small that all these persons should have been exposed to the influence of cases of cholera which are unknown to us. For it must be remembered that cholera is a disease so frequently fatal, and marked by symptoms so characteristic, that it is not likely any case would have been overlooked by the practitioner who attended it, or would not have been included, if fatal, in the Registrar-General's returns.

* I think Mr. Wagstaffe reported a suspicious case; but this was several months previously.

But now, as the *strict* contagious theory has been shown to be inadequate to explain these cases, can any modification of it explain them?—such as that which alleges that it is erroneous to limit the extension of the poison to a few yards, but that the particles of virus passing off from an infected person may float hither and thither to an indefinite distance, so that persons miles distant from the affected individual may really derive their disease from him.

If this modification of the original contagious doctrine be kept perfectly consistent with its parent theory, which maintains that the only multiplying source is the human body, it must on no account be admitted that particles of poison emitted from a diseased person can be increased in number after they leave the body. The particles are merely so far changed, when they float into the air, by the actual condition of that air itself; they may become more or less volatile, more or less stable, according to its heat, its humidity, or its electrical condition; or they may be wafted to a greater or less distance by the force of winds, or by the direction of currents of air. But they do not increase in number until they have entered another human body, and find in it the means of generating a fresh supply of poisonous particles. If this limitation be not kept in view; if it be admitted that particles given off from a diseased person may actually generate —external to the body—other particles like themselves; then of course the question is entirely different, and the relative power of the human body and of other conditions is to be settled by evidence brought to bear on the point.

But now let me inquire whether the following hypothesis can be supported. Let us put aside all the cases previous to that of John Harnold. Let us suppose that this man derived the disease either directly from cholera patients at Hamburgh, or from the sailor who died at sea. Let us suppose that Harnold entered London an infected person, and that three days subsequently, when the disease declared itself, particles of cholera poison floated off from his body into the surrounding atmosphere. Is it then possible that these particles should have drifted to Lambeth, to Woolwich, to Fleet street, and to Chelsea; and then on to other localities in which the disease appeared, and which may then have been also infected by the particles given off by these secondary foci, which also floated into the air, and augmented the number of those emitted by the first and imported case?

Such an hypothesis as this is neither supported, nor is it absolutely controverted, by direct evidence. But there are strong reasons for believing that it is not tenable. Of these reasons the following are the most stringent:

1. The above modification of the contagious theory necessarily assumes that the specific poisons derived from the animal body are not mere chemical or inorganic gases, because (putting other arguments aside) if they were, they would diffuse themselves rapidly according to the usual law of gases, and would doubtless be so speedily diluted as to make it hardly conceivable that the atmosphere should be dangerously tainted by them, over any great extent.

But if not gases, these poisons must be supposed to be organic particles, not endued with life, or with means of multiplication. They must be particles thrown out with the excretions, with the carbonic acid from the lungs, with the perspi-

ration from the skin, or with the excreta from the urinary or the intestinal surfaces.

But if this were the case, it is highly improbable that these particles would not, immediately after their entrance into the atmosphere, undergo the usual chemical changes to which organic matters are always subjected.

"The presence of the odoriferous principles of plants, the miasmata of marshes, and other matters of contagion," writes one of the most profound philosophers of our time, "although sufficiently obvious to the sense of smell, or by their effects upon the human constitution, cannot be detected by chemical tests. But it may be remarked in regard to them, that few or none of the compound volatile bodies we perceive entering the atmosphere, could long escape destruction from oxidation. The atmosphere contains, indeed, within itself, the means of its own purification, and slowly but certainly converts all organic substances exposed to it into simpler forms of matter, such as water, carbonic acid, nitric acid, and ammonia."*

If, therefore, such organic particles undergo these changes, we can hardly suppose it likely that they will thus drift in their original intensity through several hundred thousand feet of atmospheric air,—as must have been the case in the present instance, if they reached Lambeth and Chelsea from Horsleydown.

2. But whether the specific agent be an inorganic gas, or a cloud of organic particles, it is to be expected that, if given off in the manner supposed in the hypothesis, it would produce its greatest effects while most concentrated, viz. in the vicinity of the patient from whose body it was emitted. But this was so far from being the case in the present instance, that the agent must have passed innocuously over the tens of thousands of susceptible persons crowded in the dense lanes and alleys of Horsleydown, and have actually struck down its first victim at a spot separated from its source by myriads of houses and an immense space of ground; its second at a spot yet more remote; and its two next in localities still farther removed from all these places, and very distant from each other. If, indeed, the next cases had occurred in Horsleydown within two, three, or four hundred yards from the house where John Harnold died, we might have conceived the possibility of this driftage of poisonous particles; but it is surely an extreme assumption to extend this distance to an indefinite extent, more particularly when, as in the present case, there is no evidence of any cases occurring for some time subsequently between the two points of this extensive range.

3. There is another argument against such a view. There is strong reason to believe that the cholera-poison is not a very volatile one; it often limits itself in an extraordinary manner to one locality in a town; a street or narrow river sometimes separates a region in which the disease is raging severely, from one in which it finds no victims. And it must be understood that the class of persons in the affected and in the non-affected district is the same. Therefore it must surely be erroneous to note in one case the absence of volatility, and the next moment to ascribe to the poison so remarkable a volatility, that it shall drift for many miles in different directions, north, south, and east, as must have been the case, if the poison given off from John Harnold excited the disease in Murphy, Cook, Langham, or Jones.

We can hardly suppose, too, if the particles of poison were thus at the same time volatile and stable, that when three or four persons had been affected and contributed their quota of particles to the atmosphere, the multiplication of cases would have gone on in a progressive ratio, but this was not the case.

There are other arguments against this view, drawn from the direct evidence in favour of other modes of origin; but to these I do not wish now to allude.

If neither the strict contagious theory, nor this derivative opinion, can satisfactorily account to us for the production of these first cases in London, shall we

* Elements of Chemistry. By Thomas Graham, F.R.S., &c. &c. Second edition, p. 336.

derive more satisfaction from the opinions of the party who have been termed, though as I think erroneously, non-contagionists?

This opinion of this party is distinguished by the fact that, without denying to the human body a power of reproduction of each specific poison in various degrees according to the poison, or perhaps also to the condition of the body, it yet contends that the poisons can increase when altogether disconnected from the human system.

In the case of each poison, this party seeks to learn what are the circumstances most favorable for this increase external to the living organism; and these circumstances (the so-called conditions of increase and existence) are determined by actual observation of the predilection of the individual poison which may be under consideration, for special sites and localities, or for particular classes of men.

As yet, the local conditions which appear to be the most favorable for the propagation of the cholera-poison are alleged to be the following: 1. A low, damp locality, particularly one situated on the banks of rivers, or on marshy ground. 2. A close, impure air, from the accumulation of individuals, and from the effluvia arising from them. 3. Any circumstances which may cause organic particles to be continually emitted into the air, and which form over the locality a kind of canopy, which is continually dissipating itself into the surrounding purer air, and is as continually renewed by exhalations from below.

These conditions being present, it is alleged, from various reasons, that the cholera-poison increases and augments itself when once it is present in the locality.

It may become present in such a locality in four conceivable ways:

1. By the arrival of an infected person, whose body throws off the particles of poison, which then meet in the atmosphere around them with the conditions of increase.

2. By the arrival of one or more non-infected persons, to whose clothes or baggage particles of poison may have adhered during their passage through an infected district.

3. By the introduction of the poison, either by currents of air or from the influence of some peculiar force,* which causes the cholera-poison to pass over tracts of country in determinate directions.

4. By an actual generation of poison in the locality itself; a peculiar atmospheric or other dynamic state having concurred to make up, with the previously existing conditions, the sum of those circumstances whose assemblage causes the generation of the particular poison proper to that conflux of conditions.

It may be believed by this party that the cholera-poison can travel in all these methods, or may be manifested in one spot after another, in consequence now of one, and now of another, of these modes of propagation. The relative frequency of any of these methods, or their actual occurrence, it may leave to be settled by evidence; but as a general rule, it may believe that the degree of the several local conditions (the state of the receiving bodies remaining constant) will determine the prevalence of the disease in one locality rather than in another, and that by taking these conditions into account, the places where cholera will prevail most severely, supposing the poison to have once entered, may be pointed out. At the same time, in countries in which cholera is not indigenous, in addition to these local conditions, a general atmospheric state (the nature of which is as yet unrecognised) may be assumed; and in the countries in which cholera always more or less prevails, an increased prevalence of this choleraic

* The existence of this force must be admitted in the case of influenza, whose course has a singular relation to that of cholera. What this force may be—whether (for instance) it is allied to electricity in some form—cannot at present be known. May it not be something which, even yet, we have not fathomed? Who can yet say, with the wonderful discoveries in magnetism which the genius of Faraday or Oersted discloses, that some other allied or separate forces have not yet to be recognised in Nature?

stitution may be supposed occasionally to occur. If this view turn out to incorrect, it is yet alleged that it hypothetically expresses better than any other opinion the connexion of the phenomena under review. Into the proofs and general considerations connected with these subjects I do not wish now to enter, but simply to consider what support is given to any particular opinion by the early cases in London.

In twelve days, 28 cases occurred in ten different localities. In six of these localities, the circumstances which have been asserted to be efficient in aiding the activity of the cholera-poison were present in a high degree; in three others they were also present; but I have only general facts to bear on the subject, no special inquiries having been made. In the tenth locality, viz. in Prescott street, Clapham Common, in which a single case occurred, the conditions of development of the poison did not exist in a marked degree. The origin of this case, as stated in the account of the case itself, is rather obscure.

The six localities in which moisture, effluvia, and impure air were abundantly present were:—1, Lower Fore street, Lambeth; 2, White Horse court, Chelsea; 3, Harp court, Fleet street; 4, Hulk Justitia, Woolwich; 5, Spitalfields; 6, the Pool (part of the Thames).

In three other localities, viz. in Bermondsey, Horsleydown, and on board the Dreadnought, the poison must have met equally with moisture and effluvia. The case at Bermondsey occurred in the immediate vicinity of Long lane, which has long been notorious for its open ditch, its crowded and miserable houses, and for its general wretched sanitary condition. The cases at Horsleydown were also in a wretched neighbourhood close to the river; and on board the Dreadnought, although the ship is scrupulously clean, yet of course it is exposed to the effluvia from the numerous patients it contains, from the bank of the Thames near which it lies, and from the surface of the tainted water.

The point being conceded that in nine localities the poison would have met with favorable conditions of development, we have to inquire how the poison arrived in these localities;—was it brought there? did it travel there? or was it generated there?

The scrutiny to which these cases have been submitted, proves that in the greater number of the localities—in all, in fact, except Horsleydown—the poison was not imported by any person ill of cholera arriving in the locality. Nor could it have been brought by the clothes or baggage of any persons coming from infected districts in England, as there were no infected districts from which such persons could have come. There is no evidence either of any persons arriving from the Continent, from Hamburgh or from Dantzic, to these localities. If such persons did arrive in London, it is most probable that they were connected with the shipping, and were therefore many miles removed from Lambeth, Chelsea, and the other places in question.

In Horsleydown, however, the poison may have been imported; but if so, it is a very interesting point that it did not seem to propagate itself by contagion or otherwise. The next case which occurred in this locality was seven days after that of John Harnold, and after this there were only a few scattered cases for many weeks. The poison therefore died away, or rather remained at its lowest point of intensity.*

If therefore the poison was not brought to Lambeth, Chelsea, the hulk Justitia, or the Dreadnought, by infected persons or non-infected persons or clothes, did it enter in some other way, or was it generated in these localities?

Per viam exclusionis, it is necessary to admit either of these modes rather than the two former. It is not unreasonable to suppose that some moving force, altogether

* Although it so happened, that the first case in London, omitting the anterior sporadic cases, was in the person of a man who perhaps brought the disease with him, I think all the facts seem to prove, that the almost simultaneous appearance of other cases was a mere coincidence. I do not see how it can be otherwise; and yet, in the history of cholera, there are a good many of these coincidences.

independent of the bodies of men, may have driven particles of cholera-poison into these localities; for certainly there has been some power which, independent of human intercourse, and in opposition occasionally to winds, has driven this disease from the steppes of Tartary to the English shores.

With regard to the generation of the poison, there is no doubt that in Horsleydown, as well as in Lambeth, Chelsea, and in many parts of London, some of those circumstances which we have termed "local conditions of existence" existed in considerable intensity, and yet the cholera-poison did not develope itself in any commensurate degree. Was there not some essential condition wanting, whose deficiency prevented or nullified the effect which the other conditions would have had if conjoined with it? And if so, what was this condition?

And it may be remarked that this evident inability to propagate itself rapidly* was displayed in all the localities—at Chelsea, at Lambeth, and even at Woolwich; and it has continued to be marked, more or less, even to the present date (February).

At Chelsea, although the court in question was close to the river, was in a most filthy state, and was crowded with inhabitants, only six cases occurred; the disease then stopped, and has not since returned. In Lower Fore street, Lambeth, although a place which had actually been pointed out by Mr. Wagstaffe before the cholera appeared in England, as eminently displaying the local conditions favorable for the spread of the disease, only four cases occurred, and then the disease stopped for a month. And even on board the Justitia, although cases continued to be furnished until the convicts were moved from their unhealthy anchorage, yet they occurred slowly, and with intermissions of days, or occasionally even of a week, between each case.

I confess that I was much struck with this peculiarity, since in India (at any rate of late years) if in any large town presenting conditions like those of London, cases of cholera had been seen to occur simultaneously in several districts, the observer might almost certainly have predicted that that town was about to suffer from a general epidemic. The way in which the cholera almost died away out of London, is not explicable on the idea that the sanitary condition was too good to furnish the poison its local conditions of increase, nor is it explicable on the contagious theory, or on any modification of it.

The previous hypothesis of a choleraic constitution seemed to me the only likely way of accounting for the non-development of the poison after its introduction. When it is considered how cholera has lately passed like a broad belt over a particular part of Europe, leaving untouched the districts on either side, although these districts were populous, held free intercourse with infected districts, and presented hygienic conditions favorable for the reception of the poison, we shall see cause to believe that there is some element yet unindicated, connected with the actual progress of the disease,† which is wanted to complete the sum of conditions under which the poison attains its highest

* This also tells against the strict contagious theory; as, if susceptible human bodies were all that the poison wanted, these have of course been abundantly supplied to it.

† As an illustration of this point, I may refer to the "British and Foreign Medico-Chirurgical Review," Jan. 1849, p. 8, which is taken from the description, given by M. Lesseque (Archives Générales, Sept. 1848), of the course of the cholera in Russia.

"The peculiar force which impelled the cholera thus towards the north, is not only indicated by the general course of the disease, but by a closer study of its peculiarities. Whenever the malady deviated, so to speak, from its normal direction, and passed towards the west, it seemed incapable of propagating itself, and died away spontaneously, even in places which appeared to be well fitted for its reception. If, on its route, it encountered a large and densely-peopled town, it arrested itself there by preference; but the existence of a similar town out of the line of its course did not seem to be able to attract it, or to cause it to wander from its singular march. The exemption of the provinces to the east of the Volga might be perhaps explained by the nature of the country, in which the inhabitants are few and scattered; but the rich, fertile, and densely-peopled countries to the right of the Dnieper enjoyed an equal freedom from attack, which can only be explained by the fact, that they were situated beyond the line of the disease, which, as already stated, was principally between these

development. May not this unindicated element be a peculiar, as yet, unrecognised atmospheric condition which accompanies the poison, or which even, by meeting with certain local conditions, develops it? In London it may be supposed that this atmospheric condition has not yet been perfectly established, but that having been partially developed in the remarkable weather which ushered in the month of October, it has remained ever since stationary, or but slowly advancing to a more complete manifestation. In all these northern countries, to which the cholera-poison is foreign, it may be supposed that a particular atmospheric state is necessary.

Leaving, however, this conjecture—for at present it is little more—I may remark, in conclusion, that I have desired merely in this Report to apply the strict contagious theory to these early cases. I have decided that this theory cannot explain them. I have done so not merely on negative evidence that no contact or proximity between these early cases could be traced, but on positive testimony that such contact or proximity was impossible. These cases are, then, to be accounted for on other grounds; into these, however, I shall not at present enter more fully: contenting myself with the remark, that it appears to me more probable that the cholera-poison should have entered the localities first infected by virtue of some peculiar force acting irregularly and partially, so as to drive the poison into one rather than into another locality;—or that the poison should have been actually generated in those localities under the influence of a general atmospheric condition co-operating with the local conditions proper to each place;—than that it should have reached those localities by direct emission from diseased individuals in the vicinity or at a distance, or from clothes which had been in contact with infected persons.

I feel, however, that, without enlarging my basis of inquiry, I have no right to push this argument farther. To do so with any effect, facts drawn from a more extensive series of observations would have to be passed in review.

Whether or not the evidence which has satisfied my own mind will prove as satisfactory to others, I do not know; but I can affirm that I have collected this evidence with impartiality, and have stated it with sincerity.

Appendix.

As far as we can at present judge from what is known regarding the spread of cholera in Great Britain, there appears to be much evidence in favour of the position that this extension has occurred in two ways, most commonly by independent manifestation in particular localities, but sometimes by transmission through the medium of diseased persons. Some excellent Reports published in the 'Edinburgh Monthly Journal,' indicate that in Scotland in some cases importation was improbable; but that, in a few cases, the disease appeared to arise from importation, and did really manifest contagious properties. In London, while I have no doubt that contagion had no influence in originating the disease, the cases arising after contact with the Tooting children seem to prove the fact of occasional contagion; as do also the cases recently put on record by Dr. McWilliam (Med. Gaz., June 15). If we are not disposed hastily to reject all the evidence on one side, and as hastily to receive all the evidence on the other, I do not see what conclusion can be arrived at, but that cholera is occasionally, but only exceptionably, contagious.

In the April Number of the 'American Journal of Medical Sciences,' is some interesting information respecting the appearance of cholera in New York and New Orleans. On the 9th of November, 1848, the packet ship New York left Havre, with 352 emigrants and 33 crew, for New York. There had not been a

rivers. Although communication was not interrupted, as the system of cordons sanitaires was abandoned by the government, no effect was produced by this cause. The most frequent intercourse with the dense population of the west, living under very unfavorable hygienic conditions, even the lowering of the temperature, which, it might be supposed, would have caused the extension of the disease towards more temperate climates, produced no effect."

single case of cholera at Havre when they sailed. They had no case on board till the 25th of November, fifteen days after leaving the land, when a young German, in robust health, was attacked with vomiting, purging, and cramps of the extremities; he died on the third day. On the following day, the 26th, another case occurred, and proved fatal on the second day. On the 27th, a girl died in two hours with the same symptoms. On the following day, a boy died in four hours and a half. On the 29th, a man died in seven hours. On the 30th, two more children died. On the 1st of December the ship came to an anchor at the quarantine station, Staten Island, and before the passengers landed on the 3d, twelve more cases had occurred. At the time there was no cholera on Staten Island. The sick emigrants were sent to an hospital—the healthy to some large public stores, in which were about seventy persons convalescent from other diseases. A person who had been treated at Staten Island for fracture of the patella, assisted in the removal of the sick; three days subsequently, he was attacked with violent symptoms of cholera, and died the same day. Another man, who had been in proximity with the emigrants at the stores, left Staten Island on the day after they landed and went to New York; he was attacked with cholera, was sent back to the quarantine ground, and died on the same day as the former case. Three other persons, who had been resident on Staten Island, and in proximity with the emigrants, were subsequently attacked. It does not appear that these last four cases had been in contact with the sick particularly; but they were the occupants of the storehouse to which those of the emigrants who were not affected with cholera had been sent. Two more persons were afterwards affected; making eight in all. No persons were attacked except those mixed up with the supposed healthy emigrants at the stores; 43 of whom appear to have been afterwards attacked. None of the crew were attacked. Among the emigrants, all the victims were Germans; of whom there were 270, all of them *long resident* in or near Havre. The disease then died away, and did not appear again either at the quarantine station, or at New York itself.

In this case there seems no doubt but that seven persons became affected with cholera after being in contact with a body of persons, some of whose number had suffered, and others were about to suffer from this disease. A still more remarkable fact is the origin of this attack at sea, fifteen days after leaving the land. It can hardly be supposed that it was derived from Havre, where cholera did not prevail; this supposition also necessitating the allowance of an unusually long incubative period. It is perhaps more probable to suppose it to have been actually generated in the ship, which, no doubt, furnished unhealthy hygienic conditions in abundance, under the influence of which, and of some peculiar epidemic constitution, the special cause may have been brought into being.

The second case referred to in the 'American Journal' is somewhat similar to this. It is given by Dr. Fenner.

On the 11th December, 1848, the ship Swanton, 39 days out from Havre, arrived at New Orleans with 280 German and French emigrants. About ten days previously, several cases of bilious cholera had occurred in New Orleans, which all recovered, and were probably unconnected with the subsequent epidemic. When the Swanton left Havre on the 2d or 3d of November, there was no cholera in that place. The vessel was out twenty-six days before any death occurred; the first was from consumption, afterwards sixteen or seventeen deaths took place, mostly from bowel-complaints, supposed to be dysentery. On the 12th of December, a woman from the vessel was taken to the Charity Hospital with undoubted cholera. On the 13th, another emigrant was admitted, and also died. *On the same day three other cases of cholera were admitted, all of which proved fatal; none of these were passengers of the* Swanton, *and they were from different parts of the city.* On the evening of the same day, December 13th, another suspicious case occurred in a resident, who had not been near the ship Swanton nor seen any of the passengers. On the 15th of December, 8 cases were admitted into the Charity Hospital, and several occurred in private practice. On the 16th,

11 cases were admitted, and the cases in private practice were rapidly increasing in number. By the 22d, forty-five deaths had occurred in the city, and after this the epidemic rapidly extended. It was stated that the first three victims were cooks, who went every morning to the principal market, within a cable's length of the vessel; but this was erroneous, as the Swanton was nearly a mile away, and these cases occurred on the 15th, after several other cases had been noted. The disease reached its zenith about the 28th, and declined steadily from the 1st of January.

After the disease appeared, almost every vessel that left the city soon had cases aboard; persons having the disease and dying of it, were carried to all the landings, towns, and cities up the Mississippi, as high as Cincinnati. In many of these places it spread to a limited extent; in others it did not; in no place did it prevail as an epidemic. It spread among the plantations along the river and in the interior of Louisiana. To some of these the infection appeared to be directly carried; at others it began without any communication with an infected district. It prevailed at Houston, Texas, while Galveston on the sea-board escaped, although on the line of travel from New Orleans to Houston. At the Charity Hospital, as many as 50 cases occurred among the nurses, servants, and patients.

Some other facts are well worthy of attention. The Guttenburg left Hamburgh in October, and six or seven deaths occurred before she got out of the Elbe. When she got to sea the disease disappeared, and did not again return. So that in this case, getting to sea out of the infected district arrested the disease.

The analogy between the cases of the New York and the Swanton is certainly remarkable: both vessels left a port in which no cholera case was known, with emigrants; the time of departure was nearly the same, viz. the Swanton on the 2d or 3d of November, the New York on the 9th; both were bound to different ports of the same country, and therefore probably followed nearly the same track; in the Swanton, which sailed on November 2, cholera appeared after twenty-six days, therefore on or about the 28th of November; in the New York it appeared on the 25th of November. In these cases it appears possible that both vessels may have been nearly in the same position, when they were attacked by cholera. Considering, then, all the circumstances of the case, the departure of those ships from a healthy port, the length of time they remained free from sickness, their possible vicinity to each other when they were attacked, the fact of their being emigrant ships, and therefore presumably badly cleaned and ventilated, it does not appear improbable that both ships became nearly at the same time subjected to some peculiar influence, which caused in both cases an analogous and independent manifestation of the cholera poison.

Dr. Fenner, the reporter of the New Orleans epidemic, says, "Whether it be a mere coincidence that epidemic cholera broke out in this city, just at the time when a vessel arrived, having some cases of cholera on board, or that said vessel brought the infection, which rapidly spread through the whole community, is an exceedingly debatable question." And at the end of his Report he remarks, that the few facts he has brought forward do not decide about the contagiousness or transportability of cholera. It would seem, therefore, that the channel of introduction, or the reality of actual importation of cholera, into New Orleans, may yet be a matter of dispute.

C. AND J. ADLARD, PRINTERS, BARTHOLOMEW CLOSE.

To Dr. R. D. Charles
With the Author's Compliments

From Dr. Beale's "Archives of Medicine," October, 1858.

ON THE INFLUENCE OF MERCURIAL PREPARATIONS UPON THE SECRETION OF BILE.

By George Scott, M.D.,
Formerly one of the Physicians to the British Hospital at Renkioi, Dardanelles.

THE following experiments were made with the view of ascertaining whether the preparations of mercury really increase the flow of bile, as has hitherto been generally believed. The ductus communis choledochus having been tied in a dog so as to prevent any bile reaching the intestine, the gall-bladder was opened to allow all the bile secreted to escape externally. It was then collected in a way which will be described below, and, calomel being given at different periods, the effect of the same on the quantity of bile secreted was carefully noticed.

After describing the operation and the mode of collecting the bile, and before detailing the experiments with calomel, I shall say a few words regarding the normal amount secreted by the liver in twenty-four hours, as very various opinions have been offered on this point by different observers, and as the method I have employed seems sufficient to enable one to arrive at a tolerably correct conclusion.

On the 25th May, 1858, a middle-sized, black, rough, mongrel dog was operated upon for biliary fistula in the following manner:—

When fully under the influence of ether, an incision from three to four inches long was made in the linea alba through the abdominal walls, beginning just below the ensiform cartilage. The peritoneum and great omentum being incised to about the same extent as the integument and muscles, and the lower edge of the liver being a little raised, the gall-bladder was easily seen. A ligature being passed through its upper end, it was gently drawn towards the surface, and the duodenum being somewhat depressed, the ductus communis choledochus was seen without difficulty entering the intestine. The common bile-duct was tied in two places as near the duodenum as possible, and divided between the two ligatures. An opening was made in the upper end of the gall-bladder large enough to admit the forefinger; this was sewed to the edges of the upper end of the wound, and the latter was closed by ligatures up to the point where the gall-bladder was fastened. The wound was further

closed by several broad bands of adhesive plaster, and a broad linen bandage was placed over the whole.

The dog did very well, and recovered rapidly from the effects of the operation, so that on the 4th June his appetite was good, the wound was nearly healed at the lower part, and the fistula was quite patent.

7th June.—Is very lively and in good health. His weight was to-day ascertained to be 18lbs. 6oz. He has been fed on rice, milk, and boiled horse-flesh, but chiefly on the latter.

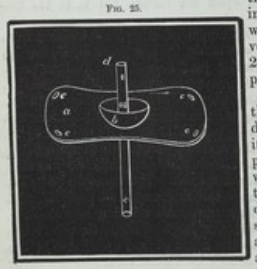
On the 9th June the dog was in such good health, and the wound was so well healed, that the collection of bile was commenced in the following way:—

Fig. 24.



The first part of the apparatus was a silver tube, represented in Fig. 24, about two inches long and a quarter of an inch in diameter, fitted in the middle and at right angles to it with a disk *a* (Figs. 24 and 25) about three inches long, and wider at both ends than in the middle. On one side of the disk was attached a circular cup-shaped piece of silver, *b b* Figs. 24 and 25, with a thick border, allowing the tube to pass through its centre, Fig. 25, *b d*. The part of the tube *d* was perforated with holes, several of which were at the base of *d*, and in such a position that if fluid were poured into the metallic cup *b*, when the tube was in a vertical position, as in Fig. 24, it would flow into the part of the tube *c*.

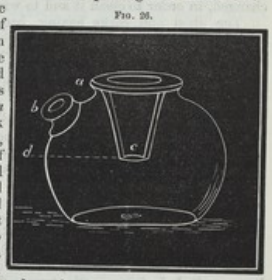
Fig. 25.



It will be observed that the tube on meeting the disk *a* is bent somewhat on itself. This is to allow the part of the tube *c* to hang vertically, notwithstanding the oblique position of the disk *a*, owing to the inclined shape of the body of the dog at the fistula, which was just at the point of meeting of the chest and abdomen.

The part *d* was inserted into the fistula, and the tube was fixed in its place by elastic bands attached to the holes *e e e e*, and fastened round the body. It was attached sufficiently tightly that the cup *b* was always applied firmly against the abdomen, so that no fluid could escape between it and the part around the fistula against which it was applied. Thus, if any of the bile escaped out of the fistula by the side of the tube *d*, it would only pass into the cup *b*, and then by means of the holes at the base of *d* it would pass into the tube *c*. Thus was prevented any loss of bile from its not all passing into the tube *d*.

Fig. 26.



The next part of the apparatus was a glass of the shape represented in Fig. 26. Up to the dotted line *d* it would contain about 1,500 grains of bile. The opening at *a* was provided with a cork perforated at the centre, so as to admit the end of the tube *c* easily, and having also several small holes perforated around the large one to admit air. The opening at *b* was also provided with a tightly fitting cork. In the first experiments the glass that was used had merely a circular hole at *b*, and was not provided with a rim, as here represented. It was found very difficult to close this opening completely, and hence some of the loss in the first experiments. From the 1st July, however, glasses similar to the one represented in Fig. 26 were used. The glass was also provided with a funnel-shaped tube *c*. By this latter arrangement it will be easily understood that when the glass was filled with bile or any other fluid up to the dotted line *d*, the same could not escape in any position which the dog might assume so long as the cork remained tightly fitted in the opening *b*.

The part of the silver tube *c* was placed through the hole in the cork at *a*, and the glass was kept attached to the tube by being tied firmly round the body with a simple piece of tape. Then over all was fitted a large piece of gutta percha, about a quarter of an inch thick, and reaching over the lower end of the thorax, most of the abdomen, and over both sides of the animal. It was moulded to the shape of the body and of the glass, so

that the latter lay in a large cup-shaped cavity of gutta percha. This shield was buckled over the back of the dog sufficiently tight to prevent any shifting of the apparatus during the movements of the animal. The cup of gutta percha receiving the glass was sufficiently large that when the dog was lying on it the shield did not press upon the tube or glass, but the latter lay in its covering quite secure from injury or pressure.

In collecting the bile the glasses were changed twice in the twenty-four hours, in the morning and evening, and the tube was removed from the fistula each time that the glasses were changed, in order to clean it and to wash out the wound.

The glass and corks having been carefully cleaned and weighed before they were applied, the weight of the fluid bile removed from the dog was easily ascertained by subtracting the weight of the glass and corks from the whole weight of the apparatus taken from the animal. The bile thus obtained in the evening being carefully covered up so as to prevent any evaporation from its surface, on the following morning, the glass was removed in the same way as before and weighed. The weight of the bile of the preceding evening being added to the amount obtained in the morning, the whole was reckoned as the bile of the same day as the morning. Thus the bile obtained on the evening of the 9th June, and that obtained on the morning of the 10th June, were reckoned as the bile of the 10th June. The bile obtained on the evening of the 10th June, and that obtained on the morning of the 11th June, were reckoned as the bile of the 11th June, and so on. If both glasses had been applied during exactly twenty-four hours, then the bile for that period was at once known; if they had been applied during only twenty-two or twenty-three hours, or any other period longer or shorter, then the amount of bile for twenty-four hours was calculated from the quantity actually obtained. In the majority of cases, the glasses having been changed at the same time every morning, the amount of bile for twenty-four hours was really collected. It was only in some cases that, owing to the glasses having been removed at a later or earlier hour in the morning, the quantity of bile was collected for either a greater or lesser period than twenty-four hours. In almost all the latter cases the time during which the bile was obtained was within an hour or two more or less of twenty-four hours. Thus, to give an illustration, the bile collected on the 10th June was for a period of twenty-two hours and a quarter, 837·2 grains, therefore the calculated amount for twenty-four hours was 903·04 grains. On the 11th June, the bile collected was for a period of twenty-four hours, 1628 grains. On the 12th, the amount collected was, also for twenty-four hours, 1767·7 grains. On the 13th, the quantity collected was, for twenty-four hours and three quarters, 2365·2 grains; therefore for twenty-four hours 2293·527 grains, and so on.

After the morning bile was weighed it was mixed with that of the preceding evening, and then a certain portion, never less than 500 grains, generally from 800 to 1,000 grains, was weighed out and analyzed, and the analysis of the whole amount of bile in the twenty-four hours calculated from this.

In the above manner I have succeeded in collecting very nearly *all* the bile secreted in twenty-four hours. Some cotton wool was always placed between the glass and gutta percha shield holding the same, so that if any bile escaped it could be easily detected from the green stain on the white wool. In the first experiments, those viz. from the 9th to the 19th June, there was always a little bile lost, owing to the cork not closing very tightly the hole in the glass at *b*. After, however, the glasses were provided with a rim at *b*, as represented in Fig. 26, this opening was so completely closed that no bile whatever escaped; and from the 1st July no bile was lost in the collecting, with the exception of a few drops on the 8th and 9th July. Of course a drop or two of bile escaped from the fistula during the changing of the glasses, but as the time employed for that process never exceeded a quarter of an hour, the amount thus lost was very inconsiderable. In the first experiments only twice, viz., on the 12th and 19th June, did the quantity lost exceed a few drops. On the other hand, it will be easily understood that although a few drops of the amount of bile obtained in twenty-four hours was lost, that by no means invalidates the results obtained with the calomel, as, of course, the loss day by day was very nearly the same, and the amount of bile collected was so great that the difference of a few drops would, in a very small degree, influence the results.

In the observations below regarding the daily amount of bile secreted by the liver, it must be understood that, owing to the slight loss above noticed, the quantity here given is rather *under* than *over* the proper quantity, as the amount has always been calculated from the quantity actually obtained.

The amount of bile secreted by the liver in twenty-four hours has been very variously stated by different observers. It seems proper to assume that the liver of any animal secretes a quantity of bile proportionate to the weight of the organ. Hence in animals with a relatively large liver, the daily amount of bile secreted for the weight of the body will be greater than the quantity of bile daily secreted by animals with a relatively

smaller liver. The weight of the liver of the dog is, from six observations of Bidder and Schmidt, as 1 to 26 of the whole body. The weight of the liver in man is, according to Quain, as 1 to 36 of the whole body. Hence we may conclude that a dog secretes daily more bile according to the weight of its body than a man. The greater relative weight of the liver in the dog than in man has always, therefore, been taken into consideration in estimating from the amount of bile secreted by the dog, the amount which a man of a certain weight should secrete in twenty-four hours.

	Fluid Bile.		Bile Solids.	
	Oz.	Grains.	Oz.	Grains.
Heller calculated that a man secreted in 24 hours	24	or 10500		
Douglas	29	or 12687		
Bouisson	6·777	or 2965		
Blondlot	7·058–8·825	or 3088–3861		
Platner calculated that a dog weighing from 19 to 21 lbs. secreted in 24 hours 5 to 6 oz. of bile, or, calculating that a human being would secrete the same proportionate amount of bile according to the relative weight of his liver, a man weighing 160lbs. would secrete in 24 hours about	31·777	or 13902		
Bidder and *Schmidt* calculate that for every 1 kilogr. or 2·20628lbs. of dog, there are secreted in 24 hours 19·99 grammes of fluid bile, and 0·988 grammes of solids. Considering the relative weight of his liver, a man weighing 160lbs. would, according to this, secrete in 24 hours ..	35·476	or 15521	1·824	or 798
Kölliker and *Müller* calculate that for every 1 kilogr. or 2·20628lbs. of dog, there are secreted in 24 hours 36·1 grammes of fluid bile, and 1·162 grammes of solids. A man weighing 160lbs. would, according to this, secrete in 24 hours	66·742	or 29200	2·146	or 939

It is an acknowledged fact that the quantity of bile secreted by the liver in a given time is very materially affected by the amount of food and drink taken. Hence it is indispensable in forming a correct conclusion of the amount of bile daily secreted to take into account the quantity of food and drink consumed. Of the above-mentioned observers only Bidder and Schmidt and Kölliker and Müller have given the quantity of food consumed by the animals experimented on; and it is, therefore, only with the results of the above four that the present experiments can be compared.

Of course, in estimating the amount of bile secreted in twenty-four hours by collecting the same from a biliary fistula, it is necessary to ascertain for certain that all the bile secreted by the liver escapes externally at the fistula. Now there are two ways in which the latter may be prevented.

1. Some of the bile may be re-absorbed into the blood by stoppage of any of the hepatic ducts, or by partial obstruction of the fistula. Under such circumstances, of course, the animal will become icteric, and bile-constituents will be detected in the urine. That no such thing took place in my dog is sufficiently proved by the fact that he had never any symptoms whatever of jaundice, and that, although the urine was many times collected and tested for bile-colouring matter and for bile acids, the results were always negative.

2. Recommunication may take place between the ductus communis choledochus and the duodenum, as has sometimes happened in cases of biliary fistula. That such did not, however, occur in the present case, will be seen by the following account of the post-mortem examination of the dog. I have thought it best to give the latter in this place in connection with the above remarks.

On the 14th August, eleven weeks and four days after the operation, the dog was in very good health, although very much emaciated. It was killed the same evening by blows on the head.

Post-mortem examination about forty-four hours after death.

Body much emaciated. Fistula quite patent, of about the diameter of a goose-quill, the edges besmeared with bile.

On opening the abdomen, the liver on its upper surface was seen to be adherent to the under surface of the diaphragm to a considerable extent—the adhesions were very firm. The duodenum and pancreas were firmly adherent over a considerable extent, to the under surface of the liver. On cutting open the fistula into the gall-bladder, some brown thick bile escaped. On tracing the cystic into the hepatic ducts, the ductus communis choledochus was seen to end in a cul-de-sac a short distance from the opening of the last hepatic duct into it. On opening the duodenum, and passing a probe through the orifice of the common bile-duct into the gut, and pushing the probe as far as it would go, this part of the duct was seen also to end in a cul-de-sac about $\frac{1}{8}$ths of an inch from the outside of the duodenum. The two portions of the common bile duct were separated by an interspace of about half an inch, and were both of them quite closed. No bile, therefore, could possibly have reached the duodenum from the time the operation was per-

formed. The liver presented at one or two parts some whitish-gray specks on the surface, of about the size of a pin's head. These were seen to enter the substance of the organ for a short distance. The liver was otherwise perfectly healthy in appearance.

The *stomach* and the upper part of the *small intestines* were full of the remains of food. The mucous membrane of the stomach and intestines was perfectly normal in appearance, except that of the vermiform process and rectum. The mucous membrane here presented a number of roundish patches of pigment, or rings of pigment, about the size of a pea, some scarcely so large, and placed very close to each other, giving the membrane a curious variegated appearance. There was no unevenness of the membrane at these pigmented rings, as if they had been caused by ulceration or cicatrization. There was some calcification of the cortical substance of the *kidneys*, otherwise these organs were perfectly healthy in appearance. *Suprarenal bodies*, *spleen*, and *pancreas* healthy.

In the *thorax*, the right auricle of the *heart* was quite filled with a clot of decolorized fibrine, which extended a long way into the vena cava inferior. The right ventricle contained a good deal of dark-coloured loose coagula, with a little decolorized clot. In the left auricle was also a little decolorized clot. Left ventricle empty. The tricuspid and mitral valves looked a little thickened and gelatinous at the base, but not much. Otherwise *heart* and its valves perfectly healthy.

Right *lung* anæmic, otherwise perfectly normal. Left *lung* somewhat hyperæmic and œdematous.

Table I. gives a full account of the quantity of bile collected each day, all the analyses of the same, the amount of food and drink daily consumed, the doses of calomel given, &c., &c., &c.

The amount of bile being estimated from morning to morning, and the food and drink always being given *after* the morning's bile was collected, it follows that the quantity of food of one day must have affected the quantity of bile of the *next* day. Hence, in Table I, I have placed opposite the bile of 10th June the food and drink actually given on the 9th, opposite the bile of the 11th June the food and drink actually given on the 10th, and so on.

In estimating the amount of bile secreted in twenty-four hours by this dog, I have taken the average of the first three days of July, as these appear to be the most correct of all the experiments. The dog was then in excellent health, a period of five weeks had elapsed since the operation, his appetite was very good, and the apparatus for collecting answered so well that the bile was obtained *without any loss*, except that unavoidable

TABLE I.

Date.	Amount of fluid Bile collected in 24 hours.	Amount of Bile-Solids in fluid Bile collected in 24 hours.	Amount of Nitrogenous matter of fluid Bile collected in 24 hours.	Pal.	Salts.	Mucus and other nitrogenous matter.	Loss.	Notes of amount of Bile collected.	Food.	Drink.	Doses of Calomel given.	Weight of Dog.	Observations.
	Grains.		Grains.		Grains.								
9 June	—	—	—	—	—	—	—	—	3500 grms. of boiled horse-flesh	18 oz. milk 10 oz. water	—	18 lbs. 6 oz. 9 drms.	
10 „	903·040	89·47	—	—	—	—	—	Some drops lost	1134 grms. of boiled horse-flesh	18 oz. milk 10 oz. water	—		
11 „	1428·000	103·93	—	—	—	—	—	Some drops lost	3780 grms. of boiled ox-lung	18 oz. milk 12 oz. water	—		
12 „	1797·700	80·032	11·657	38·073	12·321	16·709	1·972	Considerable amount lost	2948 grms. of boiled ox-liver	12 oz. milk 6 oz. water	—		
13 „	3230·347	169·947	32·864	38·696	20·038	19·079	·980	Some drops lost	3780 grms. of boiled ox-liver	18 oz. milk 11 oz. water	—		At 5 P.M. were coloured did not purge him.
14 „	1819·658	99·636	26·230	42·856	15·293	15·474	·003	Scarcely any lost	910 grms. of boiled ox-liver	11 oz. milk 10 oz. water	—		
15 „	896·680	41·961	—	—	—	—	—	Scarcely any lost	340 grms. of boiled ox-liver	18 oz. milk 6 oz. water	—		
16 „	1659·968	77·499	12·402	35·199	12·204	13·923	·9771	Scarcely any lost	3146·3 grms. of boiled ox-liver	18 oz. milk No water	At 1 past 4 P.M. 5 grms. in bread-crumb		Calomel purged him copiously in 4 hours.
17 „	818·700	43·180	10·426	13·199	6·478	10·103	1·974	Some drops lost	No food	12 oz. milk 12 oz. water	—		
18 „	1810·540	64·930	22·731	31·516	12·804	12·894	·002	Some drops lost	3900 grms. of boiled ox-liver	18 oz. milk 6½ oz. water	—	18 lbs.[1]	
19 „	817·717	61·776	27·486	13·166	11·106	10·016	·002	A considerable amount lost	No food	9 oz. water	—	17½ lbs. on 20th inst.	
30 „	—	—	—	—	—	—	—	—	7000 grms. of boiled ox-lung	14 oz. milk 10 oz. water	—		
1 July	2168·951	124·728	40·909	50·665	24·825	12·651	7·463	None lost	7000 grms. (15 lb.) of boiled ox-lung	20½ oz. milk			
2 „	2941·239	139·171	57·072	9·764	18·611	33·914	19·296	None lost	7000 grms. of boiled ox-lung	18½ oz. milk		—	
3 „	3148·400	146·333	47·870	12·725	16·938	36·097	29·869	·003	7000 grms. of boiled ox-lung	18 oz. milk 1 oz. water			
4 „	2560·300	125·613	40·843	22·469	13·933	29·767	23·998	·005	7000 grms. of boiled ox-lung	18 oz. milk 1 oz. water			It is uncertain whether this animal purged him.
5 „	2681·900	135·920	47·445	25·978	12·668	35·068	16·758	·003	6300 grms. of boiled ox-lung	About 10 oz. milk About 10 oz. water			
6 „	2644·300	134·784	54·319	8·680	14·569	31·967	19·823	None lost	7000 grms. of boiled ox-lung	20 oz. milk			
7 „	3672·900	119·771	48·748	13·833	25·882	10·698	6·086	None lost	7000 grms. of boiled ox-lung	18½ oz. milk	At 4 to 6 P.M., 12 grms. in bread-crumb	—	Calomel purged him copiously.
8 „	1963·900	94·304	40·388	8·900	14·487	21·306	9·229	·054	7000 grms. of boiled ox-lung	14½ oz. milk			
9 „	1466·400	77·062	29·008	8·775	9·710	15·932	7·907	1·610	7000 grms. of boiled ox-lung	8 oz. milk	—		
10 „	—	—	—	—	—	—	—	—	—	—	—	16 lbs.	

one of two or three drops during the period of changing the glasses.

On referring to Table I. it will be seen that the average amount of bile secreted for these three days, viz., 1st, 2nd, and 3rd July, was 2752·562 grains of fluid bile, and 134·410 grains of solids. The dog weighed on the 29th June 17¼lbs., therefore his average weight for the above three days may be taken as 17lbs. The average amount of food was 7000 grains, or 1lb. of boiled ox-lung. The average amount of drink was 19oz. or 8312·5 grains of milk.

	Fluid Bile.	Bile Solids.
	Grains.	Grains.
Or 1 kilogr. or 2·20628lbs. of dog, on a diet of 908·468 grains of meat, and 1078·787 grains of milk,* or 1068 grains meat, give in 24 hours..	357·23	17·443
Bidder and *Schmidt* calculate that each 1 kilogr. or 2·20628lbs. of dog on 882·2 grains of meat, 6·7 grains of lard, and 30·3 grains of milk, give in 24 hours	296·371	15·258
Kölliker and *Müller* calculate that each 1 kilogr. or 2·20628lbs. of dog on 988·421 grains of meat, give in 24 hours	557·528	17·945

	Fluid Bile.	Bile Solids.
Or, according to the *present experiments*, a dog consuming daily about $\frac{1}{47}$th of its own weight of meat, secretes in 24 hours	23·13 in 1000 of its own weight.	1·129 in 1000 of its own weight.
According to *Bidder* and *Schmidt*, a dog consuming daily about $\frac{1}{17}$th of its own weight of meat secretes in 24 hours	19·19 in 1000 of its own weight.	·937 in 1000 of its own weight.
According to *Kölliker* and *Müller*, a dog consuming daily about $\frac{1}{28}$th of its own weight of meat, secretes in 24 hours	36·1 in 1000 of its own weight.	1·161 in 1000 of its own weight.

Bidder and Schmidt calculate that dogs and cats in a healthy condition require for each kilogr. of weight of body, 50 grains, or $\frac{1}{20}$th of their own weight of fresh meat daily. Considering, therefore, that animals with biliary fistulæ require more nutriment than healthy ones,† the above amount of food given daily to the animals experimented on, seems to have been not more than their condition required. Hence it appears reasonable to suppose that a man *on full diet* would secrete, for the weight of his body, an amount of bile proportionate to that secreted by

* 1078 grains of milk give about 160 grains of solids. Calculating roughly that the latter amount of milk solids contain about the same quantity of nourishment as meat, then the amount of food in this case for each 1 kilogr. of dog would be 908 grains + 160 grains = 1068 grains of meat.

† See Die Verdauungssäfte und der Stoffwechsel, von Dr. F. Bidder und Dr. Schmidt. Art.: von der Galle.

the dogs above referred to, always taking into consideration the greater relative weight of the liver in the dog than in man.

	Fluid Bile.		Bile Solids.	
	Oz.	Grains.	Oz.	Grains.
Therefore, according to these experiments a man weighing 160lbs. would secrete in 24 hours	42·763	or 18709	2·084	or 912

The results arrived at above will be best understood by referring to Table II.

TABLE II.

Observers.	Daily amount of Meat in proportion to the weight of Dog.	Amount in Grains of Fluid Bile secreted in 24 hours for each kilogramme, or 2·20628lbs. of Dog.	Amount in Grains of Bile Solids secreted in 24 hours for each kilogramme, or 2·20628lbs. of Dog.
Bidder and Schmidt.	$\frac{1}{17}$th	296·371	15·258
Kölliker and Müller.	$\frac{1}{28}$th	557·528	17·945
We.	$\frac{1}{47}$th	357·23	17·443

Observers.	Amount in Oz. and Grains of Fluid Bile secreted in 24 hours by a Man, on full diet, weighing 160lbs.		Amount in Oz. and Grains of Bile Solids secreted in 24 hours by a Man, on full diet, weighing 160lbs.	
	Oz.	Grains.	Oz.	Grains.
Bidder and Schmidt.	35·476	or 15521	1·824	or 798
Kölliker and Müller.	66·742	or 29200	2·146	or 939
We.	42·763	or 18709	2·084	or 912

Kölliker tried the effects of calomel upon the secretion of bile in dogs, with biliary fistulæ, but it is difficult to form any definite conclusion from his observations in this respect. *Once* the bile seemed to be *increased*, and *twice* it seemed to be *diminished* by the administration of calomel. (Vide the Würzburg Verhandlungen, vol. v., 1855, page 231.)

In judging of the effects of calomel on the secretion of bile, I have, in all the experiments but one, calculated the average amount of fluid bile and bile solids secreted in twenty-four hours

two days *previous* to the exhibition of the medicine, and then the average amount secreted in twenty-four hours two days *after* the calomel was given. This seemed to be the most correct method of arriving at a proper conclusion, as it might be supposed that the effects of a large dose of calomel lasts longer than twenty-four hours.

The calomel was given each time after the morning's bile was collected, therefore the effect of the medicine was upon the bile of the day *following* the one on which it was given. Thus the calomel administered on the 13th June affected the bile of the 14th June, that given on the 16th June affected the bile of the 17th, and so on.

In the following observations the weight of the dog is not mentioned, as the variation in the same during the period of each experiment must have been but slight, and could not have affected the results obtained.

The first dose of three grains of calomel was given at three p.m. of the 13th June.

	Grains of Fluid Bile.	Grains of Bile Solids.	Grains of Bile Acids.
The average amount of bile secreted in 24 hours for two days *previous* to the administration of calomel, viz., 11th and 13th June,* was	1960·763	104·438	32·864
The average amount of food was, for the two days, 11th and 13th June, 4061 grains of boiled ox-liver, and the average amount of drink was 18¼ oz. milk, and 6¼ oz. water.			
After giving the 3 grains of calomel, the average amount of bile secreted in 24 hours for two days, viz., 14th and 15th June, was	1358·158	70·498	26·230
The average amount of food and drink for the two days, viz., 14th and 15th June, was 1675 grains of boiled ox-liver, 14¼ oz. of milk, and 7⅞ oz. of water.			
Here we find that after the exhibition of calomel, the quantity not only of fluid bile, but also of the bile solids and bile acids, was considerably diminished. The diminution may no doubt be partly due to the much smaller amount of food taken *after* than before the calomel was given.			
The second dose of 6 grains of calomel was given at half-past eleven a.m. of the 16th June. The amount of bile secreted in 24 hours of 16th June, the day *previous* to giving the above dose of calomel was ..	1639·968	77.599	12·502

* The numbers obtained on the 12th June are not used, owing to there having been a considerable loss in collecting the bile on that day.

The quantity of food and drink consumed on the 16th June was 3146 grains of boiled ox-liver, 18½ oz. milk, and 8 oz. water.

	Grains of Fluid Bile.	Grains of Bile Solids.	Grains of Bile Acids.
After giving the 6 grains of calomel, the quantity of bile secreted in the 24 hours of the 17th June was	518·700	42·180	10·426
No food was taken on the 17th June,* and the amount of drink was 12 oz. milk and 13 oz. water.			
Here again we find the fluid bile, the bile solids, and bile acids, remarkably diminished after the exhibition of calomel. The dog in this case took no food on the 17th June, the day *after* the calomel was given to him. Hence it might be concluded that the remarkable diminution in the quantity of bile secreted in this case as in the previous one, was due entirely to the great diminution in the amount of nutriment taken. To ascertain whether such a diminution in the quantity of food consumed was sufficient of itself to cause such a decrease in the amount of bile secreted, nothing but 9 oz. of water was given to the dog on the 18th June,† and the amount of bile secreted in 24 hours on the 19th June was	817·737	61·776	27·486

Of this latter quantity a considerable amount was lost in the collecting. Therefore we find that in this case, at all events, the mere deprivation of nutriment was not sufficient to cause such a diminution in the flow of bile, as the same cause combined with a dose of 6 grains of calomel.

The third dose of 10 grains of calomel was given at half-past four P.M., of 3rd July.

	Grains of Fluid Bile.	Grains of Bile Solids.	Grains of Bile Acids.
The average quantity of bile secreted in 24 hours for 2 days, viz., 2nd and 3rd July, *before* giving the 10 grains of calomel, was	3044.819	139·251	61·965
The average amount of food and drink for the 2 days, 2nd and 3rd July, was 7,000 grains of boiled ox-lung, and 18½ oz. of milk.			

* *i. e.* on the 16th June, but it has been placed in Table I. opposite the bile of 17th, as it affects the bile of the latter day.

† Placed, therefore, in Table I. opposite the 19th.

	Grains of Fluid Bile.	Grains of Bile Solids.	Grains of Bile Acids.
After giving the 10 grains of calomel, the average amount of bile secreted in 24 hours for 2 days, viz., 4th and 5th July, was ..	2720·900	135·436	70·662

The average amount of food and drink for the 2 days, 4th and 5th July, was 6,500 grains of boiled ox-lung, and 11¼ oz. milk. Here with a very slight decrease in the amount of nutriment taken, there is also a diminution of the fluid bile and bile-solids after the calomel, but the decrease is inconsiderable when compared with the two previous results. It will be observed that there is an *increase in the quantity of bile-acids* after the giving of the calomel.

The fourth dose of 12 grains of calomel was given at a quarter to six P.M., of 7th July.

	Grains of Fluid Bile.	Grains of Bile Solids.	Grains of Bile Acids.
The average amount of bile secreted in 24 hours for 2 days, viz., 6th and 7th July, *before* giving the 12 grains of calomel, was	2658·600	117.777	57·461

The average amount of food and drink for the above 2 days, 6th and 7th July, was 7,000 grains of boiled ox-lung, and 17¾ oz. of milk

After giving the 12 grains of calomel, the average quantity of bile secreted in 24 hours for 2 days, 8th and 9th July, was ..	1724·950	85·683	45·985

The average amount of food and drink for the above two days, 8th and 9th July, was 7000 grains of boiled ox-lung, and 11¼ oz. milk.

Here with as nearly as possible the same amount of nutriment during the whole period of the experiment, not only the quantity of fluid bile was decreased after the administration of 12 grains of calomel, but also the bile-solids and bile-acids.

In Table III, the results obtained are placed in a connected form.

TABLE III.

No. of experiment.	Period of time over which the experiment lasted.	No. of grns. of calomel given in each dose.	Average amount for 24 hours of food and drink given *before* the experiment.	Average amount for 24 hours of food and drink given *after* the experiment.	Number of grains of diminution in the secretion of Bile after giving Calomel. Fluid Bile.	Bile Solids.	Bile Acids.	No. of grns. of increase of Bile Acids after giving Calomel.
1	2 days before and 2 days after giving calomel, therefore 96 hours	3	4061 grns. of boiled ox-liver, 18½ oz. of milk, and 6¾ oz. of water	1675 grns. of boiled ox-liver, 14¼ oz. of milk, and 7¾ oz. of water	602·605	33·940	6·634	
2	1 day before and 1 day after giving calomel, therefore 48 hours	6	3146 grns. of boiled ox-liver, 18½ oz. of milk, and 8 oz. of water	No meat, 12 oz of milk, and 13 oz. of water	1121·268	35·419	2·076	
3	2 days before and 2 days after giving the calomel, therefore 96 hours	10	7000 grns. of boiled ox-lung, 18¾ oz. of milk	6500 grns. of boiled ox-lung, and 11¼ oz. of milk	325·919	8·815	—	8·697
4	2 days before and 2 days after giving the calomel, therefore 96 hours	12	7000 grns. of boiled ox-lung, and 17¾ oz. of milk	7000 grns. of boiled ox-lung, and 11¼ oz. of milk	953·650	32·094	11·476	

All the above four experiments with calomel give one result, viz., *a diminution in the amount of fluid bile and bile-solids secreted after the administration of large doses of calomel.*

In the first two, particularly in the second, the decrease was no doubt due somewhat to the diminished quantity of food taken the day after the calomel was given; but that it was not all dependent upon this cause seems to be pretty clear,—1stly., from the fact of the bile of 19th June, the day following that in which food and milk were purposely withheld from the dog, having been considerably greater than the quantity secreted on the day following the 16th June, when also no food was taken, and when in addition 6 grains of calomel had been administered to the dog; and 2ndly, from the bile in the last experiment, when almost exactly the same amount of nourishment had been consumed *after* as *before* the exhibition of calomel, having been also very much diminished in quantity. In three of the above experiments, the bile-acids were also considerably *diminished* after the calomel; but in one, viz., the third experiment, they were *increased*. Why the bile-acids should be increased in the latter case, and diminished in the other three, it must be confessed it is difficult to explain.

Although it would be rash to venture any decided opinion

from the very small number of experiments above detailed, yet the few that were made, all point so much to one conclusion that, if they be confirmed by future and more varied trials, they would throw considerable doubt upon the generally received opinion that calomel in large and purgative doses increases the flow of bile. It may be urged that although calomel does not increase the secretion of bile in the dog, that is no reason why it may not do so in man; and that, even if mercury do not excite the liver to increased secretion in a healthy state of the organ, it may still do so in certain diseased conditions of the same. If the first objection were true, the same could be brought against the results of the experiments which have been upon the lower animals to ascertain the action of poisons or, any other articles of the Materia Medica. With regard to the second objection, nothing analogous occurs in the action of drugs upon other organs; there is no medicine which diminishes the secretion of urine in the healthy state of the kidney, and increases the same in certain diseased conditions of the organ; there is no medicine which diminishes the amount of sweat in a healthy state of the skin, and acts as a diaphoretic in certain diseased conditions of the integument. Hence it seems difficult to suppose that anything which diminishes the flow of bile in a healthy condition of the liver, should increase the secretion of the same in a diseased state of that organ.

Whether it be the mere purgative effect of calomel which causes the diminution in the secretion of bile, or some specific action, further experiments must decide. Of course, it must be understood that the above remarks apply only to cases where *purgative* doses of calomel have been given. Whether small and frequent doses of calomel continued for a length of time, so as to produce the specific action of mercury upon the system, really augment the biliary secretion, is matter for further experiment.

The above analyses were made in the laboratory of Dr. Lionel Beale, to whose great kindness I am indebted for the opportunity of performing these experiments.

Dr. N. Hall
with the author's compliments

[*From the Fourteenth Volume of* "MEMOIRS OF THE LITERARY AND PHILOSOPHICAL SOCIETY OF MANCHESTER." *Session* 1856-7.]

ON THE OCCURRENCE OF INDIGO-BLUE IN URINE.

BY EDWARD SCHUNCK, PH.D., F.R.S.

MANCHESTER:
PRINTED BY T. SOWLER AND SONS, ST. ANN'S SQUARE.
1857.

XIII.—*On the Occurrence of Indigo-blue in Urine.*

By Edward Schunck, Ph.D., F.R.S.

[*Read April 7th*, 1857.]

The occurrence of urine exhibiting various peculiar and abnormal colours is a phenomenon which has frequently attracted the attention and excited the curiosity of pathologists. Of these variously tinted urines the most remarkable and striking are the black and the blue, but they are at the same time so rare, that it has been deemed of importance to record minutely the symptoms exhibited in each case as well as the chemical and physical properties shown by the urine itself. These urines have been observed in diseases of the most different kinds, as well as in cases in which the general health seemed not to be in the least degree affected. The pigments themselves, to which the colours are due, have not until lately been subjected to any chemical examination, and great doubts still prevail regarding their true nature. The blue pigment, to which I propose to confine myself on the present occasion, has been discovered in two states. In some cases it has been found ready formed so as to impart to the urine a blue colour, but merely in a state of suspension and therefore easily separated by simple filtration, whereas in other cases it has only made its appearance when the urine was left to stand or was subjected to the action

of various reagents. In the cases described by Janus Plancus,* Prout,† Braconnot,‡ and Simon,‖ it existed in the former state. Hassall developed the blue colour by means of putrefaction, in urines exhibiting the usual appearance, while Neubauer found the same effect to be produced by the addition of acids to the urine. As regards its chemical nature, the blue colouring matter seems to have been of three kinds, as far as can be ascertained from the descriptions given by the observers, which are not always very precise. In some cases, such as those described by Julia-Fontenelle,§ and Cantu,¶ the colour was evidently caused by Prussian blue, the iron of which appears to have been derived in one case from a quantity of ink which the person had swallowed. The second kind of colouring matter has been minutely described by Braconnot, who obtained it simply by filtering the urine from the blue deposit found suspended in it. It was a dark blue powder, insoluble in water and alkalies, only slightly soluble in alcohol and yielding no crystalline sublimate when heated. From its dissolving in acids and its being reprecipitated by alkalies and other bases, Braconnot inferred that it consisted essentially of an organic base, to which he gave the name of *cyanourine*. If the substance which he examined was pure, it seems certainly to have been of a peculiar nature. Nevertheless no one has since then observed any colouring matter which could be with certainty pronounced identical with it, though instances have been met with in which the blue colour not being caused, as it seemed, by any well-known body, has been attributed to the presence of cyanourine. In the third class of cases the blue colour was produced by a

* Commentarii Instituti Bononiensis, ad Ann. 1767.
† On Stomach and Renal Diseases. 5th ed. p. 567.
‡ Annales de Chimie et de Physique. T. XXIX. p. 252.
‖ Simon's Animal Chemistry, translated by Day. Vol. II. p. 327.
§ Archives générales de Médecine. T. II. p. 104.
¶ Journal de Chimie médicale. T. IX. p. 104.

substance, which on examination of its properties and reactions was found to be indigo-blue. Prout and Simon each mention a case in which indigo-blue was deposited from urine on standing, in the shape of a blue sediment. Neubauer* observed that the urine of a young man of 18, apparently in good health, when mixed with strong acids became first purple, then blue, and deposited a blue powder, which however he could not with positive certainty identify as indigo-blue. Hassall† was the first to point out that the occurrence of indigo-blue in urine was by no means so rare a phenomenon as had previously been supposed. The specimens of urine in which Hassall discovered it were mostly of a pale straw colour and acid. On standing they became thick and turbid and changed in colour from yellow to brown then to bluish-green, while the surface became covered with a blue scum or pellicle, which was found to consist of impure indigo-blue. Hassall considers that the exposure of the urine to the oxygen of the atmosphere is essential for the formation of the colouring matter, however I shall show that this exposure is by no means necessary. He also maintains that indigo-blue does not occur in healthy urine, that its presence is accompanied with strongly-marked symptoms of deranged health, and that its formation in urine must be regarded as a strictly pathological phenomenon, conclusions which are, as will be seen, quite at variance with the results of my experiments.

Such in a few words is the present state of our knowledge on this rather obscure subject.

In my paper "On the Formation of Indigo-blue,"‡ I have shown that the colouring matter exists in plants in a very different state to what had hitherto been supposed, that it does

* Anleitung zur Analyse des Harns. S. 19.
† Proceedings of the Royal Society, Vol. VI. p. 327; and Philosophical Transactions for 1854, p. 297.
‡ Memoirs of the Literary and Philosophical Society of Manchester, Vol. XII., p. 177; and "On the Formation of Indigo-blue," in the present Vol.

not exist in them ready formed nor as reduced indigo, and that the presence of oxygen is not essential to its formation, but that it owes its origin to the presence of a peculiar substance, soluble in water, alcohol and ether which by the action of acids is decomposed into indigo-blue, to which I have given the name of Indican, also a peculiar kind of sugar and a small quantity of other products. After having investigated the properties of this substance and its products of decomposition, I conceived it to be a matter of great interest to ascertain in what state indigo-blue exists in those urines, in which its presence is not indicated by the external appearance but is only made manifest by treatment with various reagents. That such urines should contain a body resembling indican seemed indeed exceedingly probable, since the same reagents which produce indigo-blue from indican lead in most cases to the development of the blue colour in particular kinds of urine. The extreme rarity however of these kinds of urine appeared to present an insuperable obstacle to the further investigation of the subject, and I therefore resolved to astertain whether any conclusions could be arrived at from an examination of ordinary healthy urine.

When muriatic or sulphuric acid is added to urine, the mixture on being heated becomes brown and begins to deposit dark brown flocks, which increase in quantity when the heating is continued. When these flocks are filtered off, washed and dried, they form a compact dark brown mass, from which cold alcohol extracts a resinous matter, leaving undissolved a brown powder, which dissolves however in a boiling mixture of alcohol and ammonia. This powder contains nitrogen and so much resembles indifuscine, one of the products of the decomposition of indican, as almost to lead one to suspect their identity. Its composition however, though it stands, as I have ascertained, in a certain relation to that of indigo-blue, is quite different from that of indifuscine. Now if the liquid filtered from these flocks be mixed with a salt of oxide of

copper and an excess of caustic soda, it becomes greenish, and if after being filtered it be heated for some time it gradually deposits a tolerably large quantity of suboxide of copper, which is a proof of the presence of sugar. That the latter has been formed during the process and did not pre-exist, may be ascertained by previously heating a portion of the urine with a salt of copper and caustic soda, before treating the remainder of it with acid. Samples of urine, which, when tried in this way, afforded very doubtful or no indications of their containing sugar, were found after being boiled with acid, then filtered and made alkaline, to reduce oxide of copper in a very marked manner. This reaction, which is so simple that it is only surprising it should never before have been observed, seems to me to prove that there is contained in urine some body, which by decomposition with acids yields sugar, the brown flocks precipitated at the same time being probably the substance with which the sugar was originally associated in the form of a copulated compound. From various considerations, which I need not detail, I was led to infer that this body could be no other than the very imperfectly known, so-called extractive matter of urine, and I accordingly commenced an investigation of this substance, which has led to conclusions of considerable interest. On discovering that the composition of the brown flocks formed by the action of strong acids on urine is expressed by the formula $C_{14}\ H_7\ NO_4$, which is also that of anthranilic acid, a product of the decomposition of indigo-blue, no further considerations were necessary to induce me to proceed with the investigation, notwithstanding the difficulties which I found attending it. Into the details of this investigation I shall at present only enter so far as they relate to the occurrence of indigo-blue in urine.

When acetate of lead is added to urine it produces a cream-coloured precipitate, which consists of chloride, sulphate, phosphate, and urate of lead, and contains also a little of the extractive matter of urine, which is, as it were, merely attached

to some of these lead compounds, since it is not precipitated from its watery solution by acetate of lead, when in a state of purity. The filtered liquid which is much paler in colour than it was before the addition of acetate of lead, gives with basic acetate of lead a second precipitate of a pale cream-colour, which consists of the lead compound of the extractive matter mixed with some basic chloride of lead. Both this and the first precipitate give when treated with sulphuric or muriatic acid yellow liquids, which after being filtered from the sulphate or chloride of lead and boiled, yield brown flocks exactly like those obtained from urine itself. The liquid filtered from the precipitate with basic acetate of lead is almost colourless. It gives, however, on the addition of ammonia an almost white precipitate, the quantity of which is much less than that of either of the two other precipitates. Now this precipitate exhibits a very remarkable peculiarity. It contains in most instances in combination with oxide of lead, a small quantity of a substance which by decomposition with acids yields indigo-blue. The first time that I treated this precipitate with acid I was surprised to observe that the liquid became immediately of a purplish-blue colour, and deposited after filtering and standing a small quantity of a substance, which on examination was found to consist chiefly of indigo-blue. This phenomenon was observed on so many occasions that I came to the conclusion that the occurrence in urine of an indigo-producing body similar to indican, was by no means an unusual circumstance.

In order to ascertain whether this body is present, I adopt the following method. The urine having been mixed with basic acetate of lead until no more precipitate is produced is filtered, and after the precipitate has been washed with water the liquid is mixed with an excess of ammonia, which always produces more or less of a white or yellowish-white precipitate. This precipitate is collected on a filter, slightly washed with water and then treated with dilute sulphuric or muriatic acid

in the cold. After the whole of the oxide of lead has combined with the acid employed, the liquid is filtered. When there is much of the indigo-producing body present the filter acquires a blue tinge, small particles of blue pigment are seen dotting the surface of the sulphate or chloride of lead, and the surface of the liquid, which is of a brownish-purple colour, in a very short time becomes covered with a thin pellicle, which is blue by transmitted and copper-coloured by reflected light, particles of the same blue substance being at the same time found attached to the sides of the vessel. When there is less of the indigo-producing body present, this pellicle only appears after some time, sometimes not until the next day. After twenty-four hours however the action of the acid is always completed, so that if no indigo-blue then appears or can be detected on examination of the deposit, the total absence of the indigo-producing body may be inferred. On the succeeding day, however large the quantity of blue deposit formed may be, the liquid no longer appears purplish, but brown, and after being filtered and boiled deposits a dark brown powder, having exactly the same appearance as that produced by the action of acids on the ordinary extractive matter of urine. The matter left on the filter after being washed is treated with caustic soda, which dissolves a portion acquiring thereby a brown colour. The portion which remains undissolved after being again collected on a filter and washed, is treated with boiling alcohol. In most cases the alcohol acquires thereby a bright blue colour. When however the quantity of deposit formed is tolerably large, the boiling alcohol first dissolves another substance, which imparts to it a fine purple colour, and which I consider to be identical with indirubine.* That which the boiling alcohol leaves undissolvd is a bright blue powder having the properties of indigo-blue.

* It is very probable that Heller's urrhodine, as well as Golding Bird's purpurine are also identical with indirubine, which, as I have shown, has the same composition as indigo-blue.

It dissolves in an alkaline solution of protoxide of tin, and the solution on exposure to the air becomes covered with a blue film. It is soluble in concentrated sulphuric acid forming a blue solution, which remains blue even after dilution with water. It imparts to boiling alcohol a bright blue colour, and the solution on cooling and standing deposits blue flocks. When heated in a tube it gives a purple vapour which forms on the colder parts of the tube a blue sublimate.

Provided with this test I proceeded to examine the urine of a number of individuals, and I succeeded in obtaining indigo-blue in so great a number of instances, that I have no hesitation in saying that the indigo-producing body, if not exactly one of the normal constituents of urine, occurs more frequently than any other of the abnormal ones. The urines containing it exhibit no remarkable or peculiar appearance whatever; they are acid, clear, and of the usual colour. Its occurrence, at least if its quantity is moderate, is not to be considered as a pathological phenomenon. I can at all events state from my own experience, that its presence is not attended by any symptoms of ill health or feelings of discomfort, and that neither from the state of the health nor the appearance of the urine can any conclusions be drawn as to its presence or absence. The small number of samples of morbid urine, which I had an opportunity of examining, yielded, with one exception, no more indigo-blue than the generality of healthy urines. Nevertheless, there are no doubt diseases in which the quantity of the indigo-producing body may become so large as to constitute a truly morbid symptom, and it may therefore become a matter of importance and interest for the medical man to have a ready means of detecting it. The delicacy of the test which I have described, as well as the small quantity of the substance usually present, may be judged of from the fact that by working for several weeks on the urine of two individuals, which contained a comparatively large quantity, I obtained one grain of indigo-blue. Even

when the amount of indigo-blue formed was very small I always found that 16 fluid ounces of urine yielded an appreciable quantity of it.

The urine of forty different individuals, all of whom were apparently in a good state of health, yielded, with one exception only, more or less indigo-blue, when examined in the manner just described. These individuals belonged to both sexes, and they were of ages varying from 7 to 55. The majority were persons of the working classes. The largest quantity of indigo-blue was obtained from the urine of a man above the age of 50, a publican by trade. The urine of a young man, aged 32, a servant in my employment, yielded almost as large a quantity. Among the rest, the urine of a young man, aged 25, an engraver, that of a clerk, aged 23, and that of a girl, aged 12, who had been a cripple from infancy, were alone remarkable for the amount of indigo-blue which they yielded. In all these cases the indigo-blue was accompanied by the substance imparting to alcohol a purple colour, and which I suppose to be indirubine. The other specimens afforded much less, sometimes mere traces. In all cases however in which the urine of the same individual was examined at different times, the amount of indigo-blue obtained from it was found to vary exceedingly, it being sometimes considerable and occasionally dwindling down to a mere trace. It was only very rarely however that none was found. In the case of the individual first referred to, the urine gave on one occasion not a trace, and this took place when he was engaged in performing labour, unusual for him both in its nature and amount. In my own case, as well as that of my assistant, the amount varied most capriciously from a tolerable quantity to a mere trace, occasionally even none at all being obtained.

I performed several experiments with different kinds of diet in order to ascertain the effect on the amount of indigo-blue yielded by the urine. Only one experiment, however, led to any decisive result. Having selected an occasion, when the

night urine gave no indigo-blue, I took on the next night, before going to bed, a mixture of treacle and arrowroot boiled with water in as large a quantity as the stomach could bear, and the effect was that the urine of the following night gave a large quantity of indigo-blue. As, however, the same phenomenon was repeated for several succeeding nights without any additional quantity of food having been taken, it remained uncertain to what cause it was to be attributed, though a repetition of the experiment on a second occasion gave the same result.

I have hitherto not had an opportunity of examining many specimens of urine in disease. Of two samples of urine from patients with albuminaria one gave a small quantity of indigo-blue, the other not a trace. Several specimens of diabetic urine yielded it. One of these, which I owed to the kindness of Dr. Browne of Manchester, gave a much larger quantity than I obtained from any other specimen of human urine.

The urine of the horse and the cow when tried in the same way as human urine gave comparatively very large quantities of indigo-blue, especially that of the horse.

I think it is highly improbable that the indigo-blue obtained in Hassall's experiments was produced, as he supposes, by the action of oxygen on the urine. Its formation was without doubt due to the decomposition of the indigo-producing body induced by the fermentation of the urine, the indigo-blue at the moment of its formation dissolving in the fermenting alkaline liquid and producing a true indigo vat, from which it was gradually deposited by the action of the atmospheric oxygen. When small quantities of indigo-blue only are formed in any specimen of urine, fermentation is not in my opinion to be recommended as a means of detecting it.

The occurrence of the indigo-producing body as an excretion seems to me to be due to a disproportion between the oxygen absorbed by the system and the matter to be acted on by it, which again may be caused either by an excessive

waste of the tissues or by an obstruction of the organs conveying oxygen, as the lungs and skin, or, as is probably the case in the majority of instances, by an excess of food being taken over and above the requirements of the system. As regards the constitution of this body, I think there can be no doubt that it contains the elements of indigo-blue and sugar, and that by oxidation within the system it is converted into the ordinary extractive matter of urine, which contains, as I have ascertained, the elements of sugar and of the black substance which is formed by the action of strong acids on urine, and which may be considered as a product of the oxidation of indigo-blue. Having prepared the extractive matter of urine in a state of purity, ascertained its composition, and examined its products of decomposition, I think it is probable that the indigo-producing body will be found, as regards its formation and composition, to occupy a place between the substance of the tissues and the ordinary extractive matter of urine. The very minute quantities of it ordinarily occurring in urine, and the difficulty of separating it from the extractive matter, make it, however, impossible to ascertain whether this is the case or not. My object in making known this portion of the investigation in its present fragmentary state, is to induce medical men, who have an opportunity of examining many varieties of urine, to endeavour to discover among these varieties some containing a sufficiently large quantity of this body to enable the chemist to ascertain its properties and composition.

The formation of a substance containing the elements of indigo-blue in the animal system is a fact which may lead to important conclusions regarding the chemical composition of the complex bodies of which the blood and tissues consist.

VERTIGO

A PAPER READ TO

THE NORTH LONDON MEDICAL SOCIETY,

APRIL 12, 1854.

BY J. RUSSELL REYNOLDS, M.D., LOND:

UNIVERSITY MEDICAL SCHOLAR.

London:
JOHN CHURCHILL, PRINCES STREET, SOHO.

MDCCCLIV.

LONDON: PETTER AND COMPANY, PRINTERS, 102, CHEAPSIDE,
AND NEW STREET, DOCTORS' COMMONS.

VERTIGO.

MR. PRESIDENT AND GENTLEMEN:

IN the following paper I shall depart somewhat from the common limitation of the term Vertigo, by using it as the expression of *two* classes of phenomena. In one of these, the Vertigo is a reality, in the other it is not: in the first, there are vertiginous *movements;* in the second, vertiginous *sensations.* Etymologically, the word is applicable to both, Ἴλιγγος in the Greek language, and Vertigo in the Latin, being equivalent to our word rotation, or whirling; and deriving themselves, respectively, from Ἶλιγξ a vortex, or whirl. If the term should be limited to one class rather than the other, it is the former to which it belongs, and not the latter, to which by common usage it has been applied.*

Although vertiginous movements have been supposed by some to depend upon sensations of an allied character, the latter may (as it appears to me) be most readily understood by an examination of the former; and considering the question in this light, I propose, first, by a brief historical sketch of its development, to lay before you the present state of our knowledge with regard to the nature and causation of rotatory movement; then to recall the principal phenomena of vertiginous sensations; and, in conclusion, to point out the relation which subsists between the two.

VERTIGINOUS MOTION.—The writings of ancient medical authors contain some scattered details of rotatory and allied move-

* "Vertigo Latinis derivatur a verbo vertere et agere: Græcis autem Ἴλιγγος vel Σκιρτος audit, quæ vox teste Erotiano in expositione vocum Hippocraticarum, saltationis genus significat, in qua saltaturus uno tantum pede terræ insistens in gyrum se vertebat, qui lusus ab inexercitato peractus efficiebat vertiginem."—Boerhaave. Prælectiones de morbis nervorum. 1761, Ed. Lug. Tom II., fol. 576.

ments as phenomena of disease, but we can gather little from these sources of their Pathology. Passing from them to the writings of the 17th century, the hints given are more instructive. For example, Bonetus says that in 1634 Nicholas Reutten described to him vertiginous movement in a lamb: and that in 1645 a similar case fell under his own observation. ("Oviculam vertigine et longa circumgyratione debilitatem mactari jussit.*") From the description of post mortem appearances, there can be no doubt that Hydatids were present in the brain. Morgagni furnishes a more complete account of a similar observation, by Valsalva. "Ovis cæterarum sui gregis consortium fugiebat, se per intervalla quotidie sæpe circumvolvebat, nec sibi caput tangi ferebat, sed ejusmodi tactus impatiens, omni conatu se subducebat. . . . Nam primum cum hoc (cerebrum) e cranio eximeret, pauxillum aquæ aciduläe excidit ex ea parte qua processus mamillares ad os ethmoides accedebant, major autem aquæ copia, cum a glandula pituitaria avelleretur. Deinde cerebrum persecando, cum ad ventriculos laterales ventum est, folliculus in his apparuit, aquam plurimam continens, ex membrana factus, quæ tenuis meningis productio esse videbatur, nisi quod in illa extabant minima quædam corpuscula, quorum substantia ad medullaris cerebri similitudinem accedebat. Folliculi radices e fundo existebant ventriculi dextri." †

In 1736, Peyer made this record—"In cane vertiginoso . . . in cellula ossium spongiosorum prope initium cerebri ad latus processuum mamillarium latebat vermis similis eidem, quem antea jam descripsimus;"‡ and the greater part of Morgagni's first letter is occupied with a discussion of the probable manner in which these vermes "have their exits and their entrances." But these facts and comments aid us little in comprehending the phenomena. Longet says that Pourfour du Petit, experimenting upon dogs in 1766, observed, after section of the cerebellar peduncles, the animal "rouler comme une boule." § This observation attracted

* Sepulchretum Boneti (by Mangetus.) Lib. II., sect. ii., obs. 8.
† De sedibus et causis. Lib. I., Epist. i., obs. 6.
‡ "Colore iridescente, planus, fimbriatus, latus superne, inferne gracilis, in conspectu nostro esse mutans ad æqualem latitudinem."—Observat: Anatomicarum, 1736, p. 38.
§ Recueil d'observ: d'anat: et de chir: 1766., p. 121. See Longet. Traité de Physiologie. 1850. Tome II, p. 215.

no attention, nor did the following, by one of the greatest naturalists of our own, or of any other time, Alexander von Humboldt. In a treatise published by him in 1792, there is this passage (appended as a note to some observations instituted for another purpose). "Dieses Drehen von Thieren, deren der Kopf abgeschnitten und das Rückenmarks noch nicht zerstöhrt ist, gehört zu den wunderbarsten vitalen Erscheinungen. . . . . Ich habe bemerkt dass besonders solche Frosche in engem Kreise umherhüpften an deren Rumpf noch etwas vom kleinem Hirn . . zurückbliebt. Es schien, als wenn das Rechts und Links drehen dadurch bestimmt wurde, dass jene Medullarportion an der linken oder rechten Seite grosser war. . . . . Das Drehen deutete also immer auf ein gestöhrtes Gleichgewicht in der Medullar-substanz des Nervensystems hin."*

The next observation of importance was made by M. Serres, in the year 1819. It was the case of a man, aged 69, who on the 5th of January, 1819, having dined with his friends, "et bu beaucoup, comme à son ordinaire, fut néanmoins plus étourdi par le vin qu'il n'avait coutume de l'être. Une circonstance qui l'avait frappé dans cet état, c'est qu'il ne voyait pas tourner les objets, comme cela arrive d'ordinaire, mais qu'il lui semblait au contraire *qu'il tournait lui-même;* ce qui porta ses amis à croire qu'il était ivre, et le fit conduire chez lui. A peine y était-il arrivé, qu'il se mit à *tourner réellement sur lui même.* Le tournoiement avait lieu de droite à gauche." In the night he was attacked with apoplectic symptoms, and complete left Hemiplegia. The former rapidly disappeared, leaving only motor paralysis of the left lower extremity. Death occurred on the 24th of May, and at the subsequent examination of the cranium, a cavity (nine lines in length and five in breadth) was found "au centre de l'entrée du pédoncule du cervelet dans l'hémisphère droit de cet organe . . . . tout l'hémisphère droit du cervelet était plus consistant que le gauche."†

Here, rotatory movements followed injury to the cerebellar peduncle, and took place from the injured, towards the healthy side. This observation was soon followed by the experimental

* Der gereizte Muskel-und Nerven-faser. Vol. II, p. 352.
† Anatomie comparée du Cerveau. Tome II, p. 623. Journal de Physiologie expérimentale. Tome IV, p. 499.

researches of M. Flourens. In a memoir read to the Academy of Sciences, in the year 1822, having for its object the "Determination des propriétés du système nerveux," we find the following conclusion from a series of observations upon removal of the cerebellum: "La volition, les sensations, les perceptions persistaient: la possibilité d'exécuter des *mouvements d'ensemble* persistait aussi; mais la *coordination* de ces mouvements en mouvements de locomotion, réglés et déterminés était perdue."* In the same memoir, M. Flourens stated his belief that the cerebral hemispheres were the necessary receptacles of sensation, as well as the centres of volition; but, at a subsequent period, and after the report upon his memoir by MM. Cuvier, Berthollet, Pinel, and Duméril,† he was led to alter this conclusion, and to substitute the word "perception" for "sensation." Accordingly, in the second edition of his "Recherches" we find "L'animal qui a perdu ses lobes cérébraux n'a pas perdu sa *sensibilité*; il la conserve tout entière; il n'a perdu que la *perception* de ses sensations; il n'a perdu que l'intelligence."‡ In the same report, however, MM. Cuvier, Berthollet, &c., pay a well-merited tribute of praise to M. Flourens, for the ability, novelty, and correctness of his observations and inductions with regard to the cerebellum. Rolando§ had previously observed many of the phenomena described by Flourens, but he had erroneously concluded that the cerebellum was the *source* of all muscular activity.

In the first edition of his "Précis Elémentaire de Physiologie," Magendie published some curious observations upon the effect of sections and ablations of the corpora striata; and, in 1825, he read to the Academy of Sciences a memoir relating farther researches, not only upon these bodies, but upon the cerebellum, and its crura. The principal phenomena described by him are the following:—

1. Removal of the corpora striata made the animal run forwards.

* Recherches expérimentales sur les prop: et les fonct: du Syst: Nerv: Ed. 2, p. 38.

† Fait à l'Academie roy: des Sciences, 22 Juillet, 1822. Journ: de Phys: exp. Tome II., p. 372.

‡ Op: cit: p. 66, 79.

§ Journal de Physiologie expérimentale. Tome III., p. 109, et seq.

2. Injury to the cerebellum caused retrogression.

3. Injury of the medulla oblongata had a similar effect.

4. Upon section of one peduncle of the cerebellum, "l'animal se met à rouler latéralement sur lui même, comme s'il était poussé par une force assez grande; la rotation se fait du côté où le pédoncule est coupé, et quelquefois avec une telle rapidité, que l'animal fait plus de soixante révolutions dans une minute."

5. The same kind of effect was produced by all vertical sections of the cerebellum, with this remarkable circumstance, that the movements were always more rapid when sections were made near to the origin of the crura. ("C'est à dire de leur communication avec le pont de varole.")

6. Toutes les sections verticales (du pont de varole) d'avant en arrière produisent le mouvement de rotation . . . les sections faites à gauche de la ligne mediane déterminent la rotation à gauche, et vice versa.

7. Un mouvement en cercle à droite ou à gauche, semblable à celui du manége se montre par la section de la moelle allongée, faite de manière à intéresser la portion de cette moelle qui avoisine en dehors les pyramides antérieures "*

Movements, more or less similar to those described by Magendie, had been witnessed by Fodéra,† Flourens,‡ and Bouillaud;§ but in respect of those depending upon lesion of the cerebellum and corpora striata, there was by no means the constancy of occurrence or direction which he maintained. Flourens obtained similar results only five times in eighteen experiments; Bouillaud four times in the same number; while Lafargue‖ failed to produce the phenomena once in ten experiments. And farther, the ninety-three cases of cerebellar disease collected by Andral present only one example of retrogression.¶ Longet also appears to consider as altogether doubtful the results of Magendie's experiments; and

* Précis élémentaire de Physiologie. 4me Ed Tome I. p. 412, et seq.

† Journ. de Physiologie expérimentale. Tome III., p. 193.

‡ Op. cit., p. 38 et seq. § Recherches cliniques et expérimentales.

‖ Essai sur les valeur des localisations encéphaliques, &c., p. 15. Not having been able to see the original thesis of Lafargue, the following quotation is cited from Longet:—"Aucune de mes dix expériences sur le cervelet (dit M. Lafargue) n'a produit le mouvement de recul; d'où il suit que le mode de locomotion observé par d'autres, à la suite des mutilations de cet organe, n'est pas assez constant pour justifier l'hypothèse de M. Magendie." Longet, Anat: et Phys: p. 746.

¶ Clinique Médicale. Tome V., p. 702. Journ. de Phys: exp: Tome, IV. p. 162.

although admitting that "la détermination précise des usages du cervelet est un des problèmes les plus embarrassants de la physiologie," yet gives his assent to the conclusion of Flourens, that it (the cerebellum) influences in a special manner "la coordination des mouvements de translation; parce que la physiologie expérimentale la confirme pleinement; parce que l'anatomie anormal ne la contredit point; parce qu'enfin. . . . elle n'est peut être pas en opposition aussi formelle qu'il le semblerait d'abord avec les faits pathologiques."*

Magendie succeeded, after many failures, in performing a vertical section of the cerebellum in the median line; and then, he says, the animal appeared swayed by two opposing powers—the momentary triumph of one being immediately overcome by the energy of the other.† But these observations have received quite another character from more careful experimentation.

In 1825, M. Giron de Buzareingues reported to the Academy his observations upon vertiginous movements in the sheep, and recognised their dependence upon hydatids in the cerebellum. This author, whose paper will fully repay perusal, observed that loss of sight accompanied the disease: a phenomenon which had been observed to follow certain lesions already described.‡

Between the years 1825 and 1828, Flourens, having been occupied with some researches upon the sense of hearing (the results of which he communicated to the Academy towards the close of 1824),§ made the important discovery, that certain sections of the semicircular canals of the ear were followed by peculiar motor phenomena, which subsequent research enabled him to systematize. These movements were found to have their direction in definite and constant relation with that of the special canal which was injured, and to be proportionate in intensity to the degree of injury inflicted. They were more marked and more lasting when the canals of both sides were injured than when only one was divided; and, upon every attempt at locomotion, they persisted so long as the animal lived. The first experiments were performed upon birds, and they may be summed up in the following statement:—

* Anat: et Phys: du Syst. Nerveux. Tome I., p. 769.
† Journal de Physiologie, Tome IV., p. 404. ‡ Ibid. Tome VIII., p. 307.
§ Op. cit. Conditions fondamentales de l'Audition, p. 452.

Division of the *horizontal* canal causes alternating movements of the head from side to side whenever there was any attempt to walk; but when not making such attempt, the head was fixed towards the injured side.

Division of the *vertical* canals causes vertical movements of the head, accompanied with sommersets forwards, when the *superior* was injured; and backwards, when the *inferior* was affected. The amount and persistence of movement was regulated, in each instance, by the conditions of injury already described. Similar experiments performed upon small mammiferæ (and especially upon the Rodentia) led to similar results. Some difficulty was encountered by Flourens in dividing the anterior canal in these animals (the homologue of the superior in birds), but the success with which he accomplished division of the other two left no doubt as to the correctness of his observation.

Having ascertained these points, the effects produced by division of the crura cerebelli were submitted to re-examination. These (crura) are three in number on each side:—

1. The transverse;—middle;—or crura ad pontem forming the connection between the lateral parts of the cerebellum, and the pons varolii, with the medulla oblongata.

2. The postero-anterior;—superior;—crura ad cerebrum;—or processus ad testes, which place the inferior folia of the vermiform process in relation with the corpora quadrigemina (or bigemina in birds) and the crura cerebri.

3. The antero-posterior;—inferior;—processus a cerebello ad medullam;—or crura ad medullam, uniting the folia on the upper surface of the cerebellum, to the spinal cord, by means of the corpora restiformia.*

Flourens found that division of the *middle* crura caused rotation of the animal on its own longitudinal axis: and movement similar in character to that following division of the horizontal canal of the Ear. Section of the *superior* (or postero-anterior) induced forward motion, resembling that caused by injury to the superior vertical (or anterior) canal; and that division of the *inferior* (or antero-posterior) crura was followed by backward movement, identical with that observed after section of the inferior

* Quain and Sharpey's Anatomy. Ed. IV., p.p., 720, 735, &c.

vertical (or posterior) canal. Flourens made this general statement of the phenomena:—"La direction des mouvements produits par la section des fibres du cervelet et de l'encéphale est toujours la même que celle des fibres coupées."*

The results obtained by Flourens have been confirmed only partially by subsequent observers. Longet says, "Dans aucune de mes expériences, je n'ai observé la tendance au recul que Flourens a notée après la blessure des pédoncules inférieurs du cervelet."† With regard to the inferior peduncles, Longet was equally unsuccessful; e. g. "L'opération préalable, qui consiste à les mettre à nu, ayant déjà troublé le mouvement, je n'ai pas remarqué que ce trouble fût sensiblement augmenté par leur lésion." But the results of his experiments upon the middle or transverse crura resemble closely those obtained by Flourens, and by Magendie at an earlier period; differing, however, from the latter in respect of the direction which these movements take. Magendie had found that the rotation took place "du côté opposé à la section." (Longet)‡ Flourens, that it was always "du côté lésé,"§ and Longet agrees with the latter, which is also in accordance with pathological observations, and with the subsequent experiments of Lafargue, who asserts that "la rotation selon l'axe du corps s'opère toujours du côté de la section vers le côté opposé."‖

This discrepancy may be partly accounted for by the close proximity of fibres having various directions, and the consequent difficulty of limiting accurately the locality and extent of section. M. Schiff, who had attended Longet's demonstrations in 1844, endeavoured to reconcile these differences by renewed experiments, the results of which he published in the following year. When the peduncles were laid bare from behind, through the occipito-atloidean space, he found that the animals turned from the injured side, and that they moved in the opposite direction when the lesion was performed from the front. This second

* Flourens Recherches: p. 495.
† Traité de Physiologie, 1850. Tome II. p. 215.
‡ Op: cit. §Op: cit. p.489. Magendie's own statement is,—"j'avais coupé le pédoncule gauche, le mouvement de rotation avait lieu de droite à gauche." (Journ: de Phys: exp: Tome IV., p 401); but in a subsequent account of the phenomena, he says,—"la rotation se fait du côté où le pédoncule est coupé."—(Précis élémentaire. Tome I. p. 411.)
‖ Thèse cit. p. 17., and Longet's Traité, &c., p. 216.

direction he accounts for thus:—"Si quidem . . . operationem eo modo facere tentas, qui ad nervum quintum secandum in usu est, rarissimis tantum casibus fit, quod vero pedunculum cerebelli medium seces, sed oblique partem externam loborum lateralium cerebelli secabis, et si partem medullarem læseris, animal quoque rotationes faciet circa axin longitudinalem, toto eodem mechanismo, ac si pedunculum cerebelli medium *lateris oppositi* secavimus, sed non tam celeriter. Rotationes ita versus latus læsioni oppositum se faciunt. . . . Actio loborum cerebelli ita directa est, actio pedunculi cerebelli est cruciata."* Longet does not admit this explanation by Schiff, which attributes "ce dernier effet plutot à la lésion de l'hémisphère cérébelleux correspondant qu'à celle de son pedoncule."† His own explanation of the matter is based upon still farther researches, anatomical in their character. These may be briefly stated thus;—that each of the middle crura cerebelli contains "en arrière des fibres non entrecroisées . . . et, en avant, des fibres entrecroisées. En me fondant sur ces données anatomiques, qui me paraient incontestables, je m'explique comment, d'une part, on obtient des *effets croisés* en lésant en avant l'un des pédoncules cérébelleux moyens, quand bien même l'hémisphère cérébelleux correspondant n'a pas été atteint; comment d'autre part, on observe des *effets directs*, quand on blesse en arrière l'un de ces mêmes pédoncules."‡

It has been already stated that Magendie observed a curious rotatory movement, "semblable à celui du manége," when a portion of the medulla oblongata was divided. Longet found that an identical phenomenon occurred when injury was inflicted upon one of the crura cerebri.§ Lafargue produced the same effect by dividing one of the optic thalami; but (he adds) "lorsque par suite de l'affaiblissement progressif, la station devient impossible, on verrait l'animal tomber sur le côté paralysé et *rouler sur* son axe."‖ Longet has not observed this transformation, but Schiff appears to have done so; "mais il l'explique par la compression exercée sur un côté de la protubérance par du sang

* De vi motoria baseos encephali inquisitiones. Bockenhemii, p. 53, 1845.
† Traité de Physiologie (ant. cit.) p. 216.
‡ Traité cit.; p. 216—218.
§ Traité cit.; p. 216—218.
‖ Thèse cit.; p. 17.

épanché."* With regard to the direction of this "mouvement de manége," there is a similar discrepancy to that described above. Longet and Lafargue agree that the movement is from the injured side; Flourens differs. The experiments of the two former were performed upon rabbits; those of the latter upon frogs. But, according to Schiff, the locality of section accounts for the difference:—"La destruction des trois quarts antérieurs de cet organe, chez les lapins, déterminait le mouvement vers le côté lésé, et celle de son quart postérieur vers le côté opposé à la lésion." And this is yet farther explained by the relative decussation or non-decussation of its anterior and posterior fibres.

The experiments of Flourens upon the semicircular canals of the ear do not appear to be confirmed or contradicted by Longet or subsequent observers; but in his attempt to elucidate farther the curious motions following their section, Flourens insists upon a distinction of function between their nerves and those supplying other portions of the internal ear. The latter (the nerve of the Cochlea) is, according to this author, the sole acoustic nerve; the former, "n'est pas un nerf des sens: la section des canaux semicirculaires ne detruit pas l'ouïe . . . le nerf est un nerf special et propre . . . Il est doué de la faculté singulière d'agir sur la direction des mouvements." In accordance with this diversity of function Flourens finds that the deep origin of this nerve is altogether different from that of the true auditory nerve. It has three roots, one from the Pons Varolii; a second from the crura cerebri; and a third from the corpora restiformia. These localities of origin are often given, in other treatises, as appertaining to the whole of the Portio mollis of the seventh pair; but Flourens says, although the nerve of the canals is "toujours accompagné du nerf acoustique . . . il ne se confond jamais."†

Some curious relations were found by Magendie, and subsequently by Flourens, to subsist between the movement or position of the eyes, and several of the lesions just described, Magendie, after division of the Pons Varolii (or rather of its transverse fibres) describes, "l'oeil du côté blessé se porte en bas et en avant; celui du côté opposé est fixé en haut et en arrière, ce que donne à la

* These later researches, which I have not seen, are in Roser and Wunderlich's Archiv: 1846, p. 681. The quotation is from Longet's Traité, p 219.
† Flourens (Recherches) op. cit., p. 494.

face une étrange expression."* When the semicircular canals of the ear were divided, Flourens found that "le globe de l'oeil et les paupières étaient dans une agitation extrême et presque perpétuelle . . . du reste, il voyait très bien; il entendait; il conservait tous ses instincts, toute son intelligence."† These movements of the eye conduct us to another series of phenomena, equally important. Flourens observed, that when one of the tubercula bigemina or quadrigemina was removed, the animals rotated towards the injured side. M. Serres had made an earlier experiment of the same character upon the horse, which was followed by irregularity of movement, and from which he erroneously concluded that these tubercles "sont excitateurs de l'association des mouvements voluntaires, ou de l'equilibration."‡ But M. Serres also observed that this injury to the corpora quadrigemina entailed upon the animal loss of sight. Magendie for some time disputed this; but it is now generally received (as proven) that these bodies are the true centres of the visual sense. Longet, admitting this relation, accounts thus for the phenomena of rotation:—"Le tournoiment qui vient d'être indiqué nous paraît tenir (quand toutefois la lésion se borne aux tubercules et n'intéresse point les fibres pédonculaires) à la perte de la vision dans un œil. En effet, ayant complétement évacué les humeurs de l'un des yeux sur des pigeons, jai vu souvent ces animaux tourner sur le côté de l'œil sain, et leur cou se tordre dans le même sens."§

If now we endeavour to gather from the preceding sketch the demonstrated causes (or occasions) of vertiginous movement, they resolve themselves into lesions of the following parts:—

1. The transverse, or middle crura of the cerebellum, with their continuation upon the Pons Varolii.
2. The crura cerebri.
3. The thalami optici.
4. The medulla oblongata.
5. The tubercula quadrigemina.
6. The semicircular canals of the ear.
7. The eye itself.

* Op. cit.: Précis élémentaire, et Journal de Physiologie.
† Op. cit., p. 457.
‡ Anat. Compar. du Cerveau. Tome II, p. 717
§ Traité cit., p. 224. Anat. et Phys., Tom. I., p. 475.

We must, for the present at all events, leave, as doubtful, the effects of injury to the following :—

1. The superior and inferior crura cerebelli.
2. The corpora striata.
3. The cerebellum itself (in respect of the direction of motion).

Having given this resumé of the phenomena, we have now to consider the various theories which have been advanced for their explanation. From the earliest period of physiological inquiry, common observation had proved that external impressions, through some mysterious "consensus of the nerves," passed over to, were returned, or reflected upon the muscles; and while philosophers were puzzling themselves to account for this conversion, by showing how "images" of external things should be conveyed inwards, and mould the "animal spirits" into their own likeness, physiologists were busy, some by one means and some by another, to discover the organ, or part of an organ, in which the consensus should take place, and by which the transformation could be effected. The corpus callosum, the corpora striata, the pineal gland, centrum ovale, medulla oblongata, &c., have all had this honour conferred upon them in their turn. Until the middle of the seventeenth century two classes of phenomena were indiscriminately grouped together, viz., the conscious, and voluntary; with the unconscious, and involuntary; and even when their separation was effected in respect of consciousness, the deeply-rooted tendency to project volition into surrounding objects would not allow the idea of "animal spirits" to be discarded. All kinds of phenomena (physiological and pathological) were referred to their caprices, forms, and actions. However, at this time, the distinctive characters of these two groups of action were more duly recognized. Hervey, whose glance had penetrated deeply wherever it had dwelt, has the following passage :—"Aliud est, musculum moveri vel contrahi; aliud vero eundem variis contractionibus, et relaxationibus regulatis, actionem aliquam perficere, utpote, progressionem vel apprehensionem." And farther, having observed the confused character of convulsive movements in decapitated animals, they occur thus (he continues) :—"Quoniam potestas cerebri ablata est, et sensus communis evanuit, cujus antea moderamine, cum rhythmo et harmoniâ, motus illi ad

progressum aut volatum regulabantur."* The idea of the "animal spirits" still held its sway; and although Dr. Willis in his treatise, "De Anima Brutorum," records many observations which appear to us, at this period, only obscured by such explanations, the human mind could not for a long time free itself from these remnants of what Auguste Comte would call its theologico-metaphysical leading-strings. However, Van Helmont, Lister, Avicenna, and others, were bold enough to deny their existence; and Unzer clearly systematised the functions of the nervous system without their aid. Unzer's idea, or at all events his method, is traced, by the translator of his Physiology, to Willis, through the medium of Wedel, Hoffman, and Stahl. However this may be, he had certainly made great advances upon Willis; for we find him saying, "We must assume what will be demonstrated in another part of this book for the first time, namely, that the external impressions on the nerves become themselves an animal motor force before they reach the brain and develope sensations there."† Unzer distinguishes clearly between the impressions which produce movement with the co-operation of sensation, and those which do so without. Prochaska carried out the views of Unzer, limiting much more accurately the locality of the consensus. He excludes part of the brain, and says :—"It certainly does not appear that the whole of the cerebrum, or cerebellum, enters into the constitution of the sensorium commune . . . it seems probably to extend through the medulla oblongata, the crura of the cerebrum, and cerebellum, also parts of the thalami optici, and the whole of the medulla spinalis."‡ But it is to the researches of Dr. Marshall Hall, M. Bouillaud, Cuvier, Flourens, and others, that we owe our more definite knowledge upon the subject; which results in the recognition of two distinct classes of movement (A), the one volitional, and (B) the other a-volitional. This second class is divided into (a) those accompanied with sensation, or emotion, or idea, as a more or less necessary link in the chain of causation; and (b) those entirely independent of either—the true reflected, or, as Dr. M. Hall has lately termed them, excito-motor, or diastaltic.

* Exercitationes de Generatione Animalium. 1651. p. 193.
† Sydenham Society Translation, (of Unzer) p. 98.
‡ Sydenham Society Translation, (of Prochaska) p. 430.

We have now to return to the subject of vertiginous movement, and in order to facilitate its explanation, must premise that, for the existence of sensation, the cerebral lobes are not required. It is the teaching of comparative and experimental physiology that the organs of sense, their nerves, and the ganglia at the base of the brain constitute the complete apparatus of sensation proper.*

We must also consider it proved, that the function of the cerebellum (whatever others it may possess) is to co-ordinate muscular movements for the attainment of a definite end. I have already pointed out the period at which Flourens announced this as the result of his experiments. This was done, as it forms an integral part of the subject now under consideration, but it would be out of place to recall the facts upon which this conclusion is based.

These points taken for granted, we may commence with the theory of Magendie. He concluded that there existed in the cerebrum and cerebellum distinct centres of impulsive force, which in the condition of health, balanced each other, and produced equilibrium or rest; but that when one or more of these motor centres was injured, and its function consequently impaired, the phenomena of advance, retrogression, rotation, &c., were occasioned by the consequent ascendancy of those which remained.† The data upon which Magendie founded this conclusion have been shown to be incorrect by the subsequent experiments of Flourens, Bouillaud, Lafargue, Longet and others; but yet farther, it would entirely fail to account for rotation following injuries to the ear and eye.

M. Giron de Buzareingues, believing the cerebellum to be the "organe de la mémoire des sensations," hinted that the vertiginous

* To enter upon this subject is quite beyond my present limits, but I refer the reader to Bouillaud's researches in the "Journal de Physiologie," "Flourens Recherches." The report upon this, by M. Cuvier, &c. M. Serres' "Anat. Compar;" and Dr. Carpenter's "Principles of Human Physiology," 4 Ed.

† *Précis élém*: de Physiologie, Tome I. p. 413. Journal cit. Tome III. "Comme notre esprit à besoin de s'arrêter à certaines images, je dirai qu'il existe dans le cerveau quatre impulsions spontanées ou quatre forces qui seraient placées aux extrémités de deux lignes qui se couperaient à angle droit; l'une pousserait en avant, la deuxième en arrière la troisième de droite à gauche, en faisant rouler le corps, &c. . . . . les animaux deviennent des espèces d'automates montés pour exécuter tels ou tels mouvements, et incapables d'en produire aucun autre." After speaking of the movement "du manége," he continues, "voilà donc deux nouvelles impulsions."

movement might be due to loss of sight.* This is probably much nearer the truth, but it is not a sufficiently general expression, as it does not include Flourens' experiments upon the semicircular canals; and besides this, Buzareingues attributes the motion to volition, which cannot in many instances be present;—"L'animal se porte constamment sur le côté où il voit, afin de s'éviter de heurter du côté opposé."

Mr. Herbert Mayo conjectured that a "sensation analogous to vertigo" was the cause.† Flourens, in the memoir detailing his experiments, uses this form of expression:—"La section de tel ou tel canal. . . . la section de tel ou tel genre de fibres *détermine* tel ou tel mouvement;" but Chevreul, in an analysis of this memoir (a criticism characterised by Flourens himself as "savante et profonde"), makes this distinction, "C'est *l'absence* de ces canaux, et non leur *présence* qui est la cause des phénomènes si singuliers . . . il faut les considérer, non comme des organes qui *produisent* les phénomènes en question, mais comme des organes qui les *empêchent* au contraire de se manifester."‡ This view of the question was adopted by Flourens, who, in a subsequent edition of his memoirs, expresses himself thus,—"La section *laisse éclater* le mouvement;" and afterwards, "l'action des canaux semicirculaires et des fibres opposées de l'encephale est beaucoup plus une action qui *modère*, une force qui *régit*, qui *contient*, q'une force qui *pousse*, et qui *détermine*."§ In this conclusion M. Flourens is probably correct; but the phenomena do not warrant the following:—"L'effet *modérateur* réside enfin, tout à la fois, et dans les canaux semicirculaires, et dans les fibres opposées de l'encephale."‖ Longet's experiments on the eye produce rotation when the parts refered to by Flourens remain intact. Hertwig, who confirmed, in main points, the experiments of Flourens, had already drawn these conclusions:—1mo, pontem magnam vim ad æquilibrium in corpore servandum exercere. 2do, sectiones longitudinales in ponte factas æquilibrium inter utrumque corporis latus, sectiones vero ex transverso æquilibrium inter

* Journal de Physiologie. Tome VIII., p. 322.
† Outlines of Physiology, p. 229. Ed. IV.
‡ Journal des Savants, 1831; p. 10.
§ Recherches: 2nd Ed., p. 496.
‖ Op. cit., p. 499.

anteriorem et posteriorem ejus partem turbare.* Inasmuch as Hertwig recognises the tendency to *equilibrium* rather than to *motion*, he appears to interpret the phenomena correctly; but it is very doubtful if he has correctly located the function. Magendie's theory of impulsive forces is little more than a statement of phenomena; but Hertwig's conclusion is an induction intended to account for them.

In order more fully to appreciate the function of *moderation*, we should recall the conclusion to which Sir Charles Bell arrived, as stated in a communication to the Royal Society in 1826. It is, that "between the brain and the muscles there is a circle of nerves; one nerve conveys the influence from the brain to the muscle, another gives the sense of the condition of the muscle to the brain."† The fact of the existence of this "muscular sense" (whatever view may be taken of its mechanism) rests upon the same kind and amount of evidence as that which we possess for any other sensation: we are conscious of its existence. The relation of this sensation to others, and to muscular motion, has been somewhat overlooked (it appears to me) by MM. Longet, Lafargue, and Schiff, in their recent attempts to explain the phenomena of rotation. Lafargue says, "Il suffit de réfléchir sur le mécanisme de la locomotion normale des quadrupèdes pour voir, qu'étant données deux conditions, la *chute sur un côté paralysé*, et *l'activité isolée de deux* membres, les efforts de ceux-ci produiront la rotation selon l'axe, par cela même qu'ils agiront seuls en poussant tout le corps vers le côté faible."‡ Schiff's explanation is somewhat different:—"Suppone in homine partem thoracicam columnæ vertebralis ita deviatam et rotatam ut pars ejus pectoralis versus unius lateris ossis ilei spinam anteriorem superiorem trahatur: adde paralysin subitam musculorum sterno-cleido-mastoidei, scalenorum, et lateralium colli lateris oppositi, musculos paralyticos suppone facultate contractionis voluntariæ, sed non tono musculari, privatos, ita ut iis post insolita extensione in statum mediæ contractionis recidendi insit conamen, et ille homo, desiderio tractionem incommodam fugiendi, et æquilibrium restituendi coactus, circa axin rotationem faciet."§ Longet differs from Lafargue, but

* Exp. quædam de effect: lasion: in part: encephi: &c., 1826, p. 23.
† The Nervous System (by Bell.) Ed. 1844, p. 194.
‡ Thèse cit, and Longet's Traité.
§ De vi motoria, &c., p. 52.

in the main agrees with Schiff. He attributes the rotation "Non à la paralysie des membres d'une côté et à l'activité persistante des membres de l'autre, mais, suivant le cas, à une paralysie directe ou croisée qui a atteint, dans un côté, les muscles de la nuque et ceux des portions cervicale et dorsale de la colonne épinière. . . . En effet on voit la rotation commencer dans ces portions qui bientôt entraînent avec elles la portion lombaire, et les membres abdominaux."* Longet bases his dissent from Lafargue upon the fact that, "L'animal étant tenu sur le dos, on voit les quatre membres s'agiter en désordre avec une certaine énergie qui ne parait pas differer à gauche et à droite;" and upon the commonly observed negative results of hemiplegia. With regard to these explanations, I may say, that they appear equally insufficient; the marked similarity subsisting between the effects of internal and external injuries, and the fact of vertiginous movements resulting from the latter, indicate another mode of causation. There are in Longet's own experiment upon the eye, and in that of Flourens upon the ear, none of the conditions described by Schiff, and adopted by Longet himself.

Before giving what appears to me the correct explanation of these phenomena, I may allude to another theory, advanced by Alexander Walker in his treatise upon the nervous system. This book is a somewhat humiliating collocation of dogmatic but unproved assertions; of what Romberg would have called "hypothetical lumber," and of self-laudation. Referring to Flourens' experiments on the semicircular canals, he remarks, "I have shown that hearing is the sense which excites the passions (as touch does ideas, and sight emotions), and which, therefore, most naturally and directly produces muscular action."† Now, the strange phenomena of Tarantulism, and other forms of the dancing mania (of which an interesting account is given in Hecker's Epidemics of the Middle Ages)‡ are sufficient to exhibit the power which impressions upon this organ of sense may exercise upon a previously disturbed nervous system; and it is probably true that, in general, the impressions which appeal to our deepest

* Longet's Traité, p. 217.
† The Nervous System, 1834; p. 684.
‡ Sydenham Society Translation, (of Hecker) p. 88, *et seq.*

feelings and emotions make the ear their medium.* But as vertiginous movements occur when the eye is blinded, Mr. Walker's explanation becomes utterly fallacious; and, farther, it is not easy to say what variety of "passion" these rotatory motions could express. The phenomena are of a nature inexplicable by states of emotion or of mind; and many of them have been witnessed when the cerebral hemispheres were removed.

The several *organs*, after lesion of which these movements occur, have been already enumerated; (p. 15) and if we now recall their *functions*, they may be resolved into:—

1. The centre of co-ordination.

2. The centres of common sensation and motility.

3. The organs of the special senses (particularly of sight and hearing).

4. The fibres placing these organs in functional relationship.

Or it may be more briefly stated thus: the vertiginous motions follow injuries to the organs of special sense, the organ for the co-ordination of movement, and the fibres connecting the latter with the former, or with the systemic muscles. In either case, some part of the nervous system, or its appendages, ministering to the "*consensus* of the nerves" is injured, and the mysterious conversion of sensorial impressions into motor impulses is disturbed, or prevented from taking place.

The facts of daily life prove our dependance upon sensations for guidance in almost all our motor exercises. The tottering gait and fearful attitude of one who has but recently lost his sight, and who is yet unable to appreciate fully the indications of touch and sound; the wandering divergence of his eye-balls, an inimitable demonstration of his loss; his pitiful helplessness, which makes one of our German poets exclaim,

"Sterben ist nichts, doch leben und nicht sehen
Das ist ein Unglück;"

the strained and discordant voice, or absolute dumbness of those who hear no sounds; the clumsy accidents of those whose "muscular sense" is lost; and the common experience of every one who has, when unaccustomed to the attempt, to walk upon some narrow plank unsupported by the sight of objects near him, are sufficient examples of our dependance upon the indications of sense. Dr. Wilson Philip said, as "the muscular system obeys the nervous, the nervous obeys the sensorial system;"* or, as Dr. Alison expresses it, "the will of the animal is constrained (by the conditions of its nervous arrangements) so to act, as to excite certain movements only;" and, farther, the disordered motion resulting from experimental lesions may be supposed to occur "either, because the injury produces permanent uneasy feelings, such as vertigo, which interfere with, and confuse the *sensations* by *which voluntary movements are regulated*," or because of "a loss of the recollection of muscular sensations."† Guided by the latter, and by the objects of the world around us, we perform, voluntarily it may be, but not by any distinct effort of the will, and sometimes unconsciously, the greater number of our daily exercises. In no case do we, volitionally, determine the muscular contractions which shall take place, but only the movement which is to result from their combination; we leave to the nervous system with which we are endowed the intermediate steps of the process. States of mind, of emotion, and of physical suffering, betray themselves not only in "expressions of the face," but in attitudes and gestures, which are as independant of the will as is the beating of the heart. It is thus that, in subjection to the laws of that organism with which it is so mysteriously blended, the immaterial mind reveals itself through the material body; and by an inverse process (of sensation and perception) as determinate and irresistible, another mind can interpret the revelation that is given, and recognise its cause. But, as by the exercise of in-

* Mr. J. D. Morrell (in his Elements of Psychology. Part I., p. 114) has made some excellent observations upon this subject, and to his book I am indebted for the following passage from Erdmann's "Psychologische Briefe":—"It is not the form and colour of an object which tells us what it is, but its sound. For that reason, the sight of a thing does not penetrate so much to the heart; it only tells us what is its appearance. On the other hand, the tone moves us; it tells how the thing or person stands to the heart itself. . . . Inasmuch as sight gives permanence and certitude, I write a bill in black and white, and that gives conviction. If I want to be moved, however, I must hear. You may read many a thing quietly, which, if you read it aloud, would make your very voice tremble."

* Exp. enquiry into the laws of the vital functions. Ed. 3rd, p. 92.
† Outlines of Human Physiology, 1839; p. 368.

genuity and a cultivated will, variously combined movements may be produced, for which (if I may use the expression) the nervous system possesses no formulæ among its connate endowments; so, to a certain extent, some of the associations which it (the nervous system) unconsciously effects may be modified, prevented, or corrected, by prolonged and determined effort.

The combined actions thus produced are easily performed after several repetitions, and then they may be put in exercise without volitional direction. They become "secondarily *automatic*" by our creating (nervous) formulæ for their guidance.* The lower animals probably depend much more extensively upon the indications of sense than those possessed of greater intelligence, spontaneity, and educability; and, a fortiori, much more closely than man. His energy of volition is displayed in subjecting thought, emotion, and muscular action to the dictates of judgment; and it is certain that the power of so doing varies widely in individuals. In accomplishing the subjugation of the latter (muscular action), and in its direction to definite ends, he is guided mainly by sensation, and principally by touch, sight, and the muscular sense; and all our involuntary attitudes, and motor impulses to change an unpleasant position, are directed in the same manner. There is a feeling of physical rest, or equilibrium, which we strive (it may be involuntarily) to attain by certain movements, and in this effort we are guided by impressions from without, and by the sense of our own muscular conditions. This feeling of equilibrium results from the harmony of our different sensations among themselves, and with the motor impulse which is their combined effect. When, therefore, any one group of the sensorial impressions is distorted, or removed, the balance is, pro tanto, disturbed, and inasmuch as these impressions are themselves the stimuli of muscular action, attempts are made for its restoration. In the lower animals these attempts become much more marked than in man, producing vertiginous, or allied movements, which he, by a judgmatic discrimination between the inharmonious impressions, and by a volitional corrective power (which they do not possess to the same degree) is able to avert.

The vertiginous movements are, then, the result of an effort

* For a most able exposition of these questions, I would refer to the last edition of Dr. Carpenter's Principles of Human Physiology; p. 740, et seq.

to produce equilibrium, an effort developing itself in muscular action, through the agency of the nervous system, and under sensational guidance. When the cerebellum itself is injured, the power of co-ordination is, pro tanto, gone; but when the organs of sense, or the tissues connecting them with their centres, or the latter with the cerebellum are destroyed, this organ loses its instructions, or receives them only in a one-sided, and distorted form.*

Having given this sketch of our knowledge with regard to the phenomena of vertiginous movements, and the conclusions which we are justified in forming upon their pathology, I pass now to the second part of my subject, to which, by common usage, the term vertigo has been limited; viz.:—

Vertiginous Sensations.—Almost every one has, at some period of his life, experienced more or less of these most unpleasant feelings; and it is, therefore, unnecessary for me to occupy much time by their description. The simplest *definition* appears to me:—the sensation of motion without (or independently of) its real existence. By some it has been termed an "apparition," (Willis); by others a "disease," (Mead); by others an "hallucination," (Sauvages); and by others one of the "hyperæsthesiæ," (Romberg). Sauvages gives this definition:—"Vertigo est hallucinatio, qua objecta, licet quiescentia, e loco moveri, vel in gyrum versari, nobis videntur. Ipse ager sibi quandoque nutare videtur;"† or, more briefly—"Apparens objectorum vacillatio, vel nutatio." The numbers of *varieties* or *species* of vertigo enumerated by different authors vary indefinitely. Thus Boerhaave says:—"Divido vertiginem in duas classes, morbosam et criticam."‡ Sauvages enumerates fourteen different kinds, their distinction

* With regard to the experiments of Flourens upon the semicircular canals of the Ear, we have some little difficulty, arising from the uncertainty of our knowledge as to the precise functions of these structures in health. Autenrieth, Körner, Duges, and many others, believed they were the means by which the *direction* of sound was ascertained. Professor Müller doubts this conclusion. It does not affect the general statement made above, although Flourens' observations would rather confirm the opinion of Autenrieth, &c. Without, however, intellectually *perceiving* the direction of sounds, it is possible that such direction may influence the motor apparatus; or that the canals themselves have some directive power upon the movements resulting from impressions on the Ear: and this especially in those animals which depend almost entirely upon undiscriminated sensational guidance.

† Nosologia Methodica. Vol. IV., fol. 256, Ed. 1746.

‡ Prælectiones de Morbis Nervorum. Tome II, p. 576.

resting mainly upon the supposed nature of their causation. Wepfer distinguishes three varieties—Vertigo titubans, vacillans, et gyrosa.*

It would be easy, but somewhat unprofitable work, to multiply instances of disagreement among authors upon this head. Instead of doing so, I prefer simply to draw attention to one kind of distinction frequently noticed, although variously considered in respect of its value as a basis of division. It rests upon the fact, that in many cases the apparent motion is referred (entirely or principally) to surrounding objects; whereas, in others, it is referred to the person of the individual himself. The former may be conveniently termed "*objective*," the latter "*subjective*" vertigo. These two groups may be formed independently of all pathological considerations; and, although the line which separates them may not be absolutely true in all instances, yet in the majority the individual is able to determine accurately which feeling is present, or (at all events) predominant.

The *conditions* under which these sensations (of vertigo) occur may, apart from any idea entertained as to their proximate causes, or pathology, be resolved into two groups. In the first of these, the cause is *external* to the individual, and is referrible to some change in his relationship to surrounding objects. In the second, no such change has taken place, and the cause is *internal*.

The *external causes* consist of unusual impressions upon the organs of sense; produced either by the movement of surrounding objects, or movement of the individual himself; or by his position in some totally unaccustomed relationship with the world around him. If the sole cause is the movement of external objects, their impressions are made almost entirely (it may be entirely) upon the sense of sight. With vertigo arising in this manner many are familiar; but some persons are much more susceptible of its induction than others. When the individual himself moves, or is moved, the organ of hearing, the muscular sense, and that of common tactile sensation are affected. At the same time, the relations of the objects of vision change, and thus the causes become complicated, and rendered more intense. That utterly horrible feeling of "sea-

* Observ: Anatomico-medicæ, 1727., p. 217, &c.

sickness" may (notwithstanding the ingenious theory of M. Semanas, see p. 38) be taken to illustrate this mode of causation. Riding in a close, or an open carriage affords another, and in many persons an equally distressing example. Rotation of the body, as in waltzing, or the rotations which children perform, as a somewhat morbid amusement, for the purpose of seeing "the room run round" afterwards, are farther and familiar illustrations. Elevation to a considerable height produces vertigo, with an uneasy feeling of falling, not to be entirely accounted for by fear; as a similar tendency, with apparent vacillation of objects, may arise by looking upwards: as, for example, from under the dome of St. Paul's, or even into the sky, when there are no houses or trees near at hand to break the line of vision, and support the gaze. It is almost as difficult to walk steadily upon the common ground, with the eyes thus removed from immediately surrounding objects, as it is to walk upon the edge of a cliff, or on the parapet of a tower. It is said that the European, in his first attempt at elephant-riding, is in great danger of falling, from vertiginous sensations to which the native is a stranger.* These are, with the exception of the last, easily verified examples of vertigo from unusual relationship with the objects of vision.

The *internal causes* are numerous, and it is not easy to speak of them without entrenching upon Pathology. Heberden says: "Hic morbus mihi visus est istiusmodi, quem crediderim sæpe oriri ex offensa quidam ventriculi, sæpius ex affectu capitis, sæpissime autem a totius corporis infirma atque adversa valetudine."† Almost every organ in the body has formed the specific name for a variety of the genus vertigo; but, (not to enter upon their several merits), we may, I think, recognise a natural basis of division into two principal classes. In the first of these, vertigo exists alone, or is accompanied only by certain nervous phenomena of an allied character: in the second, it is only one of a number of symptoms referrible to disorder of some remote organ, or to the organic system generally. Of course, it is impossible, in the present state of knowledge, to decide for all cases into which category they should be placed, but the concurring symptoms will generally enable us to do so. The causes appear sometimes to

* Darwin's Zoonomia. Vol. I., p. 231, &c.
† Commentarii de Morborum Historia. Ed. Frankfort, 1804; p. 350.

reside in the nervous system itself; at other times in the condition of distant organs; and thus they may be divided into *centric*, and *eccentric*; and the resulting vertigo into idiopathic, and remote.

With regard to the *centric* causes, it must be remembered that the essential condition of an "impression," or "stimulus," or "irritation" of the nervous system (using these terms in their broadest and most general sense, *i.e.*, as the external occasions of nervous action), is *change* from some previous state, and that it matters little what is the precise character of this change, its activity is in proportion to its degree. It is on this account that similar nervous phenomena frequently result from opposed conditions: it is not the precise nature of the morbid, or abnormal state, but the fact of its difference from the healthy, or previous condition, which induces the nervous action. Thus *variations in the quantity of blood* supplied to the brain produce some symptoms precisely similar, when on the one hand there is more, or on the other less, than usual. Congestion is a common cause; Anæmia is not less so; and all organic and functional diseases of the brain, which may induce temporarily these dynamic changes, may become causes of vertigo. Dr. Marshall Hall has drawn attention to the frequent concurrence of vertigo, and spasmodic muscular action in the neck. He conceives, that by contraction of the platysma, omohyoid, and cervical muscles generally, the venous trunks returning from the head are compressed, and the circulation through them impeded. In this manner congestion, and "various ulterior effects on the encephalon occur in their turn. Sometimes these assume the external character of a momentary oblivion, delirium, or vertigo."* Dr. Hall's views upon this subject have been misunderstood, and thought unsatisfactory by some, who, upon anatomical and other grounds, question the possibility of venous compression from transient muscular spasm. But his own expressions are sufficiently clear to remove this misapprehension. "It is by spasmodic, inordinate, and sustained contraction of the muscles of the neck, that the veins are so compressed as to lead to impeded or interrupted flow of blood along them."† Observation and experiment alike con-

* Synopsis of the Diastaltic Nervous System. 1850; p. 68, § 324, 325.
† Synopsis of Cerebral and Spinal Seizures 1852; p. 24, § 131.

firm this statement, but it is not for a moment intended by Dr. Hall that it includes all cases and varieties of vertigo, or other symptoms, having congestion of the brain as one of the links in their chain of causation.* We know so little of the modus operandi of *therapeutic* agents that it is almost impossible to classify them except by their resultant effects. Among these, vertigo is found after the exhibition of many; and it is probable that the mode of its proximate causation differs. Without entering into this question farther, we must admit a "toxic" cause. Purkinje describes the passage of a *galvanic* stream through the head (from ear to ear) as a most potent cause. "Man fühlt dann den Kopf eingenommen und einen allgemeinen schwindelhaften Zustand." It is, however, difficult to say how much of this may be due to impressions upon the organ of hearing, but the definite relation between the direction of the vertigo, and that of the current renders it probable that the brain itself is affected; for continues Purkinje, "Die Richtung der Kreisbewegung dieses Schwindels geht aufwärts von der rechten zur linken Seite, wenn der Kupferpol im rechten Ohre, der Zinkpol im linken ist;"† and *vice versâ*. Purkinje places last in his list of causes, lesions of the cerebral substance, as in Magendie's and Flourens' experiments.

Among the internal causes of *remote* origin we must place foremost, general or *systemic conditions*, such as the fevers, cachexiæ, &c., accompanied with more or less distinct toxæmia. And here, again, we must remember, that it is the fact of alteration from a normal state rather than the precise character of the change which induces vertiginous sensations. The *stomach* is placed in such close relation with the brain that its disturbances are most commonly attended with vertigo. Under all these circumstances there is one common fact, viz., an abnormal organic state, and there is surely no other impression to which the nervous system can be subjected so powerful to disturb its equilibrium.

* For farther elucidation of these principles, the reader is referred to Dr. Hall's republished Memoirs on the Nervous System, and to papers in the *Lancet* for 1850, 1851, 1852.
† Über die Physiologische Bedeutung des Schwindels, &c., in Rust's Magazin für die gesammte Heilkunde. Bd. XXIII., 1826; p. 297.

The remote causes may be divided, thus, into two classes :—

1. External.
   - Motion.
     - Objective.
     - Subjective.
     - Two combined.
   - Unusual position, (affecting principally the sense of sight.)
2. Internal.
   - Conditions of nervous system.
     - Circulation changes: anæmia hyperæmia, &c.
     - Organic lesions.
     - Toxic influences.
   - Conditions of general system.
     - Pyrexiæ.
     - Cachexiæ, toxæmiæ, &c.
     - Particular organic disturbances, stomach, &c.

PATHOLOGY.—With regard to the intimate cause of these sensations there is little possessing more than historical value among the ancient medical writings. In accordance with a law, which the great apostle of "Positivism," Auguste Comte, would make universal, Pathology has to pass through its theological and metaphysical stages. The indwelling *dæmon* of disease was expelled, only to give place to metaphysical *abstractions*. "Animal spirits" and "vital spirits" have to be conceived before vital forces can be admitted; and, by the aid of the former, a somewhat ingenious theory of vertigo was propounded by Dr. Willis. "Vero hujus apparitionis causa omnino dependet a fluxili spirituum animalium substantia. Quippe spiritus intra cerebrum scatentes non secus habent ac aqua, aut densa vaporum congeries phialæ inclusa, quæ una cum vase continente circumagitur, et facto semel vortice, etiam vase quiescentia, motum istum aliquamdiu continuare persistit. Pari etiam modo, quando hominis corpus circumgyratur spiritus cerebri incolæ, ab ista capitis, tanquam vasis continentis, circumductione, in motus tornatilis ac velut spiralis aguntur; eumque ideo solito influxu, et directo jubare nervos irradiare nequeant, hinc cum una visibilium rotatione, sæpe scotomia et pedum vacillatio inducuntur."* Mangetus, in his edition of the Sepulchretum of Theoph. Bonet, adopts this explanation.† Sennert does the same, but not so implicitly. According to him, "Vertigo est *imaginationis* ac *sensus communis* læsio ac depravatio talis ut omnia in gyrum circumagi videantur, a circulari spirituum animalium motu in cerebro ortum habens."‡ Haller has the following passage :—"Vertiginem esse apparentem objectorum rotationem, indeque sequentem membrorum vacillationem, et ab inordinato, et inæquali spirituum animalium in fibrillulas nervorum opticorum influxu circumgyrationis ideam in mente excitante originem ducere notum est."* And Wedel expresses himself in similar terms :—"Causa immediata est spirituum animalium motus indirectus et confusus."†

* De anima Bruterum. Op. om. Cap. IV., sect. 6, p. 129.
† Sepulchret. in Anat. pract.; p. 262.
‡ Danielis Sennerti, op. omnia. 1656. Lugd.; p. 71.

As the idea of these animal spirits gradually died out of the human mind, we find less circumstantial accounts of the hidden processes of nature. "Quæro nunc porro," said Boerhaave, "quænam sit sedes hujus mali? . . . Respondeo, sedem illam esse in sensorio communi, non dico causam integram, sed ultimam affectionem, quando vertigo infestat, esse in hoc loco. . . . Hoc phantasticum est illa mentis facultas, quæ res absentes sistit tanquam præsentes, et quæ sensus externos et internos motusque animales facit; sed hoc sit in sensorio communi, ergo in eo vertiginis sedes est."‡ Dr. Richard Mead said, "It is very often more a disease of the stomach than of the head, or at least, both these parts are affected together, from a quantity of bilious and viscid humours lodging in the guts." He insists, moreover, upon its near relation to epilepsy, and one reason for maintaining this opinion is, that it "obeys the lunar influences."§ Van Swieten, closely following Boerhaave, asserts that, "the whole common sensory is involved, but then especially a vertigo threatens an apoplexy, when the cause of the former is lodged within the cranium, and then the vertigo is called idiopathic."‖ Here Van Swieten has alluded to vertigo complicated with hissing noises in the ears, dimness of sight, tremors, &c. Sauvages makes vertigo the first genus of his order Hallucinationes (of the class vesaniæ). "Hallucinatio est error fugax qui pendet a solo vitio *organorum externorum*, quique ab ipso judicio et reflexione saluis facile corrigitur;" and, on the other hand, "Delirium est error constans . . . . . quique pendet a vitio *sensorii communis*."¶ It is quite fair to establish a distinction of name between those perceptions of an individual, not recognised by others, which, in

* De visu duplicato: (e disp. ad morb. hist.) 1746; p. 349.
† Disp. medic. sist. ægrum lab. Vertigine. 1682; p. 7.
‡ Prælectiones de morb. nerv. 1761; p. 576, &c.
§ Medical Works. 1762; p. 180.
‖ Commentaries. 1765. Vol. X., p. 143.
¶ Nosologia Methodica. Lips. 1786. Tom. IV., p. 256.

the one case, he is able to correct, or to consider as delusions, and which, in the other case, he is not, but believes them real; but Sauvages has done much more than this, and is certainly wrong in asserting that the former always have their origin in the external organs of sense, and the latter in the sensorium commune. It is quite competent to Sauvages to place vertigo among (or call it one of) the hallucinations, but his definition of the latter is incorrect, for vertigo is quite as frequently dependant upon a condition of the common sensory, as upon a modification of the external organs of sensation. The explanation which he gives of its mechanism is totally without proof, and does not account for the facts presented. "Objecta moveri videntur, vel quia reapse moventur, vel quia illis immotis, oculi nostri, aut quædam eorum partes, nobis insciis, moventur, eo modo, qui ad successivam objectorum in oculi partibus diversis delineationem requiritur;"* and the probable solution which he gives is, that the crystalline lens, or the globe of the eye itself, alter their position and form; or that the blood assumes a retrograde course in its passage through the retina. It is obvious that these hypotheses are utterly untenable.

Darwin,† and Marcus Herz,‡ viewed the phenomena from another stand-point, and their explanation was received by many; as, for example, by Crichton, who uses almost the same words as Darwin. The phenomena are considered psychologically, thus—"when *mental* representations and ideas crowd involuntarily, and in too quick succession, they occasion the disease called vertigo."§ This is doubtless true in some cases; but it is inapplicable to the majority, as they have nothing whatever to do with mental representations, or ideas. Dr. Mason Good criticises these views of its pathology, and remarks,—"Common as this complaint is, I have not yet met with any satisfactory explanation of its cause." His own theory is somewhat singular. Holding that the nervous force is always communicated "in jets," and not in a continuous current, he refers vertigo to "a clonic action of the nervous fibres subservient to perception;"‖ and thus, he himself

* Op. cit.; p. 260.
† Zoonomia. 1796. Vol. I., p. 231.
‡ Versuch uber den Schwidel. Ber. 1791.
§ Crichton's Nature and Origin of Mental Derangement. 1798. Vol. I., p. 324.
‖ Study of Medicine. Vol. IV., p. 525.

makes it to be the sensory analogue of choreiform movements. It is not very easy to see what Dr. Good intended; moreover, it is by no means certain that this jet-like behaviour of the nervous force is a fact; and if it were, it is impossible to understand how its occurrence in the "nervous fibres subservient to perception," could produce the appearance of vertiginous movement, which is continuous in its character. Interrupted sense impressions would result, but their effects would be totally different, and might bear some relation to choreiform movement. In fact, this analogy, which Dr. Good has himself instituted, disproves the correctness of his theory.

In the Dictionnaire des Sciences Médicales,* there is little attempt to unravel the mystery. We find only the following general expression:—"Cette espece d'hallucination parait dépendre d'une compression du cerveau, par suite de la plénitude passagère des vaisseaux sanguins. Il y a lieu de croire que les vaisseaux opthalmiques sont surtout le siége de la pléthore qui détermine cet état nerveux." This statement, and the next, "Ce symptôme . . . dénote toujours une congestion cérébrale,"† indicate the appearance of a new era in the pathology of nervous phenomena. The study of animal spirits, mental phantasmata, and dynamic changes in the process of perception, has been supplanted by that of simple physical conditions. To these now the phenomena are referred; and there is perhaps no more ingenious theory upon the subject than that given by Purkinje, in a paper already noticed. Near its commencement is the following:—"Von nun an wird dieser (Schwindel) in jenem Capitel der Physiologie seine Stelle finden, wo vom Einflusse des Gehirns auf die Anordnung und das Gleichgewicht willkührlicher Bewegungen die Rede ist, denn er ist nichts als eine Störung der dynamischen Beziehung des Gehirns zum Bewegungssysteme des Körpers, die sich in diesem als eine einseitige Aufregung äussert." Notwithstanding this statement, Purkinje gives an extremely mechanical explanation of the process. But we must notice, in the first place, the results of some carefully executed, and painful experiments performed by him upon his own person. "Vor allem kömmt uns hier die alltägliche Erfahrung entgegen, wo durch wiederholte

* Art. Vertige, (dict. des sc. méd.) 1821.
† Dictionnaire de Med. 1828.

Umdrehungen um die eigene Körperachse ein Schwindel erfolgt. In diesem Falle scheinen die Gegenstände in horizontalem Kreise . . . zu laufen, hierbei fühlt man im Muskelsysteme eine Neigung den Körper nach derselben Richtung noch ferner umzudrehen, welche Neigung im Verhältnisse zur primären Einwirkung steigen, ja bis in wirkliche unwillkührliche Bewegungen ausbrechen kann. Am besten fühlt man diese einseitige Tendenz des Bewegungsapparats, wenn man während des Schwindels die Augen schliesst, und auf das tastbare Druckgefühl an den Fussohlen, oder auf die sich an irgend einen Gegenstand anstemmenden Hände achtet. Man fühlt dann ein seitliches Anstreben der Füsse gegen den Boden, oder der Hände gegen den Widerhalt, welches ursprünglich subjectiv, da es unwillkührlich ist, aufs object übertragen wird, und so als ein Drängen, und Drehen desselben erscheint."* This experiment is followed by many others of an ingenious character, the head being held in different positions during the the rotatory movement. Purkinje finds that, "Die jedesmalige Lage des Kopfes, und mit ihm des Gehirns, während der realen Umdrehungen, bestimmt die nochmalige Richtung des Schwindels bei einer angenommenen constanten Lage jener Theile nach eingetretenem Stillstande der Bewegung." But he advances a step farther, and asserts that "eine materielle Veränderung im Gehirne vor sich gehen müsse." He then points out the analogy between the effect produced by rotating a vessel upon the liquid it contains, and that of rotation of the body upon the brain. He sees that the cohesion of the brain to the inner surface of the skull is too firm to admit much movement (of the brain) as a whole, but concludes, "so muss man nothwendig annehmen, dass in dem Grade, als es von aussen in mehr oder weniger heftige Bewegungen versetzt wird, auch seine Theile gegen einander, wenn gleich ohne Continuitäts veränderung, in gleichem Grade verschoben, und gedehnt werden."

The similarity between this explanation and that given by Dr. Willis, nearly two hundred years before, is obvious. Purkinje did good service by drawing attention to the relation which subsists between the apparent direction of movements and the axis of the head; and to the effects produced upon the muscles,

* Rust's Magazin: (loc. cit.); p.p. 289, 293, &c.

the muscular sense, and that of touch; but his theory strikes us at once as too mechanical. It, however, fails to account for the phenomena. Mr. Wheatstone, whose ingenuity has furnished so many means for the increase of our knowledge upon all matters related to the sense of sight, has assisted us in the comprehension of vertigo. He has shown (and it is an experiment very easily repeated and confirmed), that if a large sheet of paper is held before the eyes during these rotatory movements, there is no apparent motion conferred upon external objects when the body is at rest. All that remains is a feeling of incertitude in the lower limbs.* In Mr. Wheatstone's experiment, no impressions are made upon the retina, but the rotation performed by the muscles of the legs induces its effect upon the muscular sense. On the other hand, vertigo arises sometimes from external causes only; and these two facts, taken together, demonstate, first, that if such internal movement (as Purkinje imagines) does take place, it cannot cause vertiginous sensations; and secondly, that the latter may occur in circumstances which preclude the existence of the former.

Müller, speaking of Purkinje's theory, says:—"We might perhaps better explain the phenomenon by supposing it to result from the impressions of the blood in a particular direction upon the nervous substance. It is possible, however, that the revolving motion of the body may cause an aberration of a more subtile principle than the particles of the cerebral substance or the blood; in fact, an aberration of the nervous principle itself, such as affects the sensorium, so as to produce the apparent motions of objects."†

Romberg, who defines vertiginous sensations thus, the patient "feels as if he were going to fall, or were turning round, or as if everything else were doing so," and who refers to Purkinje, as having "first introduced this matter into the range of experimental physiology," tells us that "the vertiginous sensations are to be distinguished from others that occur when the eyes are closed, and in blind people; these Professor John Müller has accounted for by referring them to the after effects of visual

* Mayo's Outlines of Physiology; p. 327.
† Müller's Physiology, Vol. I., p. 848.

impressions upon the retina."* It is not very easy to understand how visual impressions are made by Romberg to account for these sensations in the blind.† Aimé, Plateau, and others, have endeavoured to measure their duration, *i.e.*, the length of time a sensation lasts after the removal of its impressing cause.‡ But this is not of much importance to us now. Müller says it is "greater in direct ratio with the duration of the impression which caused it," and his explanation of the phenomenon is the following:—"If we gaze for a considerable time upon a moving object, the spectrum appears to move, owing to its (component) spectra disappearing from the eye in the same order."§ It is very certain that many cases of vertigo are centric in their origin, and have no causative relation to visual impressions, or their persistent spectra; but on the other hand, vertigo frequently arises from external impressions upon the visual organs alone; and the moving spectra which remain, whatever may be the theory of their formation, are an essential part of the vertiginous sensation. They pass into the latter by a gradual and unintermitting process, and it is absolutely impossible to find the point through which a line must be drawn for their separation.

Romberg concludes,—"The older nosologists assumed a false, relation between this variety of hyperæsthesia and consciousness inasmuch as they placed vertigo among the hallucinations. Modern writers have erred equally, in dividing the subjects into visual and tactile vertigo. . . . It is explained as *hyperæsthesia of sensitive muscular nerves.*"‖ With regard to "the older nosologists," it must be remembered that many of them (*e.g.*, Sauvages and others), who considered vertigo to be an "hallucination," did not mistake its relation to consciousness. The term

* Romberg's Manual of Nervous Diseases. Syd. Soc. Vol. I., p. 99.

† One of the simplest examples of the persistence of impressions, is the apparent ring of light which is seen when a point only, such as the end of a match, is moved rapidly so as to describe a circle. The impression made upon one portion of the retina remains during its production upon others, and the effect is that of a continuous line rather than of a moving point. If the eyes are fixed for some length of time upon a moving object, for instance, a running stream, there is an appearance of motion conferred upon bodies at rest when the eyes are turned towards them.

‡ See Longet's Traité, (art: vision.) Matteucci's Lecons sur les phénomènes physiques, &c., p. 400; and Müller's Handbuch.

§ Müller's Physiology, by Dr. Baly. Vol. II., p. 179.

‖ Op. cit.: p. 100.

hallucination was used by them as we more commonly apply "illusion" now; and allowing their definition of the former (as an arbitrary term) vertigo was correctly placed. Others, who have explained vertigo by mental conditions, do not so frequently use the word hallucination. The division into visual and tactile vertigo, is one which exists naturally, and Romberg himself allows the distinction, but concludes, as I think erroneously, that the latter is not vertigo at all, and that the term must be restricted to the former.

It appears to me, that the two classes of phenomena are the same in kind, their difference being referrible to the quality of the sensation affected, and not to the manner in which it is disturbed. The same irritation (for example, the galvanic) applied to different nerves causes, in one case, muscular contraction, in another sound, in a third flashes of light, and so on. In a similar manner, impressions of motion, affecting on the one hand the muscular sense, and on the other the sense of sight, produce analogous effects upon the two systems of organs: in the former, there is the feeling of movement; in the latter, the appearance of it in external objects.* The word "hyperæsthesia," does not appear to me to be the most suitable. It cannot be shown to exist in vertigo of external origin, and it is questionable whether it may be fairly applied to the centric form. A sensation, arising from an ordinarily adequate cause, cannot be referred to hyperæsthesia, on account of its peculiar character; and when entirely subjective in origin, it may be that the muscular sense is only secondarily affected. It is quite immaterial what word is used, provided that it conveys a definite idea of the condition intended; but it is very important that when a word in common usage is employed, we should attribute to it no more and no less than its ordinary meaning. Dr. Romberg's definition of hyperæsthesia is, "exalted irritability, and increased irritation of the sensitive or centripetal nerves,"† and it does not appear to me

* It is not intended that these spectra, dependant upon the persistence of an impression upon the external organ only, can account for all the phenomena; but that they do explain some, and that they are the same in kind, whether referred to the sensitive muscular nerves, or to those of the visual sense. It is shown, farther on, that there is probably a centric condition of disturbance present in the more severe cases of vertigo. See p. 42.

† Op. cit. Vol. I., p. 6.

that the term can be applied in this sense to the phenomena of vertigo, which are frequently nothing more than subjective sensations, implying no exaltation of irritability whatever.

Certain impressions act so much more powerfully upon some individuals than upon others that we are obliged to assume the presence, in their cases, of peculiar idiosyncracies. For example, M. Sandras mentions the case of a strong man, who "ne pouvait pas marcher sur les trottoirs d'asphalte sans éprouver des vertiges bien caractérisés;" also that of a lady, who "ne pouvait pas manger un peu de sucre sans se sentir la tête et les sens tout bouleversés."* It is not at all uncommon to find that certain sounds, particularly when of droning or monotonous character, have a similar effect. These examples are curious, and might be multiplied indefinitely, in some individuals the impressions upon one organ being so much more powerful than in others.

M. Sandras makes no attempt to explain vertigo, but he describes several varieties, distinguishing them by means of their remote causes. M. Pellarin, writing upon sea-sickness, contends that it is "un résultat de la diminution de la force ascendante du sang dans l'aorte, et dans les artères qui naissent de la croise par suite des mouvements que le corps subit."† M. Semanas, considers the Mal de Mer "une intoxication produite par des miasmes et semblable à l'intoxication paludienne," of which the "symptomes vertigineux" are the first degree.‡ There is much to be said upon both these theories, but they refer only to one species of vertigo, and its special consideration does not form a part of my present object.

Müller, writing upon vertiginous sensations, says: "These *sensorial* phenomena afford a very interesting parallel to the revolving *motions* of the body (described by Magendie and Flourens) caused by a disturbance of the equilibrium of nervous actions in motor parts of the brain."§ It is to this parallel that we must look in order to comprehend the pathology of the former. The feelings of vertigo may be resolved into subjective sensations; and we must distinguish carefully the two methods

* Traité pratique des Maladies nerveuses. Tome I. p. 306.
† Bulletin de l'Academie de Médicine. Tome IX., p. 118.
‡ Gazette des Hopitaux. Mars 1847, and Valleix. Syst. de Medicine. Vol. V., p. 756.
§ Handbuch der Physiologie. Vol. I., p. 846.

by which they are induced. In one, the subjective sensation follows immediately some objective impression, of which it is the spectrum, and persistent after-effect: in the other, there is no external cause, and the sensation must be referred entirely to an idiopathic change in the nervous functions.

Our sensations are, in reality, although not recognised as such, modifications of our own being, and not phenomena of the external world. And, although the properties of material objects are often the occasions of their production, there is scarcely a sensation we derive from without which may not be reproduced from within alone. There is, probably, some inner harmony between the forces of external nature, and the ideal changes which they are destined to effect; but we can neither grasp its character, nor say wherein it lies. In all sensation, that which immediately affects our consciousness is the altered condition of our own organism. But by a curious connate endowment, or by the teachings of experience, we are led irresistibly to project out of ourselves, and into the external world, not only the causes but the phenomena of many of our sensations.*

We do not refer the objects we see to the retina upon which they are depicted, but to various distances from our person. We imagine sounds to exist, not in our ear, but in the next room, house, or street, as the case may be. We refer weight to the body we are lifting, and not force to the muscle which does

* That we do this, whether our sensations are derived from external impressions, or are the product of simply subjective conditions, appears to me to indicate that the impulse from without does not form a necessary part of the sensation process. Mr. Morell says:—"Externally, there is an appropriate impulse; internally, there is a direct perception of truth. . . . The result, as far as regards the nature of perception is this: that our immediate experiences of the world without are mental phenomena which arise out of the direct conflict of mind and nature; resulting, therefore, neither from the mere operations of the one, nor the mere impressions of the other, but from a combined and harmonious action of both." This may be a correct statement of the case with regard to sensations of objective origin, but the "conflict of mind and nature," does not take place when sensations arise from subjective conditions only. Instead of "a direct perception of truth," there is the perception of anything but truth; it may be of something which has no real existence whatever—as the phenomena of dreams, &c., indicate. Subjective sensations, in appearance as true, and in reality as false, occur in the waking state: and we must, I think, consider their apparent "outness" due to the same law, and their essence, or immediate action, due to the same conditions, in both instances. These conditions are modifications of our own organism. The external impulse is doubtless necessary for the perception of *external truth* (if this expression may be used), but it is not necessary for the perception of *sensations*.

the work. And again, we have a strange disposition to set ourselves in opposition to the real facts of the case; as, for example, when watching a moving picture, there is the tendency to refer its motion to ourselves, and imagine that we are sailing past a stationary object. And, on the other hand, in riding rapidly the trees and furrows of the field appear to move, and our own body to be at rest.*

In the former part of this paper we have seen that distorted impressions upon the organs of sense induce vertiginous movements; in the latter, that they also induce vertiginous sensations; and as the first may arise from internal lesions (not interfering directly with the organs of sense), so the second may have an internal or subjective origin. As rest results from the opposition and balance of contending motor forces, under the guidance of sensorial impressions, a disturbance, or irregularity of the latter, their distorted conduction, or a derangement of the organ for their direction and co-ordination, destroys this rest, induces a sense of lost equilibrium, and an attempt for its restoration. In the lower animals, whose equilibrium was destroyed by the want of bilateral symmetry in their sensations, this attempt became a definite movement of vertiginous or rotatory character; the animal probably, by this rotation, correcting to a certain extent the harmony between its sense of motion and of sight. The starting-point of causation was, in many instances,† an external impression only, which there was no power to correct.

In man, a similar lesion to one of the external organs of sense does not produce such general effects; but, notwithstanding his corrective power, he cannot escape them altogether. For example, it is by no means easy for him, when unaccustomed to the effort,

* These conversions of sensation are among the most wonderful of vital phenomena. They confirm the view already adopted—that the immediate condition of sensation is some organic change. It is questionable whether it occurs in any form without some impulse of this character; but, at the same time, these conversions (whether voluntary or involuntary) prove that in our act of perception, or reference of sensation to external objects, we are influenced as much, if not more, by mental states than by the special quality of the organic change. The latter being given, it rests with the mind (guided by experience, or by the combination of renewed impressions) to throw out and fill up the picture of external conditions to which the sensations shall be referred. This is true with regard to many sensations occasioned by impulses from without, but applies much more fully to those which are purely internal in their origin.

† For example, in the experiments of Flourens upon the semicircular canals of the ear; and of Longet upon the eye.

to walk or write rapidly in a straight line with one of his eyes closed. But in order to affect him more deeply some greater disturbance of his sensations is required. This is easily obtained by sending him to sea, placing him on the parapet of a tower, or turning him round and round several times, when the disturbed train of sensorial impressions induces not only the feeling of vertigo, but the tendency to movement, as Purkinje so well described.

The sensorial conditions are so distorted that the feeling of equilibrium is gone, and with this feeling the capacity for its production. The onesided impressions upon the organs of sense are themselves the stimuli (or occasions) of muscular actions, which result in vertiginous movement. Generally, however, in man, the tendency to movement is arrested by an effort of the will, per se, or by grasping some object for support. The sensation of its production remains, and is projected outwards into the objects of the material world. The individual feels as if one hand and one foot were pressed forwards, or that they moved forwards, and the others in an opposite direction; and, at the same time, the room appears to move round him in a circle. The arrest of real motion, when there is the sensation of its presence, necessitates the appearance of movement in surrounding objects.

The nerves of muscular and visual sense have been alike disturbed, and spectra of the impressions made on them remain; in one group of organs inducing the feeling of personal, in the other that of objective motion. The slighter degrees of vertigo may be nothing more than this; but when it is more severe, there is probably an induced condition of disturbance in the centric organs, analogous in character, but more persistent and intense in its effects. The length of time during which the irregular impressions were produced, or their greater severity, may be the cause of this internal disturbance.

When consciousness is obscured, as in a drunken man, there is much less power of correcting the erroneous sensations, and then, very frequently quasi-vertiginous movements occur; but by self consciousness, discrimination, and volition, man, in his normal state, is able to overcome slight derangements, and to compensate for the loss of one sense by the cultivated activity of another.

The vertiginous sensations of external origin may thus, I think, be referred to a confused state or disturbed equilibrium of the

sensori-motor system; induced by strained or unusual impressions upon the organs of sight and the nerves of muscular sense. But (as Müller has so well and strongly urged) all our sensations from external causes may be produced by the intrinsic conditions of the nervous system alone, and thus the feeling of giddiness may have a subjective origin. It is probable that it depends upon a condition of the sensori-motor apparatus, analogous to that described above; *i. e.*, a condition of the centric organs which disturbs or destroys the impressions made upon them from without; producing false sensational effects, which (by the law already referred to) are projected outwards into the objects of the external world. The kind of derangement thus induced is probably the same as that which follows prolonged rotatory movement, whether this has been objective or subjective in its character. In one case it may arise from a disturbance of the sensorial; in another, of the motorial; and in a third, of the connecting portions. Future researches may enable us to determine the particular part affected, but in the present state of science we are unable to do so. Whatever is the view which we take of the precise mechanism of the phenonema in question, I do not think that we can help referring them to those portions of the nervous system, and its appendages, which bring the various impressions from without into harmonious relation with each other, and with the motor impulses from within; and this, in order to establish equilibrium and rest, or to direct motion for the accomplishment of a definite end. These portions are:—the organs of sense, the sensory ganglia, the medulla spinalis and oblongata, with the cerebellum, and their various connecting fibres.

There are some farther observations which appear to confirm this view. The vertigo arising from revolution of the body, is easily corrected by movement in the opposite direction, which, by equalising the sensorial impressions, restores the balance that had been disturbed. Hyperæsthesia will not explain this result; nor can it be accounted for by the theory of persistent impressions alone. It is to be remembered, with regard to the latter, that real objects are seen in apparent motion, and not spectra of things which have been looked upon, and from which the eyes are now removed.

The relative effect of fresh sensorial impressions upon vertiginous

feelings of objective and of subjective origin is interesting. Great relief is experienced to the former when the eyes are closed, but considerable increase of the unpleasant feeling is produced in the latter. The giddiness arising from the appearance of shifting lines, (as of the surface of the sea,) may be removed by shutting out the view, and at the same time placing the body in such a posture that its muscles are as little affected as possible by the movement of the ship: but, after alcoholic stimulus, there is often a feeling of natation, when the eyes are closed, that may be at once removed by fixing them upon some stationary object. If a fixed point, or line, such as the horizon-line, can be steadily gazed at from the vessel, much of the giddiness of sea sickness is removed. In either case, the explanation is simple—the disturbed balance is restored by shutting out the distracting cause, or by producing some strong and steady impression upon which it may rest. The feeling of natation, when the eyes are closed, is vertigo referred principally to the nerves of muscular sense, but if consciousness is not too far obscured, this subjective sensation is removed by the correcting power of sight.

With regard to derangements of the general system, or of some particular organs, it must be remembered, that with the whole of our extended organism the nervous centres are placed in intimate relation, receiving from its several parts indications or impressions which become the impulses to varied acts and functions; and that the balance of conscious health depends upon a thousand unconscious stimuli. The effects which similar static or dynamic diseases of different organs induce through the nervous centres vary widely; there is sickening depression from one, irritability from another, and dull hypochondriasis from a third; but from almost any disturbance there may be vertigo, although it is perhaps more frequently connected with derangement of stomach and liver than of any other vegetative organ. Its mode of production is probably the simple effect of an unusual impression or sensation; and the degree of change from a previous condition (rather than its precise character or amount) is the measure of the impressing force.

Vertigo, like other sensational effects, obeys the law of habit. Circumstances which at first produced it, loose their power as they loose their special character of novelty. If that which was

unusual, and which was active by virtue of its strangeness, is frequently repeated, the source of its power is taken away. With regard to vertigo, the manner in which it is influenced by habit is twofold. There is, first, the simple diminution of change; and secondly, the increased corrective power, by cultivation of other senses. Mr. Herbert Mayo says very forcibly: "we lean upon our eyesight as upon crutches;"* and the man, unaccustomed to such exercises, feels that his support is gone when he attempts to walk upon the parapet of a tower, unaided by the sight of any object near at hand. But the rope-dancer learns to maintain his equilibrium by the sight of distant objects, and by the cultivation of his muscular sense; and the ship-boy stands steadily upon "the high and giddy mast," or is rocked to sleep

"In cradle of the rude, imperious surge."

It is said, too, that the Turkish Dervishes can perform any number of revolutions, and yet feel no vertigo.†

Doubtless, much of the insecurity which is felt is due to fear; but the mechanism by which confidence is inspired is the education of sensation. (For many illustrations of these subjects, and of others nearly related to them, the reader is referred to Sir H. Holland's chapter on "the effects of attention on bodily organs;"‡ to Mr. Herbert Mayo's Outlines of Human Physiology; and to Dr. Carpenter's Principles of Human Physiology.)

This paper has been intended, by its exposition of what appear to be the general causes of vertigo, to open a path for future investigation, rather than to present any practical results already gained. Published cases afford little addition to our knowledge, for most commonly the mere occurrence of vertigo is all that is put on record. In prosecuting the enquiry more deeply in those cases which have come under my own observation, I find marked differences in the phenomena, and in order to facilitate their examination. I have placed systematically in the following scheme, several points of interest, the accurate observation of which may guide us to a more correct appreciation of the symptom in question:—

* Outlines of Physiology, p. 325.
† Darwin's Zoonomia, 1795, p. 231.
‡ Chapters on Mental Physiology, p. 33, et seq.

1.—Frequency of occurrence, and readiness of production.
2.—Mode of production.
- *a*—by external causes.
  - *α*—by objective motion.
  - *β*—subjective motion.
  - *γ*—altered position—stating character of each.
  - *δ*—other impressions from without.
- *b*—internal conditions, referred to—
  - *α*—head, or nervous system, their character, &c.
  - *β*—particular organs.
  - *γ*—system generally.

3.—Duration and extent; affecting—
- *a*—vision only.
- *b*—muscular sense.
- *c*—complicated with other sensations of allied character.

4.—Special character, as stated by patient; whether—
- *a*—objective, apparent motion being—
  - *α*—circular, (horizontal, or vertical).
  - *β*—oscillatory.
- *b*—subjective; whether feeling of—
  - *α*—falling.
  - *β*—natation.
  - *γ*—rotation.
- *c*—two combined, which predominant.

5.—Relation to consciousness.
6.—Relation to movement; of—
- *a*—eyes.
- *b*—limbs.

7.—Relation to sensation generally.
- *a*—vision, cæcitas, muscæ. &c.
- *b*—audition, surditas, tinnitus. &c.
- *c*—smell, odor moschi, &c.
- *d*—taste.
- *e*—cutaneous, sensibility, anæsthesia, hyperæsthesia, formicatio, &c
- *f*—pain, its locality: nausea, &c.
- *g*—sensation of motion, natation, &c.

8.—Effects of fresh sensational impressions, &c.
- *a*—fixed gaze.
- *b*—grasping objects.
- *c*—postures.
- *d*—closure of eyes.

The cases which have fallen under my own observation hitherto are not sufficiently numerous to justify the formation of any general conclusions; but the constancy with which this symptom

presents certain characters in one group of cases, and different phases in another, induces me to hope that, at some future period, I may have the honour of laying before this society more valuable observations and more matured results. If the diagnostic worth of this symptom can be increased, not only will an addition be made to the science of Pathology, but we shall receive some aid in the more slowly-progressing science and art of Therapeutics. That such aid is wanted, every practical physician must have felt. It is not true, as Boerhaave has said, that "Vertigo est omnium morborum capitis levissimus et facillime curabilis."* The elaborate prescriptions of older medical authors, containing from one to thirty or forty ingredients in each dose, plainly indicate the difficulty with which they had to contend.† The directions of modern writers are less lengthy, saying that the treatment must be regulated by the cause, for the discovery of which they afford but little assistance. The causes and intimate pathology of this symptom are so varied, that its treatment can be only accidentally successful until guided by an accurate diagnosis. If the latter can be duly formed, the number of cases will be small with regard to which some future Swift might truly say:—

> "And that old vertigo in his head
> Will never leave him till he's dead.

If this paper has any influence in diminishing that number, its purpose is fulfilled.

* Prælectiones de Morbis Nervorum; p. 475.
† Vide Boneti Mercurius compitalitius, art. Vertigo, for numerous instances of these remarkable prescriptions.

PETTER AND COMPANY, PRINTERS, 102, CHEAPSIDE, AND NEW-STREET, DOCTORS' COMMONS.

ON

THE NATURE AND TREATMENT

OF SOME PAINFUL

AFFECTIONS OF BONE.

READ BEFORE THE MEDICO-CHIRURGICAL SOCIETY OF QUEEN'S COLLEGE, BIRMINGHAM, ON TUESDAY, OCTOBER 19, 1852.

BY LANGSTON PARKER,
PROFESSOR OF ANATOMY IN QUEEN'S COLLEGE; SURGEON TO THE QUEEN'S HOSPITAL; ETC. ETC. ETC.

LONDON: JOHN CHURCHILL.
BIRMINGHAM: JOSIAH ALLEN AND SON.
1852.

BIRMINGHAM:
PRINTED BY JOSIAH ALLEN AND SON,
2, COLMORE ROW.

ON THE NATURE AND TREATMENT

OF SOME

PAINFUL AFFECTIONS OF BONE.

THERE are many affections of bones, whose predominant character is pain, which are hardly to be referred to any of the more well known diseases of these parts, and which certainly are not ameliorated by the means generally resorted to for their cure or relief. The more common affections of bone, marked by severe pain, are inflammations of the periosteum; of the medullary membrane of the shafts of the long bones; of the bony tissue itself; circumscribed or diffused abscess of bone, and neuralgia. All these varied affections are accompanied by intense pain; in fact, pain appears to be the chief symptom of the disease, and in many instances is so great as to threaten the death of the patient, if not relieved even by so extreme a measure as the amputation of a limb.

The differential diagnosis between some of these affections is, in many cases, obscure, and even the

most experienced surgeons have often found it difficult to decide on the exact source or nature of the pain which has had its seat in a diseased bone.

It has appeared to me that many of these painful affections of bone are of inflammatory origin, and that the inflammation is seated in the medullary membrane of the shaft, or that which lines, imperfectly, the cancelli and canals of the extremities of long bones.

The chief anatomical characters of the medullary membrane of the long bones are extreme tenuity, great vascularity, and excessive sensibility. In many characters it differs essentially from the periosteum, which is neither so thin, so sensible, or so vascular. It is through the medium of the medullary membrane, which is supplied in each long bone with its special artery, that the whole of the internal structures of the bone are nourished and supported; and hence it is that death of the bone follows the destruction of the medullary membrane as the experiments of Troja have already proved; and it is equally probable that necrosis of portions of the shafts of the long bones may follow a partial destruction of the medullary membrane from inflammation.

I have just stated that the medullary membrane is very vascular, so much so that it frequently forms a species of hernia from the medullary canal of a long

bone in young persons after amputation, this protrusion is the medullary membrane turgid with blood.

It is also common to see the blood issue in a stream from the divided vessels of the medullary canal after an amputation, particularly if the amputation be performed high up, either in the arm, thigh, or leg. There is no question about the source of this blood, it is from the vessels of the medullary membrane. The medullary membrane, highly vascular, serves to nourish the internal layers of the bone, it possesses great sensibility and a high degree of vitality. The medulla, or fatty tissue of the medullary canal, is altogether insensible. It has been shown by vivisectors, that if a probe be introduced into the centre of the medulla of a long bone in a living animal, no sign of pain is produced as long as the instrument does not touch the walls of the cavity, but whenever the walls are rubbed or scratched, the pain becomes excessive, and is manifested by piercing cries or violent struggles.

A membrane so extended in its ramification, so sensible, so vascular, must necessarily be the common seat of disease; and when we reflect that this membrane is enclosed in an unyielding bony case, it is quite evident that many of its diseases must be accompanied by extreme pain.

In the spring of 1847, Jane Hartshorn, a female of stout, healthy appearance, was admitted into the Queen's Hospital under my care, suffering from pain in the shaft of the left tibia, which she said had originated from a fall on the knee of that side some months previously, by which the patella was much injured but not fractured. It was considered by the surgeon who attended her in the country, that an abscess had formed over the patella as a consequence of this injury, and an incision was made by him down to the bone, but without the discharge of any matter. From this period she dated the commencement of pain in the leg. At the time of her admission there was severe and constant pain down the whole shaft of the bone, which did not appear to be enlarged, but was tender when it was handled or pressed. A great number of remedies had been tried before she entered the hospital without any relief; and after that time the means resorted to failed in ameliorating the patient's condition; even the complete division of the periosteum, from the knee to the ankle, failed in giving more than a temporary cessation from pain. She remained in the hospital for nearly four months, and left it because at that time I refused to amputate the limb.

A residence in the country and attention to her

general health did not in any measure tend to mitigate the pain, which continued to deprive her of appetite and rest; her health began to suffer, she emaciated, perspired at night, and her constitution exhibited symptoms of giving way. A surgeon in the country proposed amputation, and she was actually on the table, preparatory to the amputation being performed, but preferring to lose her limb in the hospital, she came back, and I at length consented to remove the limb, which was amputated in the lower third of the thigh. An unusual quantity of dark coloured blood flowed in a stream from the medullary canal of the femur when sawn through; and the cavity when this had ceased, was completely filled with dark coloured coagula. The medullary canal of the tibia was carefully examined throughout; the periosteum was not so firmly adherent to the external surface of the bone as in a bone quite healthy. There was no medulla in the canal, which was quite full of dark, grumous blood, and the membrane universally dark throughout. Mr. Stanley* quotes a case from the *Journal Hebdomadaire*, where a very similar pathological condition of the medullary membrane was present. "It was of a deep red colour, resembling the conjunctiva of the eye in chemosis; and in places it was black, with a

* Diseases of the Bones, p. 49.

gangrenous odour." Portal says, "The contents of the medullary cavities may be reduced to a species of putrilage, as a consequence of inflammation; which has been proved by the examinations after death, of bones which have been the seat of severe and continued pains." Hartshorn's stump healed well; and the patient, who had before the operation some threatening of pain in the femur, similar to that which had existed previously in the tibia, continued free from pain till six or seven weeks after the stump had quite healed. Pain then came on in the thigh, and extended to the hip, at first slight, and in the lower part of the bone merely, afterwards becoming more severe, till, as in the first instance, appetite and sleep were lost, the patient began to fall away, and the general health to suffer so much, that it was evident to all some decided course must be adopted to save her life.

It was clear that all ordinary measures would be of no avail in such a case, though many were tried, without success, as might be supposed. The question of a second amputation was entertained: but here it appeared, that unless performed at the hip, we might have again the same condition returning as after the first operation. It occurred to me, after reflecting on Sir B. Brodie's operation for the dis-

charge of pus confined in the interior of bones, that if a piece were cut out of the femur, and a seton passed through it, that the patient might possibly be relieved, if not cured, without subjecting her to further mutilation, involving the risk of her life. A proceeding of this kind offered some chance of success, from the fact that she had no pain as long as there was discharge from the stump, but when the stump closed, the pain again came on.

Sir B. Brodie records a case which first induced me to think the operation I have mentioned would be of service to my patient, Hartshorn: "A young gentleman," says Sir Benjamin, "was brought to me from Brixton, with violent pain in one arm, the bone itself being enlarged in the part to which the pain was referred. The pain continued, and an abscess was suspected. Under this impression, an opening was made with the trephine so that the matter might be discharged, if there were any there. The trephine penetrated to the centre of the bone, but no matter escaped. I persevered," continues Sir Benjamin, "but still there was no matter; and at last the instrument penetrated completely from one side of the bone to the other." Sir Benjamin thought he had made a blunder, and there being no matter, the operation would not

be attended with any benefit; the wound, however, healed well, and the relief to pain was complete.

It was exceedingly probable, that in Hartshorn's case the state of the femur was like that of the tibia, from what was observed of the condition of the medullary canal at the time of the operation. There was some pain in the thigh before this, but the loss of blood at that time, and the discharge from the stump after, kept down for the time the inflamed or congested state of the medullary membrane.

Having determined to perforate the femur, and pass a seton through it to prevent the cavity being too soon closed by callus, I had a trephine prepared with a long, narrow crown, sufficiently long to pass through the bone. While the patient was under the influence of chloroform, I made an incision, about two inches and a half long, in the inner side of the limb, about four inches above the extremity of the stump, divided the periosteum, and perforated the femur with the trephine; I then passed a long, narrow seton needle, made for the purpose and armed with a thick web of cotton, through the hole in the femur, and brought it out on the outside of the thigh.

There was a great discharge of black blood from the medullary canal of the femur when it was opened by the trephine, which gave relief to pain; and after

the discharge produced by the introduction of the seton (which was profuse) was fully established, the pain in the bone, which had so long tormented her, was no longer felt. The seton was suffered to remain through the femur about three months, when it was withdrawn, and the wound suffered to heal. Since that time the patient has remained in good health, and there has been no return of pain in the bone. I have already alluded to the case recorded by Sir B. Brodie, in which he perforated the humerus for pain in the arm, no matter was discharged, but the operation succeeded in curing the pain.* It is exceedingly probable that this case also was an affection of the medullary membrane, probably of an inflammatory character, which had been cured by the division of the distended and overcharged vessels in the interior of the bone.

In the year 1848 a young female, named Chatwin, was admitted into the Queen's Hospital, under my care, for a painful affection of the tibia of the right leg. She was of delicate appearance, by occupation a house-maid, and entirely free, as was the last patient, from all venereal taint. Some time before her admission, she had felt shooting pains down the whole shaft of the tibia, which gradually

* *Pathological Researches*, p. 410.

increased in intensity till she could no longer bear the weight of the body upon the leg. The shaft of the bone was exceedingly tender to the touch, but not enlarged, nodulated, or uneven; the pains were rather in the shaft than in the extremities of the bone: in fact, the greatest pain was felt in the centre. Blisters, opiates, iron, the iodide of potass, and mercury, pushed to salivation, were absolutely useless, and did not procure even a temporary alleviation of pain. It was quite evident, that whatever the nature of the disease might be, that it was seated in the interior of the bone. After waiting a reasonable period of time to see the effects of the remedies employed, and finding my patient's health breaking, I determined to pass, as in the last case, a seton through the centre of the tibia, as this appeared the only means of saving the limb and life of the patient. The integument and periosteum were divided in the centre of the inner surface of the shaft of the bone and the bone perforated, so that the trephine came out on the external surface of the bone. The needle, armed as in the last instance, was passed through the bone and soft parts covering the external surface. The pain caused by the operation was considerable for a few days, but the pain for which the operation was undertaken soon diminished, and at the end of ten days had

entirely disappeared. If the seton were left unmoved for two or three days, there was a difficulty in moving it, owing to the firm deposition of callus around it; and on its final withdrawal, at the end of six weeks, the perforation was very soon closed by new bone.

It is most probable that operations for the relief of certain diseased conditions of the medullary membrane would be rendered in many instances abortive unless the opening in the bone were kept open, by seton or tent of some kind, and daily watched, as the closing of the opening by new bone is exceedingly rapid. Provisional callus is almost immediately thrown out after the perforation of a long bone by the trephine.*

It is quite evident, from the details of these two cases, that the shafts or extremities of the long bones may be opened with success for the purpose of relieving diseased conditions of their interior, besides those which are marked by the formation of matter. It may be asked, what are the symptoms of disease by which such a proceeding is indicated. I should say, fixed and continued pain in a bone, with or without

* See Bèclard *On the Formation of Callus.* Propositions sur quelques points de médecine; Thèse, Paris, 1813. Also Bonn: Thesaurus ossium morbosorum. The observations of these authors were published before the researches of Dupuytren on the same subject. Leçons orales de Clinique Chirurgicale; art. Formation du Cal.: tome iv, p. 70. Brussels edition.

enlargement of its shaft, of sufficient violence to threaten the destruction of the health and life of the patient, and the failure of other remedies adopted for its relief. In many cases, amputation has been resorted to as the only resource in such cases, but I am fully persuaded that the perforation of the bone, and passing a seton through it, will save many a limb which otherwise must have been lost.

Many, if not most, of these cases are doubtless congested or inflammatory condition of the medullary membrane of the interior of the long bones. In some instances such diseases may exist with affections of the periosteum; in others they may be present alone. It is probable that, in venereal diseases, many of the pains in the shafts of the tibia and other bones are due to disease of the medullary membrane, in the absence of all evidence of the periosteum being affected. Astruc and others, it is well known, attributed such pains to affections of the medulla itself; but it is now well established that adipose tissue, wherever met with, is insensible; and the experiments of Bichat and others have shown the seat of sensibility in the interior of the long bones to be in the medullary membrane, and not in the medulla itself. Scrofula, syphilis, rheumatism, and local injury are the most frequent causes of inflammation of the medullary

membrane, and are amongst the most frequent causes to which painful affections of the bones can be traced.

I have performed a similar operation, for a painful disease of the tibia, which was of venereal origin, in August of last year, on a girl named Eliza Cooper, but in this case the medullary cavity was opened only. I did not carry the trephine through the bone. The hole made by the trephine was kept open by a tent of lint, changed every morning. The relief to pain was complete; whilst previous to this operation it had resisted all the usual remedies in such states, as blisters, opiates, iodide of potass, &c., and had at times been so excessive that she had frequently importuned me to amputate the limb.

I have now performed the operation I have just alluded to, in six different cases for painful affections of the long bones, which have resisted all other modes of treatment, and in each instance with success. No constitutional disturbance of any importance has followed any of the operations. In Chatwin's case the pain consequent upon the perforation of the bone was severe for some days, but this was much more bearable than the pain for which the operation was performed. It subsided at the end of that period, and the patient has since enjoyed the best health. I have seen her two or three times since. She has

resumed her occupation as a domestic servant, and suffers nothing from her leg: she walks well on it, and is in good health.

I think, from what I have said, it may be concluded that the shafts of long bones may be perforated or opened for diseased condition of their interior, similar to those I have mentioned, with every probability of safety and success, where all other modes of treatment have failed.

Birmingham: Printed by Josiah Allen and Son, 3, Colmore Row.

From the Philosophical Magazine for June 1854.

ON

A NEW AND SIMPLE METHOD

OF

DETERMINING THE AMOUNT OF UREA

IN THE

URINARY SECRETION.

BY

EDMUND W. DAVY, A.B., M.B.T.C.D.,

LECTURER ON CHEMISTRY IN THE CARMICHAEL SCHOOL OF MEDICINE, ETC., DUBLIN.

UREA has long been regarded with much interest by scientific men, on account of its physiological and chemical relations. It represents one of the last stages of a series of metamorphoses or changes which nitrogenous matter undergoes in the animal œconomy, and is the form under which the detritus of pre-existing nitrogenous tissues which have become effete, principally pass from the system. This interesting organic base, urea, is not only formed during the exercise of the vital functions in man and some of the higher animals, but is also produced during the chemical decomposition of a number of substances containing nitrogen; and the chemist can now obtain it in any quantity by artificial means, and thus imitate one of the most important results of the chemistry of life.

In reference to medicine, urea is not without some practical interest, as it is well known that during various diseased conditions of the system the quantity of urea eliminated from the blood by the action of the kidneys and excreted in the urine is occasionally subject to great variation, and some ready means of ascertaining its quantity in that secretion might frequently aid the physician in forming his diagnosis of certain diseases.

Different means of effecting this object have from time to time been proposed; but all the methods hitherto recommended, with the exception, perhaps, of Baron Liebig's recent one, require for

their execution much time and trouble, and in some cases complicated and expensive apparatus; and though capable in experienced hands of yielding tolerably accurate results, would in the case of those not much practised in chemical manipulation, and perhaps unaware of the many sources of error to be guarded against, give anything but correct results, and are therefore inapplicable to the greater number of those desirous of a quick and easy method of determining the quantity of urea in urine.

The method I propose is one of extreme simplicity, and can be performed by almost any one in a very few minutes, and is capable of yielding results sufficiently accurate for all practical purposes. It is founded on the fact I have recently observed, that urea is very readily decomposed by the chlorides, or rather hypochlorites of soda, potash or lime; and its constituent nitrogen is evolved in the gaseous state, and from the quantity of gas evolved I estimate the amount of urea present.

After trying different means of carrying out that fact with a view of making it available to determine the quantity of urea in urine, I found that the following very simple one seemed to answer the purpose completely. I take a strong glass tube, about 12 or 14 inches long, closed at one end, and its open extremity ground smooth, and having the bore not larger than the thumb can conveniently cover. This I fill more than a third full of mercury, and afterwards pour in carefully a measured quantity of urine to be examined, which may be from a quarter of a drachm to a drachm or upwards, according to the capacity of the tube; then holding the tube in one hand near its open extremity, and having the thumb in readiness to cover the aperture, I quickly fill it completely full with a solution of the hypochlorite of soda (taking care not to overflow the tube), and then instantly cover the opening tightly with the thumb, and having rapidly inverted the tube once or twice to mix the urine with the hypochlorite, I finally open the tube under a saturated solution of common salt in water, contained in a steady cup or small mortar. The mercury then flows out and the solution of salt takes its place, and the mixture of urine and hypochlorite being lighter than the solution of salt, will remain in the upper part of the tube, and will therefore be prevented from descending and mixing with the fluid in the cup. A rapid disengagement of minute globules of gas soon takes place in the mixture in the upper part of the tube, and the gas is there retained and collected. The tube is then left in the upright position till there is no further appearance of minute globules of gas being formed, the time being dependent on the strength of the hypochlorite and the quantity of urea present; but the decomposition is generally completed in from three to four hours; it may, however, be left

much longer, even for a day if convenient, and having set the experiment going, it requires no further attention; and when the decomposition is completed, it is only necessary to measure the quantity of gas produced by transferring it into a graduated tube or measure.

I have generally used a graduated tube in the first instance, as it saves the trouble of transferring the gas and incurring the risk of losing some of it in the process. That which I would recommend as being convenient for this purpose is a stout tube having a bore of half an inch in diameter, and capable of holding from 2 to 3 cubic inches. A tube having this bore and about 14 inches in length, will hold $2\frac{1}{2}$ cubic inches, which will be quite large enough. Each cubic inch of it should be divided into tenths and hundredths of a part of a cubic inch.

It is scarcely necessary to remark, that in cases where great accuracy is required, due attention must be paid to the temperature and atmospheric pressure, and certain corrections made if these should deviate from the usual standards of comparison at the time of reading off the volume of the gas; but in most cases sufficiently near approximations to accuracy may be obtained without reference to those particulars. From a number of experiments, I have ascertained that the quantity of gas evolved from different amounts of urea, treated in the way I have just described, very closely approximates to the quantity of nitrogen gas which should be furnished from the urea by calculation. This will be seen from the following, taken from many experiments.

The fifth part of a grain of urea should furnish by calculation 0·3098 parts of a cubic inch of nitrogen gas at 60° F. and 30′ bar.; the same quantity of urea treated as described furnished in one experiment 0·3001, and in another 0·3069 parts of a cubic inch of gas at the same temperature and pressure; which shows that the calculated quantity of nitrogen differs from the amount of nitrogen gas obtained by only a few thousandths of a part of a cubic inch. I may observe that I was obliged to operate on such small quantities of urea, on account of the graduated tube I had at the time being only of one cubic inch capacity.

Seeing, then, that the quantity of gas evolved agrees so very closely with the calculated amount of nitrogen present in a certain quantity of urea, I take the calculated amount as being the more correct; and knowing the relation that exists between a certain quantity of urea and nitrogen, I can from the quantity of gas evolved in any case easily calculate the amount of urea present by the simple process of rule of three. Thus the fifth part of a grain, or 0·2 of a grain of urea, gives by calculation 0·3098 parts of a cubic inch of nitrogen gas. Then 0·3098 : the

volume of gas found : : 0·2 : to the required quantity of urea; or multiplying the first and third terms by 5, we have 1·549 cubic inch of gas representing one grain of urea, which is a simpler proportion. Using these data, I made several comparative experiments on different samples of urine with my method and that of Baron Liebig's, which I believe is considered one of the most accurate of the methods of determining urea in urine at present known. The following are the results of three comparative experiments on different samples of urine, using the same with each method.

Amount of urea in grains and parts of a grain in one fluid ounce of urine.

	Liebig's.	New method.
1st experiment . .	3·680	3·712
2nd experiment . .	5·328	5·472
3rd experiment . .	4·976	4·976

In the first and second experiments the quantity of common salt present in the urine was taken into account, as it is found to increase to a slight degree the apparent quantity of urea in the urine by Liebig's method. In the third this was not taken into consideration, and the quantity of urea was compared with the mean of two results obtained by my method. These experiments show how very closely the results obtained by the new method agree with those by Baron Liebig's, and therefore show the correctness of the former method.

I prefer the hypochlorite of soda to that of potash as a decomposing agent, because the soda salt is an article of our pharmacopœia under the name "Sodæ chlorinatæ liquor," and therefore can be so easily procured; whereas the potash salt, not being used in medicine, would require to be specially made for the purpose. As to the hypochlorite of lime, I do not think it so effectual, and it has the disadvantage of soiling the sides of the graduated tube by the carbonate of lime formed in the reaction.

In reference to the quantity of hypochlorite of soda to be employed, it should always be used in excess; and I think that about five or six times the volume of the urine employed would be found generally to be quite sufficient, and ensure there being an excess of the hypochlorite. The amount required may be easily determined also by direct experiment, by adding to a certain quantity of urine to be examined in a glass a measured quantity of the hypochlorite, and leaving it for a short time till the evolution of gas is nearly over; then if, on the addition of more of the hypochlorite, the effervescence is renewed, it shows that there was not enough of the decomposing liquor first employed, and more must be added from time to time till no further

evolution of gas is produced; and the quantity of hypochlorite used to arrive at this point indicates the amount necessary. I found by experiment that one grain of urea requires somewhere about half a fluid ounce of the ordinary sodæ chlorinatæ liquor for its complete decomposition. The amount of mercury employed requires some little attention. It should, as a general rule, be never less than the volume of gas produced; for if the volume of gas evolved is more than that of the mercury used, it will be more than that of the solution of salt, and therefore some of the mixture of urine and hypochlorite will be forced out of the tube before it is completely decomposed, and consequently some of the gas will be lost; so that if this occurs, we must repeat the experiment, using either a larger quantity of mercury if our tube will allow, or diminishing the quantity of urine employed.

It might be supposed on first sight that this method would be liable to the following source of error, viz. that some of the gas would be evolved and lost during the pouring in of the hypochlorite; but this is not the case, as several seconds elapse before there is any apparent reaction or evolution of gas on mixing the hypochlorite with the urine, and there is therefore full time to perform the experiment without any loss of the gas. I have also ascertained that the acid reaction of the urine does not affect my method. I should observe that this new method, like all the others known, is not perfectly free from some slight sources of error; the principal one being, that ammonia, if it exists in the urine, gives rise to nitrogen gas, and therefore increases the apparent amount of urea; but the same objection holds equally in Liebig's and Ragsky's methods, which are perhaps the two most accurate at present known. Uric acid also is similarly affected by the hypochlorite; but it and ammonia ordinarily occur in such small proportion in urine, that the error produced from these substances would be but trifling, and is partly corrected by taking the calculated quantity of nitrogen, which is, as I have shown, something more than that obtained from a certain quantity of urea by direct experiment.

In cases where ammonia or uric acid occurs in more than ordinary quantity, these substances must be separated by the usual means employed before having recourse to my method.

I should think that gently heating the urine with a certain quantity of baryta water as long as the odour of ammonia is disengaged, and then filtering the solution, as recommended by Liebig, for the separation of ammonia before applying his method (see the Quarterly Journal of the Chemical Society, vol. vi. p. 30), would effect the object very easily, and separate not only the ammonia, but also the greater part, if not all of the uric acid present.

There is one other source of error which may arise, and which can be easily avoided. It is the following: that if a solution of the hypochlorite of soda alone, or standing over mercury, be exposed to the light for several days, it will very gradually evolve a minute quantity of oxygen, which shows that in determining urea we should not allow the experiment to go on for too long a time; but if left for a day, or even two, it will scarcely make any appreciable effect on the quantity of gas evolved in testing for urea.

The reaction which appears to take place in the process seems to be the following. The hypochlorite of soda acting on the urea gives rise to the formation of carbonic acid, water, and chloride of sodium, together with the evolution of nitrogen gas. Thus

Urea. 3 Hypochlorite soda.

$$C^2.H^4.N^2.O^2.+3(Cl,O.NaO)=2CO^2+4HO+3Cl.Na.+N^2.$$

The nitrogen is evolved and the carbonic acid is absorbed by some of the hypochlorite of soda in excess, for I find that this salt absorbs carbonic acid very quickly without evolving any other gas; and I failed in several experiments to detect the smallest portion of carbonic acid in the gas produced by acting on urea, though I have always noticed the presence of a very minute quantity of oxygen in the nitrogen gas. These appear from my experiments to be the changes produced; but this part of the subject I have not as yet minutely examined, and my experiments have hitherto been made on healthy urine; I have, however, ascertained that several of the substances found in urine during disease, as for example, sugar, albumen, bile, and excess of urinary colouring matter, produce scarcely any effect on the results obtained by this new method of determining the quantity of urea in the urinary secretion.

Edmund A. Parkes, Esq. M.D. F.R.S.
With the Author's Compliments

ON THE

FŒTUS IN UTERO

AS

INOCULATING THE MATERNAL

WITH THE

PECULIARITIES OF THE PATERNAL ORGANISM:

AND ON THE TRANSMISSION THEREBY OF

SECONDARY OR CONSTITUTIONAL SYPHILIS

FROM THE MALE TO THE FEMALE PARENT.

BY

ALEXANDER HARVEY, M.D.,

Late Physician to the Aberdeen Royal Infirmary; and formerly Lecturer, sometime on the Institutes, and afterwards on the Practice of Medicine in the University of Aberdeen.

(*Read before the Medical Society of Southampton, December* 2, 1856; *and reprinted from the* GLASGOW MEDICAL JOURNAL, *January*, 1859.)

GLASGOW:
WILLIAM MACKENZIE, 45 & 47 HOWARD STREET.
MDCCCLIX.

ON THE FŒTUS IN UTERO

AS

INOCULATING THE MATERNAL WITH THE PECULIARITIES OF THE PATERNAL ORGANISM, Etc.

BY ALEXANDER HARVEY, M.D.

(*Read before the Medical Society of Southampton, December* 2, 1856; *and reprinted from the* Glasgow Medical Journal, *January*, 1859).

It is now several years since first, in a series of papers in the *Edinburgh Monthly Journal of Medical Science*,* and afterwards in a separate pamphlet, dedicated to the Highland and Agricultural Society of Scotland,† I directed attention to a class of facts which seem to indicate that the *fœtus in utero* may, and in fact habitually does, inoculate the female with the constitutional qualities of the male parent. I did not myself suggest the theory of inoculation. It had been advanced a short time before, in the columns of a provincial newspaper,‡ by Mr. James M'Gillivray, a veterinary surgeon in Aberdeenshire. But I may claim the merit of introducing it to the notice of the profession, and giving currency to it among the agricultural body; as well as of imparting to it a more scientific form, and placing it on a broader basis, than had been done by Mr. M'Gillivray.

In both the first and the second of the series of papers referred to, I pointed out the application of this principle to the question—Whether *secondary syphilis* may not thus be transmitted from the male to the female; and it is to this question that I now wish specially to call your attention, premising that my own has

* October, 1849, and October and November, 1850.
† On a Remarkable Effect of Cross-Breeding. Edinburgh, 1851.
‡ The Aberdeen Journal for March 21 and 28, 1849.

recently been recalled to it by two communications on the same subject, published almost simultaneously, but quite independently of each other, both supporting the same views, and both pointing to that principle of inoculation. One of these is in the *Edinburgh Medical Journal* for October last (1856), by Dr. James B. Balfour of Edinburgh; the other is a "Report" of illustrative cases, in the *Medical Times and Gazette* for October 11th, prepared by Mr. Hutchinson, of the Metropolitan Free Hospital, and to which is appended a summary of the principal conclusions arrived at; the report itself forming the groundwork of a paper which was read by him a little while ago to the Hunterian Society of London, and will shortly appear as an original communication in the pages of that periodical.* And my object is to bring together, as far as known to me, the facts that have been ascertained regarding it by these and other writers; and, by presenting those facts under one connected view, to aid in the further investigation of a subject which is as interesting in its physiological, as it is important in its pathological and practical relations.

But (*reculer pour mieux sauter*) it will be expedient, before entering upon it, to lay before you a brief outline of the general doctrine of maternal inoculation through the fœtus, as derived from other facts, and resting on other grounds. Such an outline will, I hope, enable you the better to see clearly the bearings of our proper subject, and to appreciate its importance, not merely as a practical question, but as supplying a test or criterion of great delicacy, and of corresponding value, in regard to the whole general doctrine itself.

I. The general fact on which this theory of inoculation is founded is, that the peculiarities of a male animal that has once had fruitful intercourse with a female, of the class *mammalia*, may be more or less clearly discernible in the progeny which that female may subsequently have by other males; or, in other words, that a male animal that has once had such intercourse with a mammalian female may so influence her future offspring begotten by other males, as, to a greater or less extent, to engraft upon them his own distinctive features and his own constitutional qualities; his influence thus reaching to the subsequent progeny, in whose conception he himself has had no share, and his image and superscription, so to speak, being more or less legibly inscribed upon them. Accordingly, if the female be of a *different* breed or species from that male, and have thus borne a cross or hybrid by him, her future offspring, got by males of the *same* breed and species with herself, may yet have more or less the characters of a cross or hybrid. And if it be true, as is maintained by Mr. Orton of Sunderland, that, in the reproduction of the animal species, the male parent imparts mainly to his offspring the parts

* Since published therein—December 20, 1856, and January 10, 1857.

and qualities that are characteristic both of the animal in general, and of the species to which he belongs, as well as of himself individually—to wit, the external structures or the organs of animal life—the brain, nerves, organs of sense, the skin and hair, together with the bones and muscles—and for the most part, therefore, determines the outward character and general appearance of the offspring—it is precisely certain of those parts and characters which may be expected to appear, and which are, in fact, discernible in the subsequent offspring which the female bears to other males.*

1. This general fact was not unknown to the great Haller. It was known even to Becker, who lived long before Haller; both these writers stating, that when a mare has had a mule by an ass, and afterwards a foal by a horse, the foal exhibits traces of the ass.†

It was not, however, till the publication in the *Philosophical Transactions* for the year 1821,‡ of the cases of Lord Morton's mare, and of Mr. Giles' sow, that the subject attracted any particular attention. A young chestnut mare, seven-eighths Arabian, was covered in 1815 by a quagga—a species of wild ass from Africa, and marked somewhat after the manner of the zebra. The mare was covered but once by the quagga, and, after a pregnancy of eleven months and four days, gave birth to a hybrid, which took strongly after the quagga. Subsequently, in 1817, 1818, and 1821, this mare was served by a very fine black Arabian horse, and produced successively three foals, all of which bore unequivocal marks of the quagga; the first, however, in a higher degree than the third. Again, a sow, of the black and white breed (known as Mr. Western's breed), became pregnant by a boar of the wild breed, of a deep chestnut colour. The pigs produced were duly mixed, the colour of the boar being in some of them very prominent. The sow being afterwards put to a boar of the same breed as her own, some of the produce were observed to be stained or marked with the chestnut colour that prevailed in the former litter. And on a subsequent occasion, the boar being still of the same breed as the sow, some of the litter were also slightly marked with the chestnut colour. What adds to the value of the fact now stated is, that, in the course of many years' observation, the breed in question was *never* known to produce progeny having the slightest tinge of the chestnut colour.

* See two papers by Mr. Orton, on "The Physiology of Breeding," in the *Newcastle Chronicle* for March 10, 1854, and November 10, 1854; and a paper by the author, "On the Relative Influence of the Male and Female Parents in the Reproduction of the Animal Species," in the *Edinburgh Monthly Journal* for August, 1854.

† Haller, Element. Physiol., vol. viii., p. 104; Becker, Physic. Subterran. Lips., 1703.

‡ Pages 20 and 23.

These two cases were long looked upon as solitary examples of an exceptional phenomenon, for the reference made by Haller and Becker to a precisely analogous case had come to be forgotten; and the phenomenon exemplified by them was long regarded as scarcely, if at all, admitting of a plausible explanation. By some, however, it was suggested that it might be ascribed to a partial fœcundation by the quagga and chestnut boar, respectively, of others of the mare's and sow's ova than those actually impregnated by them; or at least to some influence of their seminal fluid on the ova that remained, of a permanent kind, but strictly *local* in its agency, and modifying in some way the development of these ova, when afterwards impregnated by other males. By others, again, the phenomenon was regarded as an instance of that class which come within the category of "mothers' marks," and are popularly ascribed to the agency of the imagination or of the mind on the body, and supposed to be illustrated by a reference to the case recorded in Genesis (chap. xxx.), of Jacob and his peeled rods. Neither the phenomenon itself, however, nor these conjectures regarding it, gave rise to much discussion, or long continued to engage the attention of physiologists. The subject was altogether passed over by Dr. Bostock, in his elaborate System of Physiology. It was referred to by Mr. Mayo and by Dr. Alison, in their "Outlines," and by Dr. Kirkes, in his "Handbook," of Physiology. But a brief and passing allusion to it was all the notice it received.

Meanwhile, instances of a like kind were becoming known to persons engaged in the breeding of horses, cattle, sheep, dogs, and swine; and in 1849 Mr. M'Gillivray adduced, in the paper I have referred to, a collection of cases sufficiently large to show, that the phenomenon exemplified in Lord Morton's mare, or rather in her progeny, is by no means so unique as it was thought to be. And it may now be confidently affirmed, that since then, and within these few years, enough has transpired in this department of inquiry to warrant the presumption, that the phenomenon in question is so uniform in its occurrence, whenever the requisite conditions obtain, as to constitute a general fact or law of nature. It is so regarded by a large number of our great breeders of cattle, by dog-fanciers, and others of large experience in the rearing of horses, sheep, &c., and is habitually taken into account by them in the selection of their breeding-stock, and in their estimate of the purity of an animal's blood; the *practical* belief of all of them on this subject being, that a female animal that has once been impregnated by a male of a different breed or species from her own, and has borne a cross or a hybrid by him, is thereby *destroyed* for a time at least, if not permanently, for the purposes of breeding *pure* stock of her own kind; and the *theoretical* belief of many among them, that this result happens because (as was first suggested by M. M'Gillivray) the blood of the female has,

through the fœtus, been *contaminated* by the blood, and charged with the qualities of the male she first had intercourse with—so charged therewith, so contaminated thereby, that she herself imparts the blood and the qualities of this male to the progeny she afterwards has by other males.

2. It is not my intention to lay before you any additional examples of this kind as occurring in the lower animals. I have elsewhere brought together a tolerably large collection, and to this it must suffice to refer. But I cannot forbear just briefly alluding to the fact, for the mention of which you will be quite prepared, and the reference to which will naturally pave the way to our proper subject—that instances of the like sort are, perhaps, equally common in our own species. It has long been known—and it is, in fact, a popular observation—that, in the case of a woman twice married, and fruitful by both husbands, the children of the second marriage often resemble their mother's first husband or his family, and that not in features only, but in mind also, and in disposition. It is obvious, indeed, that in any such case, where all the individuals concerned—the woman, her children, and both the husbands—are of the *same* variety of the human family, the alleged resemblance must often be exceedingly difficult to trace or to substantiate. But it is equally obvious, that means exist for ascertaining clearly whether it obtains or not. There are equally distinct varieties of the human species as there are of any of the lower animals; and all that is requisite for bringing the question to a decisive issue is, to observe accurately whether the children of European parents—when the mother has, in the first instance, had offspring by a negro—exhibit traces of the latter in the colour of the skin, and still more in the quality of the hair, the form of the features, &c.; or, contrariwise, whether the children of negro parents, when the mother had formerly been impregnated by a European, manifest the peculiarities of the latter.

Of the former case, I have heard of two instances as occurring in this country, one of them under the notice of Professor Simpson of Edinburgh:—A young woman, born of white parents, but whose mother had sometime before her marriage had a bastard child (a mulatto) by a negro man-servant in Edinburgh, presented distinct traces of the negro. Of the latter case, that in which the parents are both negroes, but in which the mother has previously had fruitful connection with a European, Dr. Robert Balfour, of Surinam, wrote me some years ago to say, that repeated instances of the European influence under such circumstances, had come under his own observation.* Doubtless, if looked for, examples

* "Among the coloured population" (Dr. Balfour writes me, April 19, 1851), "consisting of negroes and their offspring by Europeans, the most striking illustrations of the truth of your or Mr. M'Gillivray's theory are constantly occurring. Shortly after my settlement here, I was led to remark the circumstance (which I had never noticed recorded by writers), that if a negress had a

of the influence now in question would be found as general in our own species as in the lower animals. And it may just be observed that, in the one as in the other, they can only be unequivocally and satisfactorily determined in cases where the individuals or the animals that are the subjects of observation are of a different race or species; or rather, where the male, the female, and the offspring, are of the same breed and species, but the female has first of all been impregnated by a male of a different breed or species.

3. In ascribing this very remarkable phenomenon, as seen both in our own species and in the lower animals of the class Mammalia, to an agency exerted by the fœtus on the mother's system, of the nature of inoculation, I do not intend to consider in detail, or in connection with other explanations that have been given of it, the grounds on which that doctrine of inoculation rests. The questions involved in it are too numerous, and the discussion of them would be too tedious, to admit of my entering upon them at present. I have elsewhere considered them pretty fully, and must here content myself with this general remark, that the facts there advanced in support of the theory of inoculation have always seemed to me sufficient, not indeed absolutely to prove the truth of it, but to impart to it a probability short only of absolute certainty; and, further, that while I deemed it expedient to suggest to the agricultural body certain experiments in breeding, which, if carefully made, could not fail to be decisive of it, I have always thought that a sufficiently large collection of facts, of the description of those now to be laid before you in regard to the poison of syphilis, would supply all that is really wanting to make the *demonstration* of its truth complete. The *reasonableness* of the doctrine no one can well dispute. Connected as the fœtus and its mother are by the placenta, a mutual interchange of fluid matters is continually going on between them; the fœtus drawing supplies from its mother's blood for its growth and maintenance, and the mother (though, no doubt, in much less quantity) abstracting materials from the blood of the fœtus. Is it unreasonable to suppose that the matters abstracted by the mother may be charged with (or have inherent in them) the *constitutional* qualities of the fœtus; or that passing into her body, and mingling there with the general mass of her blood, they may impart those qualities to her system? And this supposition will perhaps appear the less improbable, if regard be had to the length of time during which the connection between the mother and the fœtus is kept up, and during which this transference of materials must go on—a period of some weeks, or even of several months. But the qualities inherent in the fœtus must in part be derived by it from its male

child or children by a white, and afterward- fruitful intercourse with a negro, the latter offspring had generally a lighter colour than the parents. . . . I could particularize many such, the observation of which had led me to form an opinion similar to that which you have more elaborately worked out."

parent, and be to that extent identical with his. The distinctive peculiarities, therefore, of this parent may thus come to be *engrafted* on the mother, or to attach in some way to her system; and if so, what more likely than that they should be communicated by her to any offspring she may afterwards have by other males?

4. Hitherto we have had under consideration the transmission of such qualities only as are strictly normal or healthy, and so far the subject may be regarded as one of physiological interest merely, or at least of practical importance to breeders alone. But it is conceivable that *morbid* qualities may be transmitted also, and be in the same way, or through the same channel, engrafted on the mother's system. In our own species, scrofula may, peradventure, be thus transmitted. In my first paper on this subject, I not only refer to the possibility of this, but give two instances in which it apparently happened.* A serious difficulty, indeed, attaches to the investigation of this point. So *widely* diffused is the scrofulous diathesis among mankind, that, in any single case in which it has apparently been acquired in this way, it would be difficult to determine whether it was *really* thus derived or not. Still, observations on the large scale, long and carefully conducted, as to the health and constitutional tendencies of females before and after marriage, as compared with those of their husbands and children, and carefully distinguishing between females that have, and females that have not had children, might in the end, and after full allowance for errors or fallacies of observation, sufficiently demonstrate the reality of the occurrence. Certainly it is not an uncommon observation, that women born of healthy parents, and themselves previously healthy, fall into a state of general ill-health after becoming mothers†—an observation which may yet be connected, in a certain proportion of cases, with the principle now in question. So, likewise, may the syphilitic poison, in its *constitutional* forms, be transmitted after the same manner from the husband to the wife, and be by her conveyed to the children she may subsequently bear to a second husband, himself perfectly free of all syphilitic taint. Whether any *other* morbid states

* Edinburgh Monthly Journal, Oct., 1849.—The one, reported to me by Dr George Ogilvie of Aberdeen, of a woman who was twice married, and had children by both husbands, and whose children by both marriages were scrofulous—although only the first husband had marks of that diathesis—the woman herself and her second husband being, to all appearance, quite healthy;—the other, by Professor Pirie, and as follows:—Mrs. H——, apparently perfectly free from scrofula, married a man who died of phthisis. She had one child by him, which also died of phthisis. She next married a man who was seemingly equally healthy as herself, and had two children by him, one of which died of phthisis, the other of tubercular mesenteric disease, having at the same time scrofulous ulceration of one of the lower extremities.

† Even this observation has not escaped Mr. Kingsley. Speaking of Mrs. Vavasour, he says—"And when children, and the *weak health* (on the part of the mother) which comes with them," &c.—*Two Years Ago*, vol. i., p. 70.

besides these—*e. g.*, mania, epilepsy, gout, &c., which are unquestionably hereditary—may thus be imparted and transmitted, it is at present impossible to say. Enough appears, however, from a consideration of the transmission of healthy qualities, and from what is known in regard to the transmission of syphilis, to impart to the whole inquiry, as to the transmission of morbid qualities, an interest of the highest practical kind, and that in a *social* as well as in a purely *professional* point of view. So important may it yet be ascertained to be, in contemplating marriage with a widow, to have regard to the constitutional peculiarities of the deceased husband!

So much for the general outline I spoke of at the outset—fuller, indeed, than was intended, but, perhaps, not more so than it was necessary to premise in order to the right understanding of our proper subject—the fœtus in utero, as the channel of transmission of secondary or constitutional syphilis from the male to the female parent.

II. Taking up, now, the subject of *Syphilis*, in connection with the alleged inoculating power of the fœtus, let me just observe at the outset, that there are two points of view from which it may be regarded—two objects with which its investigation may be conducted:—*First*, As a part of the history of syphilis, and in relation to pathological and practical ends; and, *secondly*, as a test or criterion of the general doctrine of inoculation itself, its special value as a test lying in this, that nothing like *mental* influence can possibly be imagined to have any share in the transmission of the virus. And these two objects, although so far distinct, need not—nay, cannot be separated, in the prosecution of our scientific inquiries. That is to say, if such inquiry shall demonstrate that the syphilitic poison in its constitutional form may be, and actually is, transmitted—nay, is only communicated, and is communicable only, from the husband to the wife through the fœtus—while this fact would form a real and practically important addition to our knowledge of the laws of that poison, it would, at the same time, establish the general doctrine of inoculation, as exemplified in other modes, and resting on other grounds; and it would impart, besides, a peculiar strength to the evidence in support of it thus derived, because absolutely devoid of all leaven of the fallacy which may be supposed to attach to that and every other source of evidence.

1. The *first* question I shall consider, as more obviously connecting itself with the general features of the cases already referred to, and exemplified in Lord Morton's mare, is—Whether a woman, twice married—first to a constitutionally syphilitic husband, next to a husband untainted by that poison—and fruitful by both, may herself (not otherwise tainted than through her former husband) infect the children of her second marriage?

In seeking formerly (in 1849) for a solution of this question,

which I thought I had been the first to suggest, I was unable to do more than adduce in illustration of it an imperfect observation from Messrs. Maunsell and Evanson, to the effect, "that they have notes of the case of a syphilitic child, whose mother had been infected by a former husband, and to all appearance cured five years before its birth; the father of the child, her second husband, being in perfect health."* They do not say, however, in what way she was infected; and as it may have been in that of primary affection or chancre, the case, as it stands, is if no value in relation to the question before us.

Dr. Montgomery, of Dublin, I afterwards found had been beforehand with me in this question as to syphilis. Referring to Lord Morton's mare, and to Mr. Giles' sow, Dr. Montgomery remarked, in 1837, in the first edition of his great work "On the Signs and Symptoms of Pregnancy:"—

> "Such occurrences appear forcibly to suggest a question, the correct solution of which would be of immense importance in the history and treatment of disease. Is it possible (he asks) that a morbid taint, such as that of syphilis, for instance, having been once communicated to the system of the female" by a conception, "may long linger there, and influencing several ova, continue to manifest itself in the offspring of subsequent conceptions, where impregnation has been effected by a perfectly healthy man, and the system of the mother appearing to be at the time, and for a considerable period previously, quite free from the disease? My belief" (he adds) "is certainly in favour of the affirmative."

What Dr. Montgomery here suggested as a probable occurrence, and then merely suspected, he has since been able to substantiate as a fact:—

> "Such" (he observes in the second edition of that work, recently published) "was the opinion I expressed in 1837; and further experience and observation have, I think, shown to be a fact, what I could then only venture to say I believed to be likely."

He then proceeds to quote two cases in support of this observation—one from M. Vidal, the other from M. Cazenave—remarking afterwards that, while he thought the observation of the fact was original with himself, he had found it was made long ago by others—by M. Gardien, in his Traité d'Accouch.,† and by Dr. F. H. Ramsbotham, in the *Medical Gazette* for May 23, 1835.‡

* On the Management and Diseases of Children, 5th edit., pp. 452, 453.

† Edition 1824, vol. ii., p. 29.

‡ We shall see by and by how clearly Dr. Ramsbotham had divined it. It seems to me, however, very doubtful whether M. Gardien had any conception of it whatever, judging at least from the passage referred to by Dr. Montgomery, and which is as follows:—"Le moment de la conception devient quelquefois une circonstance qui favorize chez la femme le developpement de differens virus qui étaient restés sans action jusqu'à ce moment. L'état de grossesse manifeste quelquefois des symptomes de virus vénérien qu'on n'avait pas encore aperçu, et qui, sans cela, aurait pu rester encore plus ou moins de temps engourdi. Le virus syphilitique, avant la conception, pouvait circuler dans la masse generale

In the case given by Cazenave,* the syphilitic taint was transmitted by the mother to the children of her *third* husband, as well as to those of her second; but the case is essentially defective, inasmuch as it is not stated in what manner the woman *originally* became affected. It would even appear from the narrative of the case, that it was while a widow that she became affected; and as it does not appear that she had a child subsequently till she married her second husband, it is probable that she acquired the disease in the primary form, at least otherwise than through impregnation. M. Vidal's case, however, is in every respect unexceptionable, and exactly to the point:—

"A woman, whose husband was affected with constitutional syphilis, gave birth to a child, which in two months showed symptoms of that disease, of which it died. The woman never had any appearance of syphilitic affection, not even sufficient to soil her linen. Her husband died, and, after remaining some time a widow, she married a healthy man; and about twenty months afterwards, being four years after the former birth, she bore a child, which in two months presented the same form of syphilitic eruption which had appeared on the former child."

It is plain that opportunities for observing cases of this description must be comparatively very rare. A second marriage, indeed, on the part of widows, is not uncommon; but with the conditions requisite for thus tracing the transmission of the syphilitic poison, very uncommon. And it must be kept in mind, that in some cases of *this* kind, the results of observation (although, as we shall see, of great value notwithstanding) will be of a *negative* character, as in the following case given by Dr. James B. Balfour:—

"A respectable young woman from the north was married to a tradesman, who had no trace of syphilitic disease at the period of marriage; but he afterwards acknowledged that, two years before, he had the disease, followed by slight secondary symptoms, which had entirely disappeared under medical treatment, and he had seen or felt nothing since. This woman complained of nothing until about three months after she became pregnant. Then, however, symptoms of secondary syphilis became apparent—spots of psoriasis appeared on various parts of the body—hard knots were felt on the perineum, and on the external labiæ—and within the vagina there was a hard, knotty feeling over the whole mucous surface. Her child exhibited a distinct syphilitic appearance, which was removed by the usual treatment. Shortly after delivery all symptoms of syphilis entirely disappeared; and as she shortly afterwards removed to the country, she was subjected to no medical treatment. A few months afterwards her husband died. She subsequently married a farmer. About six or eight months since I saw her, and she has borne three children to her second husband, and certainly more healthy children I could not wish to see. She informed me that she had never been under medical treatment since I had attended her—indeed, she never had a medical man near her, except during her confinements—and that she had never suffered, during any of her pregnancies, from anything like what she had done at the first one."

sans affecter aucune glande, et sans donner de marques de sa présence; parceque ces organes n'étaient pas sensibles à son action. La grossesse augmentant la sensibilité, manifeste des symptomes d'une infection générale, parceque les organes glandulaux ressentent, à cette époque, les impressions du virus vénérien, auquel ils étaient insensibles auparavant."

* Traité des Syphilides, etc., p. 133.

I shall not trouble you again with the details of individual cases. But I have thought it desirable to quote at length the two I have taken from Dr. Balfour and M. Vidal, as being fair examples of the kind of cases we may expect to meet with, and on which our conclusions must be founded; few of them exactly tallying in every point; most of them having each its own special features, and the comparative value of the several cases being widely different—none, however, being of any value in which it does not clearly appear that the virus was imparted to the female in its secondary or constitutional form only. In both the cases adduced this essential condition was plainly apparent, but they differed in other respects. In M. Vidal's, the woman never exhibited in her own person any symptoms of syphilis, but gave proof of being tainted with it by giving birth to a syphilitic child, got by her second husband, a healthy man. In Dr. Balfour's this evidence was wanting; but the fact of the woman having imbibed the poison from her first husband, appears from the symptoms of it having shown themselves upon herself during her first pregnancy; the evidence thus afforded deriving confirmation from this other fact, that the child she then bore was also affected with it.

2. And this, for the most part, is the *sort* of proof we should look for—direct evidence furnished by positive manifestations in the person of the mother, and confirmed by like manifestations in that of the child, of syphilitic taint. Although always interesting, and indeed important to do so, it will not actually be necessary, except in a very few cases (as in M. Vidal's), to seek to trace the transmission of the virus to the children of a second marriage, and of a healthy father. The proof we require lies within a narrower compass, and may be reached by a shorter process. Dr. Balfour's case, rather than M. Vidal's, is the type of the cases we have to search for and to consider; and, fortunately for the investigation, they are of much more frequent occurrence. And the question to be considered, in the first instance, is simply this—Whether ever a woman derives secondary syphilis from her husband *when* she conceives by him, and *because* of her doing so.

The evidence now available on this point, as far as known to me, is of two kinds:—*First*, General statements of experience supplied by M. Ricord, Dr. Montgomery, Dr. Tyler Smith, Dr. Carpenter, and likewise by the author of the review, in the *Edinburgh Medical Journal*, of the new edition of Dr. Montgomery's work, and by Dr. Ramsbotham; and, *secondly*, Specific instances observed and recorded by various writers.

Let us first consider the former branch of the evidence. M.

Ricord, one of the highest authorities on all points relating to syphilis, observes that his experience goes to prove, that in the case of a woman pregnant of a child whose blood is contaminated with syphilis acquired from the father, this child may, and often actually does, contaminate the mother's system :—

"So long as a diseased father (says M. Ricord) is under the influence of constitutional syphilis, the germ which is by him conveyed into the uterus carries along with it the syphilitic diathesis. . . . There is no such thing as an infection of the child by the mother, she having been contaminated by the father; but the husband procreates an infected child, which may then propagate the secondary poison to the mother—*for where there are no children, the mother does not suffer.*"*

Dr. Montgomery, in the last edition of his "Signs and Symptoms of Pregnancy," speaking of this very point, remarks, that "there can be no doubt of the *frequent* occurrence of the fact." Dr. Tyler Smith's testimony is, that the most common mode in which women become affected with syphilitic *uterine* disorder, is, he believes, that in which the fœtus is the medium of communication.† Dr. Carpenter, speaking of the inoculation theory, says—

"This idea is borne out by a great number of important facts; and it serves to explain the circumstance, *well known* to practitioners, that secondary syphilis will *often* appear in a female during gestation or after parturition, who has never had primary symptoms, whilst the father of the child shows no recent syphilitic disorder."‡

And the reviewer referred to observes—

"That a woman may get syphilis from a man without any external disease—namely, by bearing a syphilitic child in the womb of her previously healthy body, which child infects her with the disease; and that she may then exhibit outward signs of the disease now in her constitution—or, without this, may prove her syphilitic taint, by producing syphilitic children to another perfectly healthy father."

Let us see, further, on this branch of the evidence, what Dr. Ramsbotham says. Among the first, if not *the* first, to suggest the possibility—nay, the great *probability*—of this mode of transmission of the syphilitic poison, nothing can be clearer or more explicit than his observations on it :—

"It is a commonly received opinion (says Dr. R.) that syphilis, in its secondary stage, is not communicable directly to either sex from the other—that the disease is not propagated unless there exist an open chancre, and this accords with my observation. But it appears to me *probable*, that if a previously healthy woman conceive of an ovum tainted by syphilitic virus derived from its father, her system may become *inoculated* during the progress of gestation, in consequence of the close vascular connection existing between it and herself; for it has fallen to my lot *to see more than one case*, in which a young woman, united to a man labouring under obstinate secondary symptoms, remained *healthy* for *some months after marriage*, but became the subject of the same disease in its secondary form, *soon after impregnation had taken place;* and *I have considered* that, in such a case, the mother derived the disease, *not directly from the father*, but *from the affected infant* which she carried in her womb."—(*Loc. Cit.*)

* The Lancet, April 8, 1848, p. 383. See also Mr. Acton's work on Syphilis, p. 632.

† See Association Medical Journal, July 14, 1854.

‡ Principles of Human Physiology, 5th edition, p. 826.

These are strong and unequivocal statements, and the specific evidence furnished by individual cases is equally decided. To what extent the records of medicine can now supply examples of this description, I cannot say. But I have already repeatedly alluded to those brought forward by Dr. Balfour and by Mr. Hutchinson; and it is to these that I would now particularly refer. The former gives four or five cases, the latter as many as fifty. To be duly appreciated, these cases must be examined individually and compared together—a task which it were foreign to my object to enter upon at present, even were it possible to accomplish it, without actually reproducing the cases in detail. It must suffice, therefore, thus to refer to them, and to remark that, having carefully considered them, I think they conclusively establish in the affirmative the question presently before us; proving that a woman may, and often actually does derive syphilis from her husband, when she conceives by him, and in consequence of her doing so.

3. The next question to be considered is—Whether at each pregnancy there may be a *renewal* of the symptoms of secondary syphilis in the woman—implying that she has from some source, or from some cause connected with the state of utero-gestation, imbibed a fresh dose of poison?

On this point, and speaking apparently of uterine syphilitic affection, Dr. Tyler Smith stated at a meeting of the Medico-Chirurgical Society, that "he had observed in such cases, that at each pregnancy a fresh dose of the syphilitic poison is imparted to the mother, unless in the meantime the husband had been the subject of anti-syphilitic treatment."* Mr. Hutchinson's experience as regards the ordinary secondary symptoms is to the same effect. Our time will not allow us to refer specially to his cases, as bearing on this question; but it will suffice to quote his own general conclusion :—"Increase of symptoms and relapses may be produced by repetition of exposure to contagion"—*i. e.*, by the woman again becoming pregnant. And in one of Dr. Balfour's cases, the first in his collection, there was a recurrence of the syphilitic symptoms during the second pregnancy.

The positive value of the fact thus clearly ascertained, and still more, its value in relation to other facts, and as checking or testing these, I am disposed to rate very high.† The consideration

* See Association Medical Journal for July 14, 1854.

† As this point is important, I hope I may be excused, if, in order to bring it fully into view, I adduce here so much of Dr. Balfour's first case—the only one in which relapse occurred—as is illustrative of it :—"By the time the lady

of it, however, in this view, must be reserved till another question yet to be proposed comes before us. Nevertheless, to meet an exception that may be taken to it, it will be necessary at this stage to point out how the fact really stands. The reappearance of the syphilitic symptoms in the mother during a second or subsequent pregnancy, seems certainly to imply that she has received a fresh dose of the virus, and likewise that she has received it directly from and through the fœtus. But it does not absolutely follow that in every such instance the fœtus derives the virus from the father. In most instances it probably does. M. Vidal's case, however, is sufficient to show that it may derive it from the mother exclusively; and, in as far as it does, the reinfection of the mother comes remotely from herself. But this does not alter the character of the fact, or impair its value. Why, while long previously dormant in her system, the virus should thus again become active and reappear, we shall afterwards consider; merely remarking at present that the like question may be raised, and admits of a like solution, in regard to the power of the virus when long dormant in the father's system to infect the child.

4. Another question still remains — Whether ever a woman derives secondary syphilis from her husband *unless* she conceives by him? And the question to which this stands opposed is— Whether she may not derive the virus through the *seminal fluid* also, and indifferently *at any time?*

This question, which was suggested to me by Mr. Paget, as it was, I believe, to him by the perusal of my own observations on this subject in my first paper, is perhaps the most important of all.* Previously, however, to taking it up, the consideration

recovered from her confinement, and the lochial discharge had ceased, all trace of the syphilitic affection had entirely left, and she was so completely restored to health that I did not deem any medical interference necessary. She enjoyed excellent health, and made a good nurse, and she continued quite free from any return of the disease *for more than fifteen months*, when she again became pregnant. About two months after this occurred, a train of symptoms precisely similar to the first appeared, and continued as formerly up to the time of her confinement, when they again disappeared, as on the former occasion. The second child was also syphilitic." It cannot well be supposed that during the interval between the two pregnancies, no sexual intercourse obtained between this lady and her husband till just previously to the second; and that the relapse and the reimpregnation were merely coincident.

* The alternative question might have been framed in more general terms so as to include tainted *preputial* as well as *urethral mucous secretions*, together, with exudations from vesicular or pustular eruptions on the penis. But I have restricted it to the seminal fluid in accordance with Mr. Paget's communication. "I would venture to suggest," Mr. Paget wrote me, "that you should try to find whether ever a woman derives secondary syphilis from her husband *unless she conceives by him*. Facts bearing on this point might prove that secondary syphilis is not communicated directly by the *seminal fluid*, but by the child begotten with it; and this mode of inoculation being proved, would go far to prove the foundation of your theory."

already referred to, as to the power of a seemingly *dormant* virus, demands attention. And it does so as enabling us to understand why—if the fact be so—it is through the fœtus only, and during pregnancy alone, that the female derives the virus from her husband, or, in a few cases, virtually reinfects herself; and, likewise, in the absence of positive proof, and until such proof is adduced, that she may and does, making it *probable* that she will not and cannot derive it through the seminal fluid, and indifferently at any time.

Now, although in many cases, in the lower ranks of life particularly, the syphilitic symptoms in the husband may continue to show themselves, and be severe or well-marked even after marriage, the general fact, I apprehend, is, that among all classes, and certainly in the higher, they have long previously disappeared; that in a large proportion of the cases they were originally mild, or very slight in the later periods of their manifestation; and, further, that, to all appearance, the virus itself has been completely eradicated from the system. Facts, indeed, clearly demonstrate that it must still be present there. But facts demonstrate, also, that it must be present in such quantity and in such condition as to be incapable of again exciting actual disease in *him*. And the question arises—how, under such circumstances, should the virus be capable of tainting and sensibly affecting the *child* begotten by him? The answer is not far to seek. In accordance with the known laws both of physiology and of the morbific poisons, the fœtus, being an entirely new formation, and the seat and subject of nutritive changes of great activity, will be *peculiarly susceptible* of the virus; and the virus itself, although existing in the father's system, and imparted by him to the fœtus in a quantity which may be infinitesimally small, acting as a *ferment*, multiplies itself in the blood of the fœtus, and at length *accumulates* there in such quantity, acquiring at the same time such *efficiency*, as to produce in it *manifest* syphilitic disease. The like explanation will apply to the cases in which (as in M. Vidal's), the virus is derived by the fœtus from the mother. And that the fœtus thus contaminated, whether by the mother or the father, and thus actually affected by the poison, should readily infect or reinfect the mother, is only what might reasonably be expected. Thus, strictly speaking, it is not the father that *directly* affects the mother. It is the fœtus that does so. The virus, in passing from the father to the mother, passes first into the system of the fœtus, and there multiplying, acquires the requisite power. And it may be, and I confess I cannot but believe that it is *thus* only that, while *latent* in the system of the husband, or appearing only in the *slight* and *mild* tertiary form, it ever *will* or *can* affect his wife.

To revert now to the questions put a little ago. We have already seen that the female may, and often does derive syphilis

from her husband when she conceives by him, and that—whether remotely derived now from herself or not—she may, and often does *again* exhibit the like symptoms on a subsequent impregnation. But may she not also do so independently of conception, and at any time? May not sexual intercourse alone, and the mere application of the seminal fluid, or of tainted preputial secretions, suffice to impart the virus to her? I am inclined to think not. It were a rash assertion to say that its occurrence is impossible, and one at least which it would require a large amount of negative evidence to make good. We know that a syphilitic infant may infect its nurse, and it is clear that the infection must come to her through her own nipple and the infant's mouth—abrasion or fissure of the nipple being probably a condition essential to the result. In this case, however, we have an infant labouring under *actual* syphilitic disease; and if infection be possible in the case of man and wife without impregnation, I apprehend it is so only in cases in which the man is at the time *actually diseased*, and of these, probably in those only, in which the female is labouring under ulceration or abrasion of the os uteri. But that it is possible in cases in which the syphilitic affection in the man is altogether latent, or shows itself in its slighter forms, and the mucous surfaces in the woman are entire—I do not believe. Yet it is in cases of this description that it is so often seen in connection with pregnancy—the woman healthy, and the man without any ostensible syphilitic affection.

The question, however, is one of fact. A single well authenticated and unequivocal instance of infection occurring independently of conception, would establish the infecting power of the seminal fluid; or else of certain others of the male genital secretions. Have any such cases ever been met with and recorded? Dr. Balfour refers to none, and seems to consider it certain "that the mere fact of coitus between the woman and her husband is not sufficient to produce the disease." Mr. Hutchinson states that he has met with but one case of this class; but he regards it as a doubtful one, and he holds it to be "extremely doubtful if 'contagion by the seminal fluid' be possible"—observing "that cases are extremely rare in which married women who have never conceived, become the subjects of constitutional taint without having had primary symptoms, and are in all probability to be explained as errors of observation." And, as we have seen, M. Ricord states emphatically, that "when there are no children the mother does not suffer." I have not myself met with any, although for nearly eight years I have had my attention directed to cases of this description; nor have I been able in the course of my reading to find any described. It is true that, writing in 1850, I said—"I fear it will turn out on inquiry, that secondary syphilis may be transmitted directly by the seminal fluid, independently

of conception."* But this apprehension was founded, contrary to my hope, on answers I received to inquiries made of some professional friends of experience in this department of practice, who *thought* they had seen cases in which the female had been tainted by her husband, and (the idea of inoculation through the fœtus being new to them, and appearing strange) were persuaded that the contamination *must needs* have occurred through the seminal fluid. To the apprehension, however, then expressed, I appended this remark:—"but perhaps it may appear also that its transmission in this way (*i. e.*, by the seminal fluid) is *occasional* only and *uncertain*, while it is *very frequent*, or almost *inevitable*, when conception follows intercourse"—adding, that "a comparative observation of this kind, if clear and undoubted, would be nearly equally decisive."

And such I think is the ground we should still take, and the spirit in which our investigations should still be carried on:—An admission, on the one hand, that inoculation by the seminal fluid, or otherwise than through conception, is not *so* impossible in certain cases as to prevent us carefully watching those in which, if it be possible, it may peradventure occur, or make us absolutely to reject all evidence which may be offered in support of the transmission of the virus in that way; and, on the other hand, a reference of the whole question at issue to trial by *comparative* observation so soon as a sufficiently large number of observations have been made. And I think we are now in a position, and that mainly through the labours of Mr. Hutchinson and of Dr. Balfour, to submit the question to this issue. The inquiry is still in its infancy; but the facts already ascertained appear to be sufficiently numerous to warrant our drawing certain inferences from them, and, in particular, the following:—*first*, that if inoculation, independently of conception, be possible, it at least occurs very rarely—so rarely as to make it doubtful whether it ever does, and, further, that no proof yet exists to show that it ever has occurred; and, *secondly*, that inoculation by the fœtus—never suspected till within these few years, and by many still thought improbable—is not only possible, but is at once the mode in which it almost invariably (at least) occurs, and of very frequent occurrence besides.

Nothing, I think, can now set aside these conclusions. Further observations may modify them; but the evidence on which they rest is large enough and clear enough to enable us confidently to rely upon them. And its strength lies not merely in the number of instances in which different females have become infected with secondary syphilis when they conceived, *and then only*, but in which the same female has been reinfected simultaneously with

* Monthly Journal of Medical Science for October, p. 303.

reimpregnation, and *at no other time*—continuing *free* from syphilitic affection *for months together*, although cohabiting with the tainted husband, and then *suffering again* on *again* becoming pregnant. It is this which makes the evidence not merely convincing, but irresistibly conclusive.

III. This properly concludes what I have to say on the *fœtus in utero* as the channel of transmission of secondary or constitutional syphilis from the male to the female parent. Practically important on its own account as the subject is, it is physiologically important also, as a test or criterion of the general doctrine of inoculation through the fœtus, as originally suggested by Mr. M'Gillivray. Did our time permit, I would endeavour to point out its bearing in this relation, and its peculiar value. I would proceed to show you that there probably are in nature two sets of cases, or two classes of facts connected with the peculiarities seen in the offspring of mammalia, *analogous*, nay, *identical* in their *external* characters, but widely different in their origin and in the conditions of their production; the one referable to *mental* states in one or other of the parents, but oftenest to mental states in the female, and operating on her either at the time of conception or during the early stage of pregnancy;* the other to a change effected in the constitutional and reproductive powers of the female by a *physical* agency, originally extrinsic to her, but inherent in a former fœtus by derivation from its male progenitor, and conveyed to her in the way of inoculation by that fœtus while *in utero*.

To establish this, and to demonstrate also the relative proportion in which the two sets of cases obtain, together with the comparative efficacy of their respective causes, and the conditions that are essential to the action of each, would be an achievement in physiology of great general interest; and, in relation to the practice of medicine on the one hand, and the breeding of stock on the other, of real practical importance.

But, in the investigation of one of the branches of this subject, the first and essential point is to make good the law or principle

* Ex. gr.—"A mare and a horse (a gelding) had for some years worked together on the same farm, occupied adjacent stalls in the same stable, and pastured together in summer in the same fields. The gelding was of a black colour, with white legs and face, and had a singular peculiarity in the form of the hind legs, which, when the animal was standing, appeared as if quite straight, there being no appearance of the leg being bent at the hough-joint, as in ordinary cases; the pasterns, likewise, were very long, so as to cause the feet to look as if placed almost at right angles to the legs. After having been some years thus associated with this gelding, the mare was covered by a stallion of the same colour with herself—both stallion and mare being of a bay colour, with black legs and a small spot of white only on the forehead. The foal, which was the produce of this connection, very exactly resembled the gelding in *colour* and in *shape*; and very remarkably in the shape of the hind legs, as above described."—On a Remarkable Effect of Cross-Breeding, p. 13.

on which it rests—to be well assured that the foundation is secure. And it is in this point of view that the inquiry as to syphilis is so important. As regards *it*, there can be no room for suspecting that *mental* causes can possibly be concerned in the transmission of the virus from the father to the mother. A child born into the world with unmistakeable manifestations of syphilis on its body, begotten by a healthy man, but the fruit of a second marriage, and the first husband and the children of this marriage syphilitic also, could not for a moment be supposed to have come by it *per fortem imaginationem* of its mother, as it might be, if, instead of syphilis, it had the nose or the features of its mother's former husband. As little likely is the mother, by such an effort of mind at the moment of conception, thus to contaminate herself.*

Our time, however, will not allow me to do more than to indicate in this brief manner the value of the syphilitic test of the inoculation *per fœtum* theory; and to add, that, while it seems to me to establish it beyond the reach of all doubt or cavil—the great extent to which recent observations have shown the general fact exemplified in Lord Morton's mare to hold among cattle, sheep, dogs, horses, and others of our domestic animals, as compared with the limited extent to which mental causes are seen to influence the development of offspring, sufficiently demonstrates that in *the great majority* of cases of this description, the *inoculation* principle is that concerned in their production.

IV. There is yet another test of this inoculation principle, for the suggestion of which, as of the former, I am indebted to the sagacity of Mr. Paget. The phenomenon exemplified in Lord Morton's mare *ought*, by the theory, to be *restricted* to animals of the class mammalia, that is, to animals in which the young are developed within the body of the mother, and between which and the mother there exists a placental connection during the whole period of intra-uterine life. But is it actually so restricted? May it not be seen in *birds*, in which the young are wholly developed outside the mother's body, and between which and the mother there is no placental connection?

Now, among bird fanciers crossing is perhaps as extensively practised as it is with our domestic quadrupeds. Nothing, for example, is more common than to breed the barn-door hen with the bantam cock one year, and the next year with a cock of her

* There seems to be with some as little limit to the physiological agency of the imagination as to their own exercise of this faculty. In a case of legitimacy that came before it, the parliament of Grenoble went as far as to declare the issue legitimate, although the husband had been four years absent from his wife, on the ground of an allegation by the latter, that she had conceived through the power of the imagination alone:—"Ut mulier per fortem imaginationem putaverit se in somnis rem habuisse cum marito, atque sic concepisse." Had the child, however, been born syphilitic, it is possible that the parliament might have come to a different decision—straining at a gnat, albeit making no difficulty of swallowing a camel.

own kind; or the hen-canary one year with either the cock-linnet or the cock-goldfinch, and the next with the cock-canary. In these and other cases of the like kind, cross birds or mules are obtained the first year; and these, in accordance with the law laid down by Mr. Orton of Sunderland, take very strongly after the cock. But are the birds obtained the following year, when the parent birds are both of the same species and of the same breed, perfectly *pure*, that is, without taint or trace of any of the characteristics of the male bird with which the hen was bred from the previous year?

On this point I made very extensive inquiry several years ago in quarters where information was to be had, carefully concealing the object I had in view in making the inquiry; and the uniform result was, as I stated in 1851, in my pamphlet "On a Remarkable Effect of Cross Breeding,"—that no trace or admixture of those antecedent male birds is ever seen in subsequent broods, the birds produced being in every instance as pure as in those in which no such crossing had been previously effected with the mother-bird.

It is but right, however, to state that two different individuals, writing quite independently of each other, have alleged that the reverse is the true state of the case. One of these is Mr. Orton of Sunderland; the other, the reviewer in the *Aberdeen Journal* newspaper (February, 1851) of my pamphlet on cross breeding.

The latter, quoting my remarks on this point in the pamphlet, says:—

"He gives Mr. M'Gillivray's theory one advantage which is not its due. So far from it being the case that the peculiar effect of crossing here in question, fails to be seen in birds, it is in them that it is most of all apparent. It is well known that a hen-canary is rendered useless for breeding pure canaries by crossing for mules. After a cross with a goldfinch or linnet, no peculiar feature of these birds may turn up in subsequent broods by a male bird of the hen's own species, but every nest will be discoloured; and so long as the hen produces young, it will be remarkable indeed, if a clean yellow canary be found among her progeny. This," he adds, "may easily be certified; and we are inclined to think that it must be regarded, as in a great measure destructive of the theory of the purely physical cause of inoculation."

And so it would if true, but for the syphilitic test. This, it seems to me, places the theory in question beyond all doubt. But without speculating at present on the conclusions to be drawn from the fact, if the case be as this writer represents it, let us first consider whether it be a true or a "false" fact. Now, on reading the statement I have quoted, I sought and obtained a personal interview with the writer, and found that he could speak much less confidently than he had written, and, in particular, that he wrote from hearsay, and without any personal knowledge or experience in the matter. Having ascertained this I subsequently renewed the inquiries I had formerly made, with the same precaution as before to conceal the object I had in view, but among a larger number of

bird-fanciers, and with respect to a much greater variety of the bird-tribe. The result of this inquiry was, in every instance, the same as that of the former inquiry. None of my informants had ever seen anything that corresponded with the statement of the writer in the *Aberdeen Journal*.

More recently Mr. Orton, taking exception to the inoculation theory altogether, observes:—

"The hen does not carry her offspring *in utero*. Notwithstanding, her offspring, *as we have already seen*, are as liable to be influenced by the action under discussion (i. e., by crossing) as are those of mammalia."

And again—

"Hens and their offspring have not this uterine connection; consequently, they cannot undergo the process of inoculation laid down, yet they are, equally with mammalia, the subjects of this peculiar law of breeding." *

In the latter statement, Mr. Orton speaks absolutely; in the former, from an instance in point previously referred to, or adduced in some part of his paper. In all probability the *absolute* statement is founded on the *specific*, and to be judged of by it. Now, on referring to the instance which proves the ground of the latter, it is seen to be such an instance as would not be relied on, or regarded of any value whatever, in the case of a mammal. It is a case, not in which a hen-bird was bred from with a cock of her own breed, after having previously been bred from with a cock of a different species, but the reverse of this—a bantam hen having been bred from with a cock silk-fowl, and producing cross birds, after having been bred from the previous year with a bantam cock; and the observation relied on by Mr. Orton as fatal to the inoculation theory, was, that some of the birds of this brood bore a greater resemblance to the bantam, and less to the silk-fowl than did others. But this by no means warrants the inference, that the more decided resemblance of them to the bantam was due to the influence of the bantam cock of the preceding year. And I will venture confidently to affirm, to take a case in which a considerable number of young are thrown off at each litter, that, if a sow of a particular breed and colour be served with a boar of a widely different colour and breed, the sow never having been bred from before, the litter will be *mixed*; some of the young pigs taking chiefly after the boar, others of them chiefly after the sow. This test therefore, which may be called the *bird test*, if it cannot as yet be used in support of the inoculation theory, cannot at any rate be brought against it.

V. In my former papers on this subject of maternal inoculation through the fœtus, I referred in connection with it to a remarkable statement made by the excellent Count de Strzelecki,

* On the Physiology of Breeding, in the Newcastle Chronicle Nov. 10, 1854.

as to the effect of fruitful intercourse between the aboriginal female of certain countries and the European male, on the subsequent fertility of the female with a male of her own race. The count's statement, founded on "hundreds of instances" coming under his own observation, unqualified by the observation of a single exceptional instance, and bearing on the aborigines of Canada, of the United States, of California, Mexico, the South American Republics, the Marquesas, the Sandwich and Society Islands, and those of New Zealand and Australia, is, that "whenever such intercourse takes place, the native female is found to lose the power of conception on a renewal of intercourse with the male of her own race, retaining only that of procreating with the white men."*

Forbearing to trouble you with the application I made formerly of this statement, on the assumption of its being the expression of a general law of nature, to the theory under consideration—I think it right to mention with regard to the statement itself, that while I then suggested the propriety of "keeping the mean between the two extremes of too much stiffness in refusing, and of too much easiness in admitting" it to be a matter of *fact*, I have since received such information as warrants me in refusing to admit it as being *universally* true at least, in respect of certain of the races specified by the Count de Strzelecki. My friend and former pupil Dr. William Sim Murray, then of Her Majesty's 20th regiment of foot, writing me in November, 1851, from Montreal, where he was quartered, says:—

"Since my arrival here I have had an opportunity of speaking to Dr. French on the subject of the Count de Strzelecki's observation, and he tells me that, his attention being directed to it some time ago, he had made inquiries regarding it, and that he had obtained *positive* assertions that *squaws* frequently do have children by an Indian after bearing children to a European."

Again, Dr. Balfour, of Surinam, writing me in April, 1851, observes:—

"Living for many years in this district of South America, surrounded by a motley population of Whites, Negroes, and Indians, I have had very frequent opportunities of observing that Strzelecki's opinion of the effect of fruitful intercourse of the European male with the women of the aboriginal race (Indians here) is not borne out by the facts constantly occurring. The Indians of this district, known as the tribes of Warou and Arrawacka Indians, are certainly decreasing fast, but principally from the effects of that curse of the aborigines—*fire-water*. The women of these tribes are notoriously profligate during the earlier part of their life after puberty; but after bearing children to Europeans are *frequently* fruitful with men of their own race. This is particularly observed with the women of the Warou tribe, a camp of which tribe is in the immediate neighbourhood of the barracks, where a large detachment of European troops is constantly stationed. Any person resident in this district could at once refer to examples of this."

* Physical Description of New South Wales and Van Diemen's Land. pp. 345, 347.

And if I mistake not, a paper was published in the *Edinburgh Monthly Journal* in 1851 or 1852, in which the author, either writing from the antipodes or giving the results of his observations while there—whether in Australia or New Zealand, I do not now recollect—states that *exceptional* instances, at least, to Strzelecki's observation are of frequent occurrence among the aboriginal females of one or other or both of these countries.

I have thus endeavoured as impartially as one partial to the inoculation theory well could, to lay before you a complete general view of this interesting and important subject. I have referred to every fact known to me as bearing upon it, and have omitted nothing that seems in any way adverse to the theory in question. And submitting, in particular, the *bird test* and the *syphilis test* to the consideration of those among you who may take an interest in that theory, I will conclude by quoting a suggestion made by Dr. Carpenter in regard to the latter, but in its principle as applicable to the one test as to the other:—"As this is a point of great practical importance, it may be hoped that those who have the opportunity of bringing observation to bear upon it, will not omit to do so."*

Since the foregoing paper was written, and read to the Medical Society of Southampton, several important communications bearing on the subject of the inoculation theory have appeared in the *Medical Times and Gazette* and in the *Lancet*, from Mr. Hutchinson,† Mr. Savory, of St. Bartholomew's Hospital,‡ Mr. Langston Parker, of Birmingham,§ and Mr. De Mérie, of London.‖ I purpose considering these several communications in a future number of this journal, and will merely remark, meanwhile, that, by his well-conceived and admirably-executed experiments with strychnine, whereby he has demonstrated (what Magendie and Williams failed in demonstrating by theirs), that fluid matters do, in fact, pass from the fœtus to the mother through the placenta, Mr. Savory has added to the chain of evidence in support of the inoculation theory, the only link wanting to make that chain complete. His discovery, for such it is, may be said to furnish the keystone of the arch on which the theory now rests, and rests securely.

* Principles of Human Physiology, 5th Ed., p. 826.

† "On the communication of Syphilis from the Fœtus to the Mother." Medical Times and Gazette, December 20, 1856, and January 10, 1857.

‡ "An Experimental Inquiry into the Effect upon the Mother of Poisoning the Fœtus." Lancet, April 10 and 17, 1858.

§ Lectures on Infantile Syphilis. Lancet, May and June, 1858.

‖ Lettsomian Lectures—Lecture III. "On Hereditary Syphilis." Lancet, September 12, 1858.

THE

FACTS AND LAWS OF LIFE;

BEING

An Introductory Lecture

DELIVERED AT THE OPENING OF THE MEDICAL SCHOOL

OF

THE WESTMINSTER HOSPITAL,

ON OCTOBER 3RD, 1859.

BY

J. RUSSELL REYNOLDS, M.D. LOND.,

FELLOW OF UNIVERSITY COLLEGE, LONDON; FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; ASSISTANT PHYSICIAN TO THE WESTMINSTER HOSPITAL; AND LATE ASSISTANT PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN.

LONDON:
JOHN CHURCHILL, NEW BURLINGTON STREET.
1859.

LONDON:
R. GEOGHEGAN, PRINTER, 6, BOUVERIE STREET,
FLEET STREET.

THE

FACTS AND LAWS OF LIFE.

GENTLEMEN,

WHEN William Tell had resolved to aim his arrow at the apple on young Tell's head, neither the malignant taunt of his oppressor, the timid entreaties of his friends, nor the innocent confidence of his boy, could deter him from his purpose, or diminish his chances of success. Gessler may smile grimly as young Walter takes his stand beneath the distant tree, still as a statue, but without one touch of fear—the crowd of friends may be breathless with horror at the thought of failure—but yet Tell lifts his "trusty yew," and draws its well-tried string. But there is a lengthened pause; and, although the skill and daring of the man are "household words" among the young huntsmen of the Alps, Tell lets his cross-bow fall;—and why? Strong passion has dimmed his sight; he cannot see the mark at which he aims. No matter how vigorous may be his arm, nor how firm his resolve; no matter how skilled he may be in his art, how pure in his motives, how earnest in his effort; no matter how strong may be the confidence with which long years of success have inspired his soul; no matter how much may hang upon the issue of his attempt, he cannot, he knows that he cannot, succeed. He might display daring, energy, and skill, but yet he would miss the mark.

In the drama of "Wilhelm Tell" great historic events have been preserved, with such marvellous fidelity, beneath the poetry with which the genius of Schiller has adorned them,

that the strong limbs of the world's real history give all its form and movement to the poetic dress his art has so thrown around them as to hide only the ugly or unimportant, and reveal only the beautiful or the true. The same genius may be seen, perhaps less conspicuously, but not less really, in the minute incident, or "aside" allusion, I have quoted. In it we may so recognise the faithfulness with which the artist could observe and render Nature, as to see, that in her finer features there are signs of the oneness that gives authority to all her teaching, and renders her lessons applicable to all conditions and all times.

That which is true of the Alpine huntsman's cross-bow, is also true of every kind of endeavour that man may make. If he would be successful in his work, he must keep the object at which he is aiming, both well and constantly in sight. But it often happens that his sight is so dimmed that he cannot see his mark; and then, no matter from what source the dimness comes, should he persist in his effort, the results would be disastrous. The fault of sight is not to be conquered by energetic exertion; for, indeed, the very energy with which an effort may be made, is not unfrequently, the cause of failure.

Such fault is not peculiar to one profession; it is common to our race. Man, whatever his work may be, does often lose sight of his true and acknowledged aim. The admitted aim of the Counsel's presence at a trial is the just application of evidence and law to the particular case under consideration; but it has occurred, in practice, for the barrister to forget his mark, and his aim has sometimes appeared to be the establishing of what is false, and the perpetration of a wrong, the more keen because inflicted under the garb of justice. Political leaders, whose true aim is the welfare of their country, have been thought, sometimes, guilty of such dimness of sight that, to the ignorant lookers-on, their most energetic efforts appear directed rather to secure the welfare of their party. "Conservatism" is the avowed aim of some; yet so strong has endeavour been on its behalf, that one, at least, has exhibited the *reductio ad absurdum* of his principle, and has avowed his object to be the maintenance of that which is, no matter whether it be a state of stagnation or of progress. On the other hand, we have seen the Reformer so eager in his work, so "radical" in his resolution to turn all things upside down and inside out, that he has

brought again into being and pre-eminence the very abuses that his forefathers had renounced and removed.

If then, the barrister, in his most earnest pleading, may be so wide of the mark that he regards simply his client's welfare, or still worse, his own reputation for fine speaking; if the political leader may be so benighted as to think that the interests of his party are identical with those of the community; if the Conservative may do the Reformer's work, and the latter accomplish a retrogression in the affairs of State, we must not feel surprise if the medical student,—whose true aim is so to learn the facts and laws of life as to utilise his knowledge, in every way and to the highest degree, for his fellow-men,—we must not blame the student of medicine too severely, if, in his eagerness to attain this end, it should sometimes appear that he is but learning the names, and the most easily acquired means of so distinguishing the most ordinary diseases, as to pass an examination, and make use of his diploma for himself.

But, although things are not what they ought to be, neither are they what they seem; and these are but the extremes —the one the highest, and the other the lowest—between which the student of medicine practically takes his aim. It is scarcely possible that he should, perfectly and exclusively, realise either the one or the other. There is no man, no matter how lofty may be his aim, nor how earnest his endeavour, whose whole career is free from all taint of either selfishness, prejudice, or short-coming; neither is there, I trust, any student of medicine,—no matter how poor may be his conception of the work he has taken upon himself to do, nor how lazily he may be inclined to do as little as is possible to "pass" just safely, rather than to practise ably,—there is, I believe, no one, even thus unworthy of the rank he now holds,—who is not sometimes moved to sympathy with the sufferings of his fellow-men, sometimes eager to know more than mere "passwords" of the great mysteries of Nature by which he is surrounded, and into which he is urged to look; and sometimes desirous of hereafter throwing in his mite into the treasury of scientific knowledge, and lightening, by his aid, the heavy burden of humanity.

But, if the aim and work of the student may graduate between such extremes, and if in all individuals there is some battling of the higher with the lower tendency, our object should ever be so to cultivate the one and repress the other, that the student of medicine may become the true man of

science, of art, and charity; and that he may become this, he must constantly keep his aim in sight; it must be that which gives unity and form to all his actions, his purposes, and thoughts.

The huntsman's arrow is not, however, always on the wing; nor is he always, with strained eye, keeping the mark in sight: there are moments when he burnishes the shaft, or mends the broken string; times when he plays with his weapon or lays it aside in rest. And so with another life-work: it is not that, at every moment, the eye, and heart, and hand must be fixed on one end, and working in one groove. Such treadmill-like occupation might grind corn, and be more wholesome than utter idleness; but the man would be thus using only a fraction of his energies, and would himself make no advance. It is not that the constancy of your purpose is to be the fetter to confine your limbs, nor the dead-weight to drag their movements down to a dull uniformity; but it is to be the harmonising power—that which gives resultant direction to, and ensures progress in, the thousand-fold differently-pointing and acting forces of your being. There are, however, special occasions upon which a man's occupation may, with advantage, be the reconsideration of his "aim;" the removal from it, and from himself, of obstructing and confusing elements; and the gathering together of his powers for renewed and more successful exertion. No occasion presents greater claims to be thus employed by you than does the first day of a new period of your work, when you resume old studies and enter upon new—take stock of your intellectual property, and ascertain the balance of memory and forgetfulness—arrange your plans of work and of recreation—and start again forward in your life-career.

The aim of the student of medicine is, as I have already said, so to learn the facts and laws of life, in both health and disease, as to utilise his knowledge, in every way, and to the highest degree, for his fellow-men.

The *subject-matter* of your study, then, is LIFE.

There are many things and many principles which it is almost impossible satisfactorily to define; but yet, upon these very subjects we know so much, that nothing is easier than to criticise and pull to pieces the attempts at definition made by others. I remember to have seen a number of gentlemen —barristers, physicians, clergymen, and others—formerly the pupils of a college in the metropolis,—who were

assembled to discuss the principle lately adopted by the University of London, of admitting candidates to the examination for degrees in Arts independently of their having attended any lectures at a college. These gentlemen, congregated for the very purpose of arguing in favour of a college education, were absolutely silent when asked by the distinguished historian of Greece to define what a college was! We may know very much without being able to define, for a true definition is much more than a mere means of recognition, with which it is not unfrequently confounded. We may recognise an individual by his walk, or by his hat; but neither a military gait nor a peculiar head-dress would be a definition of our friend. Again with man, he may be the only animal that cooks his food; but the definition of man that he he is "a cooking animal" is eminently unsatisfactory. It is comparatively easy to discover means for distinguishing one object from some other, or from some other hundreds of objects; but a definition must be the statement of that which invariably distinguishes it from all others. In order truly and comprehensively to define, knowledge is required co-extensive with the facts, and accurately correspondent with them; and there is little—nay, there is nothing,—with regard to which our information is thus perfect; for, at the best, we know but in part, and see all things, "as in a glass, darkly," as unsolved problems for our study or our faith. This partial character of our knowledge is eminently true in regard of life. Much as we know about it, there is yet so much more that we do not know, so much that we feel not only may be, but is, and yet is behind some veil our sight cannot penetrate; near to us, and yet so placed that we cannot grasp it with our hand; so like ourselves in another that we feel that we are one, and yet so far separated by the dense wall that comes between soul and soul that we know each is individual and alone; so much that points to some principle where consciousness and experience may be one, and yet so much that rises up between us and that principle, that hitherto it has escaped our most earnest gaze:—there is so much that is mysterious in life, so much that is attractive in its mystery, that other sciences have been made but stepping-stones to a knowledge of this, the highest science; and yet, with all these aids, and with our two-fold method of arriving at its truths—viz, consciousness and observation—there are none which have so persistently and entirely eluded our grasp. Still the attempt is carried on,

and life is the subject-matter of your study; and your endeavour must be to know accurately that which can be known about it, and to know it all.

The view which we take of life will influence deeply the method of our study and our action. If definitions are defective, still they indicate tendencies, or directions, along which investigation has proceeded; and our adoption of one or the other will affect, if not determine, the direction of our own effort. It is not my purpose to examine definitions now; I will merely enumerate four classes of definition, for the purpose of showing that all they have yet accomplished is to give hints of the directions in which investigation should proceed. Whether we define life in some such abstract terms as the "tendency to individuation"—a definition which confessedly includes stocks and stones as well as trees and men;—or, in such chemico-mechanical phraseology, as "a double interior movement of composition and decomposition"—an expression which, if adopted, renders it difficult to see wherein life differs from a galvanic battery or a common fire;—whether we regard it as a "divine aura," and thus place it at once beyond the reach of all physical investigation; or as a particular expression of some general physical force, thus making it to agree, in essence, with heat, light, and electricity; whether we choose the one or the other of these phrases, by which it has been sought to define the nature of life, we shall fail in finding it to be a satisfactory definition, and we shall gain little real help from any one of them, unless we recognise that no one of them is in itself fully comprehensive; but, on the other hand, we shall lose much unless we see that there is in each some truth about life which it would not be well for us to pass by. Each expression I have quoted, although it points out some striking feature of life, yet leaves undefined that which it seeks or pretends to define; and we are left with these phrases, these hints of direction, reared as finger-posts, at long distance from each other, and each pointing from the light or twilight in which it stands to some spot in the darkness, at which their converging lines may meet. We may take our start from either of them, and advance freely to a certain distance, for the light is still around us, and our path beaten by the steps of those who have gone before; but soon the darkness comes, our path wanders and is lost, and we must retrace our steps, or stumble on into the still deeper night. Yet we know, we feel certain, that there is some point wherein all these lines must meet; we feel

confident that they do not pass onwards and onwards into the infinite, without relation to each other, without meaning, and without end; but that there is a central truth towards which they lead; and hidden though it has hitherto been, and still may be, from human eye, yet it is a reality which gives meaning to all that tends towards it, to all that lies around it and above; and that its discovery is the goal towards which all successful investigation of life must be directed.

Life, then, as the subject-matter of your study, embraces much more than it sometimes appears to do in your physiological hand-books: in it is wrapped up, besides bones, muscles, and intestines, the being and destiny of humanity. It is to be studied in the silent and solitary depths of your own consciousness, as well as in the lecture-theatre and dissecting-room:—you must see it in the minds and hearts of your patients, as well as in their limbs and viscera:—you have to deal not merely with that which may be analysed, experimented upon, cauterised, bandaged, or cut off, and cast away; you have to do not only with those pains which may rack the body or disturb the mind; you have for your study not only the material fabric, which is so mysterious in its facts and processes;—but you have also for your study its still more mysterious tenant; you have to do with that which can rise superior to all weakness, and can triumph over all pain; and which, in the very article of death, may utter the prophecy of a life,—as yet unseen, but still felt to be, and to be more real, more strong, than that which, though so real, is now just sinking into silence and decay,—a life, whose witness here, having just burned into your soul's deepest creed the facts that it is, and that it cannot die, is itself, in another moment, carried beyond your sight.

If, then, you would so learn of life as to alleviate its woes, you must not regard your patients as merely interesting clinical phenomena; you must not look on them as cleverly-acting physiological machines, upon which you may experiment for your own diversion; you must not imagine that "cases" are brought before you for the purpose of illustrating books, or of proving so-called statistical laws—that diseases are existing for the sake of teaching you the long names which men have given them, or for the sake of establishing this or the other pathological opinion; that limbs have been created and broken that you may feel the "crepitus" of fractured bones; or blighted, that the surgeon may display his skill in their amputation;—you must

take far other views than these, and see, in each sufferer who may come before you, that there is an individual history, as interesting, perhaps far more so than your own;—you must see that disease is something far more important, and far deeper than an aching head, a hurried breathing, or a fluttering pulse; that disease is something much more serious than the mere interference with the mechanism of physical life; that the measure of its evil is, not the increased rapidity of pulse, not the daily wasting of the body, nor its numerical frequency in the bills of mortality, but the degree to which it so tells upon the mind, heart, will, and power of man that it prevents him from doing that work in this world which it has been given him to do. Whatever pathological creed you may adopt as to the essence of disease, be assured that this is the true measure of its magnitude, the true standard of its evil. He, who after having toiled arduously and successfully for years, yet when having completed his threescore years and ten, amidst much weakness, distress, and pain, can still carry on the work that he was accomplishing, and, while on his death-bed direct others in their work, and aid them by dictating his own latest thoughts, is far less diseased, though his malady may invade whole organs, and may be of the most so-called "malignant" order,—yet for all the highest ends of life he is less diseased than is the wretched and useless hypochondriac, who, with strong limbs and mighty appetite, is, and has been for thirty or forty years, a mere drag upon society and friends, a torment to himself, a useless, actless, complaining cipher, always fearing the evil, which at last comes, it may be, by so-called accident, and then the most acute pathologist, with micrology and chemistry to help him, can find no trace of malady in any one of his long-maligned, but, even at the last, most creditable organs.

The mysterious material body which is your care, must be regarded as but a subservient part of a more mysterious whole; and you must never forget the higher elements in your eagerness to understand the lower.

While the subject-matter of your study is vast, complicated, and attractive, it is such that you cannot know it in its essence. Nevertheless, you may learn its *facts* and *laws*, for these come within the range of our powers. We have to learn about life just that which we have to learn about heat, light, electricity, or gravitation. The nature or being of any one of these we do not know; but, by reason of their less

degree of complication, we have become acquainted with a larger number of their facts and laws. The same kind, however, if not the same degree of knowledge, is possible in regard of life.

The word FACT is used in two senses. It is employed to designate an actual occurrence, a something done. Fact, in such sense is equivalent to that which is, or was. But it is used, as commonly, to denote a statement of what was, or is, according to the testimony of an observer. Fact, therefore, means correctly that which is; incorrectly, that which is said or believed to be. In the former sense, we speak of the "*facts of Nature;*" in the latter, of the "*facts of Science.*" The process of respiration is at this moment going on in each of us; certain movements are effected in our bodies; certain changes, textural and chemical, are wrought in our tissues; the air of the room is affected in composition, temperature, and weight:—these are the facts, the actual occurrences, of respiration; they exist independently of all physiological treatises; they are unchanged by our having written anything about them, right or wrong; they are the subject-matter for investigation and thought. But the process of respiration is described in books, and has been described from the time of Hippocrates to our own; the muscles and nerves that effect the movements of the chest-walls have been enumerated; the precise changes in blood and air have been detailed, and that with extreme minuteness; theories have been introduced, and respiration has come to mean a certain more or less definite chemico-vital change; but, apart from theory, and apart from all secondary meanings of the word, we say of our physiological treatises that "the facts of respiration are to be found" in them.

Now, the moment that the word "fact" is applied to a description, or to any statement of what occurs, this supposition is made, that the individual who observed the occurrence was not only capable of observing correctly, and of detailing accurately, but moreover that he did so. In other words, that a correspondence existed between his mental conception and the reality; and further, that he has represented as truth that which did really exist as fact.

Without pretending to decide whether this correspondence ever has been, or ever can be, absolutely correct; without affirming that the facts of science must always be only approximative in the accuracy with which they describe the facts of nature, this, I think, may be stated, without fear of maligning either

our capacities or our scientific systems—viz., that, at present, the correspondence is by no means perfect; and that, therefore, the facts of science should be constantly submitted to re-examination, and to comparison with the facts of nature, that their true value may be assigned them.

In order to learn the facts of life, you have to observe them for yourself, and you have to become acquainted with those which others have described; and, that you may do this successfully, great caution and circumspection are required. "What!" you may ask, "am I not quite competent to take notice of any phenomenon, and just describe faithfully what I see? Surely, this is the simplest thing that can be required of me: common sense and honesty are the only faculties required for such a purpose!" True! common sense and honesty are faculties that you require; but how rarely can even these be found, and these alone! We bring to our observation not only our abilities but our weakness; not only the truths we have learned, but the errors we have received as truths; not only consummated information on some points, but very partial and imperfect information upon others; not only honest intentions, but preconceived opinions; not only an earnest wish to know the truth, but as earnest a wish that the truth should turn out to be something we have fancied it; not only a humble desire to learn what nature teaches, but a vain and very foolish desire to find our own pet theories, or the favored theories of others, confirmed by her great authority.

But further, for the observation of certain facts, a special kind of education, and much anterior knowledge, are required; and with this very education and its results there are attendant sources of fallacy. If the means to be employed in an investigation are imperfect, the result will share that imperfection. If the object you are inspecting is distorted by the glass you employ, so long as you are ignorant of the distorting power of your glass, your idea will be out of correspondence with the fact. If either chemistry or physics be defective, when you describe a vital function in terms of chemistry or physics your description will be, *pro tanto*, depreciated by that defect. You have to guard against all these errors; and simple, therefore, as it may at first sight appear to be, to observe a fact, let me assure you that there is no faculty in which the scientific man exhibits his right to that title more distinctly than in this very power of observation; and that there is no faculty, the

absence of which from many minds has brought more confusion and more error into our scientific annals.

The facts of nature are unchanged; and surely the mind of man was as able and as honest in the days of Aristotle as it is in our own; but many of the scientific facts of his time are not those of the nineteenth century;—nay, the scientific facts of to-day are not those of the past year. We have thrown aside as errors the statements which our fathers, or even we ourselves, had regarded but a few months ago as facts proved by the latest science.

Scientific facts are not, then, very easily observed; and why they are not you will see, if you think for a moment what a scientific fact really is. It is the description of a certain kind of phenomenon, or process, in terms of two or more other kinds. It is the division of a scientific word into its syllables or letters. For example, the scientific facts of life, *i.e.* of the kind called "vital," are described in terms of other kinds, such as chemistry, mechanics, physics, chronology, and the like. The facts of the vital process termed respiration are thus resolved into certain chemical changes in the air and blood; certain mechanical arrangements of chest-walls, muscles, and nerves; particular relations in regard of time, of expiration and inspiration; certain changes in the colour of the blood; the temperature and hygrometric conditions of both the body and the medium in which it is placed. The "facts of respiration" undergo, therefore, in the attempt to state them, a process of reduction to their lowest terms; and these latter are not "vital" in their character, but physical, chemical, and mechanical. What is vital, is the link of connexion or association of these facts: *i.e.* not the letters themselves, but the words they spell.

One half of the facts of life cannot be submitted to this process of reduction; they can be stated in no simpler terms, and in no terms which are otherwise than figurative. You may compare one kind of sensation to another kind; you may qualify it by comparison with external conditions or processes; and thus you may speak of a "silvery tone," or of a "tearing pain;"—but these qualifying terms are figurative, and the words tone, pain, taste, light, feeling, and odour defy all analysis, and are insusceptible of reduction. The same is true of all the processes of mind and heart: you cannot express these in chemical or mechanical phraseology; they must have a language of their own. It is not, however, with regard to these facts that I was speaking,

but of those which are to be witnessed in the material body, and which it is the effort of science to reduce.

The reduction is constantly advancing; but at the same time, the sciences of physics and chemistry progress, and any change in them necessitates some change in the statement of the facts of life. Until all the sciences are perfected, both in quality and quantity, there must be some want of correspondence between the facts of nature and those of every science; and in proportion to the advance of the simpler sciences, the more complex must undergo changes in the statement of their facts.

These are the necessary obstacles in the way of your appreciating facts; but there are some very unnecessary hindrances, which you may at once perceive and avoid, such as careless observation, and prejudiced reporting; and there are others, to escape from which will require all your care. The capacity of humanity for creating difficulties, and then blundering among them, seems almost infinite; see to it, therefore, that you do not take *fancies* for facts.

There have been, and still are, in science, mere fictions of the mind, having no counterpart in nature; perfectly gratuitous assumptions, or groundless assertions of things which the Magi of a past, or of the present generation, may have invented, in accordance with their preconceived notions of what nature ought to be and to do. We need not go back to past centuries for the discovery of such fictions; the "archeus" of Van Helmont was not less real than "the vital knot" of M. Flourens; and in our own day, and in our common conversation, there are terms used, and that very freely, of which it is by no means easy to show the counterparts in fact. I would not say that such words as "hysteria" and "rheumatism" have no meaning, for it is well known that there are certain definite conditions associated with those names; but let me ask whether the adjectives "rheumatic" and "hysteric" are not daily used without any approximation to a meaning, and without the shadow of belief that the aches or pains so denominated bear any relation to that which is denoted by the words rheumatism and hysteria respectively? Not content with the limits of pathology, as an arena for confusion, some men have gone, with such words as their passport, into the regions of religious feeling and social history; and the present so-called "revival" in Ireland has been deliberately denominated "hysteric," and that in scientific quarters! As well might "the builders' strike,"—because the

sweat of the labourer is stopped, and his temper somewhat soured by the process,—be termed "rheumatic!" These words are, nine times out of ten, but figures of speech; the clothing of ignorance or laziness in verbal finery or would-be scientific grandeur; and this even where the substantive use of the words is something more than a mere refuge for destitute diagnoses.

Do not mistake legitimate *hypotheses* for facts; but understand, and always bear in mind, that they are hypotheses, and as such they may be of great utility. The hypothesis is useful, inasmuch as it is often a hidden thread, binding together otherwise disjointed facts into provisional groups, so that the mutual relations of these facts may be studied, and also that the group as a whole may be compared with other groups. The hypothesis, moreover, suggests an explanation; or, at all events, the direction in which an explanation may be sought. It supplies temporarily some fact, some link in a chain of phenomena, or some relation between them, which is at present unobserved. But unless your hypothesis is of such nature that it is susceptible of future verification, or the reverse, by direct observation and experiment, its continued employment is illegitimate, and it is useless, and further, it is worse than useless when its nature (as an hypothesis) is forgotten, and it is taken for and employed as fact.

Many systems of "classification" which have been of great service, contain in them much that is purely hypothetical, and which eventually yields to an improved scheme of grouping. Thus, in the Linnæan vegetable system there are numerous guesses at truth, underlying very superficial phenomena, which latter were used as lines of classification; and in the more "natural systems" of De Candolle and Jussieu, there is still some hypothesis in the construction of every "alliance," and indeed of every "order" and "genus." The hypothesis is, that there exists between the individual species of a genus some nearer and deeper relation than any which is apparent in petal, leaf, or fruit; for in one species this character is absent, in a second that character is wanting, and no one contains all, and yet not more than all, the features proper to the group. Each species ranges itself around an imaginary centre—a typical, but non-existent combination of qualities—supposed to represent and witness to other characters than those which meet the eye; and the hypothesis is, that in the essence of each individual member of that genus, there is a greater likeness or oneness than there is between members of one genus and those of another. We know not what the essence is, but all our

attempts at classification, in so far as they are successful, and that because true to nature, are approximations towards its discovery, *i.e.*, towards a knowledge of the facts of its history

There is no difference of kind between the principles upon which we base our classification of those natural objects with regard to which systematic arrangement has arrived at its highest perfection—as, for example, in botany and chemistry—and those principles by which we classify other natural objects, in respect of which our information is far less perfect, as in physiology and pathology. Our classification of disease therefore contains much legitimate hypothesis. Of this let me give you an example. In the "Letters" of Morgagni, there are to be found cases—minutely and graphically described, in which we can now readily distinguish the features of a well-known disease. For many years such cases had been, and continued to be, observed; but it was the merit of the late Dr. Bright to form them into a group, and to suggest the link of connection between their diverse symptoms.

But if you ask, "What is 'Bright's Disease?'" I reply, that it is the expression of a hypothesis. Let me remind you that there may be three individuals suffering symptoms of this disease, and yet, at certain periods of their history no one shall present any symptom precisely identical with those of either of the others. In one there is anasarca; in another, hydrothorax, or ascites; in a third there is no dropsical effusion: in one there is albuminuria; in the others it is absent; in one the head is principally distressed, headache and blindness are present; in a second, the chest, there is dyspnœa and cough; in the third, the abdomen, there is vomiting and diarrhœa: in one, the kidney is enlarged; in another, it is atrophied; in a third, it is cystiform. There is little in either their general appearance, their age, the onset of their malady, its subsequent history, or in their mode of death, in which either two of these three cases resemble each other; and yet each may be an unquestioned example of "Bright's Disease." What, then, is "Bright's Disease?" Every feature it presents in one case is absent in another; you cannot define it by this, or the other symptom; you cannot limit it to any one condition of the kidney. No; the name expresses a hypothesis—viz, that there is something common to all these cases; that there is between them a closer and deeper affinity than that of their merely

presenting in common—should they do so—albuminuria, anasarca, or the like; that in their essence—*i. e.*, in the essential character of the departure of each from healthy vital action—there is a similarity or oneness which we cannot yet define; may, perhaps, never accurately and comprehensively describe, but which, nevertheless, is; only, like life itself, it will not be compressed into our formulæ, nor expressed in our words. The various cases range themselves around some non-existent case, the centre and type, from which all exhibit more or less marked divergence. That which constitutes the centre was supposed to be a particular condition of the kidney: the hypothesis was that therein was the fundamental fact, associating the various forms of Bright's Disease. Prolonged investigation, however, appears to show that this idea is incorrect. But, in whatever direction future research may prove the true essence of Bright's Disease to exist—whether it be found in some hitherto undiscovered condition of the kidney, in the blood, in the nervous system, or in that which is denominated "nutrition"—the construction of a group of cases, by means of this hypothetical consideration, has been of the very greatest utility.

Hypotheses of the kind I have just sought to explain underlie every attempt at scientific classification, for there is no perfectly "natural system" save that which pertains in nature itself—viz., the division into species. All grouping beyond this is artificial, and but an approximation towards the truth: each grade is a step upwards towards some generalisation, or some law of life; but there are no steps, any more than there are "leaps," in nature itself: the slope, in reality, is a continuous incline; the steps are but aids, artificial lines and halting-places, which we have cut out to assist us in our progress.

In one department of your study—viz., that of "Medical Jurisprudence," you will have to recognise the existence of artificial lines, even more arbitrary than those which have been introduced into scientific classification. As students of nature with a "scientific" purpose, you fail to find points through which lines of perfectly true demarcation can be drawn, and you begin to believe that such lines do not, and cannot, truthfully exist. But, for "social" and "legal" purposes, such means of distinction must be employed; and, if natural lines cannot be found, artificial ones must be created. Scientifically considered, a man may be "insane," or "guilty" of this or that special crime; but, legally re-

garded, because he is on one or the other side of a line, which it has been convenient to draw, and which is, or was, well drawn at some period of our history, he is to be treated socially as "sane," or "innocent" of the alleged transgression. You must distinguish, therefore, between legal tests and scientific hypotheses; although the former may be based upon the latter.

But the legitimate use of hypothesis must not be confounded with its illegitimate abuse. Care is required in the formation of this implement for study,—care that it is not contradicted by facts, and that it is of such nature as to be susceptible of verification; for without such caution the hypothesis is a hindrance rather than an aid. The words "pretubercular stage of phthisis" may not only have some meaning, but may express a chronological fact, as well as, upon certain premises, a logical necessity; but the existence of such a stage as a clinical fact is yet unproven; and the introduction of such phrase into symptomatology is, I think, precipitate and undesirable—not because it is a hypothesis, but because its verification is, from the very nature of the supposition, impossible. Again: the words "suppressed gout" may, in years that are past, have had relatively some more definite meaning than they can have now, inasmuch as our forefathers did not know what they meant by the very gout that was suppressed; but in the present day we have heard, and may still hear, trains of symptoms that have lasted for a lifetime set down most ruthlessly, and without the shadow of evidence, to this mysterious thing—"suppressed gout!" Do I mean to say that gout cannot be suppressed? By no means; such assertion would be as absurd as to say that there are no "mute, inglorious Miltons" in our country churchyards. The one statement would be unwarranted by history, and in the teeth of all the poetry of agricultural life; the other would be unwarranted by pathology, and in direct opposition to the authority of most venerable practitioners. But what I do assert is this, that any one who uses such term should be liable to some pains or penalties if he does so as a guess; that he should be compelled at once to verify his hypothesis, or to devour his own words; and that if, in these days of pathological chemistry, such verification is impossible, the term should be cast away from our nosology as an illegitimate hypothesis.

Modern pathology presents a profusion of these illegitimate hypotheses, and they are obstacles in the way of its advance. Sometimes they are created for the purpose of

concealing ignorance, and sometimes for the purpose of securing fictitious and temporary fame. Showy as they are unsubstantial, they often appear fashioned as the so-called "balloons" of the "circus" may be, to be held up for the purpose of destruction; glittering, gilt-edged baubles, through which critics, horsed on their editorial "we," may leap unhurt, to the admiration and astonishment of those who know not which to admire the more, the flimsy speculation, or the equestrian's agility in its demolition; but which, to all who are earnest in the search for truth, and who see that it is not to be found by an everlasting iteration of this circle of creation and destruction, is a lamentable waste of time, energy, and skill, and a still more grievous fallacy, because often mistaken for real progress.

Again, mere matters of *opinion* are not to be confounded with scientific facts. Such confusion often occurs from either carelessness or ignorance. The individual committing such mistake does not substitute a fiction of his own imagination, a deliberate creation of his own fancy, for a fact; neither does he produce anything which is worthy of the name of hypothesis. His guess is neither ingenious nor philosophic; it may be right, or it may be wrong; but if either, it is so by chance, for it is a hastily or ignorantly formed opinion passed off for fact.

It has been said that "one fact well observed is worth a cartload of opinions;" but opinions, whether single or in cartloads, are of some value, and that in proportion to the information and ability of those who frame and utter them. The opinion of an individual, either absolutely ignorant of auscultation, or totally deaf of both ears, would be of but little value in most cases of heart-disease; but minor degrees of these incapacities appear at times to confer upon the dicta of some people an extraordinary amount of importance.

But, whether particular opinions are of great or of little moment, it is of the utmost consequence that they should be distinctly separated from scientific facts. For example, it is the opinion in some quarters that an intimate connection subsists between disease of the supra-renal capsules and bronzing of the skin; it is the opinion in other quarters, that the relation is an accidental one. The facts are, that the two elements are often associated, often not; and it has yet to be shown that when they co-exist there is not some other element essential to their relation. It is the opinion of some that bronzing of the skin and emaciation of the body

depend upon disintegration of certain nervous ganglia in the neighbourhood of the supra-renal capsules, and that it is only when disease of the latter extends to these nervous elements that the cutaneous and nutritive effects are observed; and hence, that the supra-renal capsules have nothing to do with either the chromatogenous function of the skin or the nutrition of the body. These are opinions, and while the unquestioned facts are still *sub judice* in regard of their explanation, what is to be avoided is the expression and use of these opinions as facts.

It is because this kind of distinction has not been maintained that so much evil has arisen in many of those judicial investigations, where not only medical facts but scientific opinions form part of the evidence. Whether the case to be investigated be one of suspected lunacy or poisoning, we almost invariably have the spectacle of scientific witnesses called up in violent hostility to each other; and the report of this warfare is followed by an outcry from the public and the press, that, as the doctors disagree so widely their evidence, is of little value. This, gentlemen, is an opinion; it is not the fact; but there are some grounds for its existence in the public mind. A jury has to form its conclusion from two classes of scientific evidence, viz., facts and opinions; it has to hear a description of what actually occurred—of what are the symptoms, for example, of natural disease, and of what are the symptoms of poisoning. In order to decide upon such evidence, the jury is supposed to be educated up to a sufficient scientific eminence to form a rather difficult diagnosis, and to settle a question with regard to which the most careful and the best-informed of our profession might feel doubtful! But the evidence does not stop at this point; scientific witnesses are asked their opinions upon, or interpretations of, the facts they have stated. These opinions are given; and generally between those of the witnesses on different sides, and sometimes between those upon the same side, there is as great a divergence as is possible within the bounds of common sense; nay, sometimes even these limits are overleaped! The difficulty of the juryman's task is now increased tenfold: before, he had to judge of scientific facts, now he has to judge of scientific men; he has to weigh in the balances, not only their statements, but their opinions—not only their veracity, but their ability; and he has to form his own conclusion,—and one which must be at variance with a large amount of scientific evidence and opinion from one side or the other—by a judgment amongst scientific men, by the

differences in their mode of stating facts, and by a discrimination among the facts based upon the scientific credibility of the men.

Now, I readily believe that some plain-thinking, common-sensed artisan is quite competent to perform the task of at once recognising that some opinions given in evidence have no value whatever; that the man who has framed them has done so in carelessness or ignorance, or both: and that they are self-contradictory, or at variance with facts which he himself admits. On the other hand, the high value of some opinions may be immediately perceived and correctly estimated. But, when neither of these extremes is present, or when they are so combined or opposed as to neutralise each other, the position of the juryman is one of intense difficulty and complication. This it is by the very nature of the case; but its difficulty is increased to a most unnecessary degree by a want of distinct separation between the two kinds of scientific evidence, viz., the facts and opinions. This distinction is frequently neglected in all parts of the trial; scientific witnesses, counsel on both sides, and judge himself are alike guilty.

To exclude either element of evidence is, I think, impossible; to banish the facts would be to deprive the jury of all standard for the valuation of opinion; to exclude the opinions would be to take away all hints towards an interpretation of the facts; while, on the other hand, to allow both elements of evidence, is to give opportunity for a curious and somewhat unsatisfactory mental process, viz., the balance which an "inexpert" juryman has to strike between his own scientific opinion and that of the highest scientific authority in the country; and it furnishes, moreover, the opportunity for almost endless blundering between these two things, which are essentially distinct, viz., facts and opinions.

Again, do not treat and use as facts mere *half-facts*, or other fractional parts of them. Many statements may be true, but imperfect; and they become not only useless, but injurious, when their relation to the whole truth is disregarded.

There are two different ways in which you may know but half the facts of a question; you may be acquainted with the evidence on one side, and be entirely ignorant of that upon the other, or you may know only half of the evidence on both sides. He who, as a matter of habit, knows only the one side of a question, must have his mind diseased or crippled by prejudice; and if he act upon such information, his action will be that of the bigot. On the other hand, he who

habitually rests satisfied with half knowledge, on both, or all sides of a question, must, to begin with, care little for the point at issue; and if he have any interest therein, it is of some secondary or trifling moment.

If the medical student rests contented with the teaching of one book, or of one school, and cares not to know the views contained in or entertained by another, he exhibits a prejudice which may seriously injure him in after life; but, if he is contented with acquiring only such a smattering of information on all sides as to have no opinion of his own, and no valuable knowledge on any one disputed question, it will very probably be found that he cares little for truth for its own sake; and further, that his scraps of information bear a striking relation to the pet views of reputedly "crusty examiners," and that his magnanimous aim in their collection is, to "get through the Hall."

Avoiding the sources of fallacy which have been enumerated, keeping steadily in mind the broad distinction between facts and fictions or fancies—those scientific "delusions" which demonstrate the scientific insanity of a man;—further, maintaining hypothesis in its proper place, and perceiving the difference between that which is legitimate and that which is not; distinguishing between opinions and facts, and between parts of the truth and the whole, you may learn much of the facts of life; and having learned these well, you advance by their aid, as by the alphabet of a new language, to the study of VITAL LAWS.

The term "*law*" is one with which we are very familiar, but its applications are most various. It has been used to denote those ten great utterances of man's moral and social responsibility, which have come down to us, with sublime mystery and authority, from a far distant antiquity; and the same word 'law' has dignified the last percentage announced by the Statistical Society. Those bare generalisations of geometrical facts, revealed by the motion of the planets about the sun, and of the satellites around their primaries, have been termed, from the period of their institution to our own day, "Kepler's laws;" while, on the other hand, the proscriptions and restrictions of the last act of Parliament form the latest addition to the "laws of our Country." The general statement of a progression in the order of development, from the general to the special, forms one of the "embryologic laws of Von Bär." The "positive philosopher" states, that the three stages through which scientific study passes are

"laws of the progress of humanity;" his critic may state that they are, or are not "laws of the human mind." And again, the conditions upon which individuals are admitted into this excellent Institution, are termed the "Laws of the Westminster Hospital."

Disregarding mere differences of detail in the objects, to the phenomena or relations of which the word may be applied, it is very commonly said that there is one great distinction to be observed between the several uses of the term which I have just enumerated, and that by means of this distinction we may form two categories of laws. In the one, it is said, is prominently conveyed the idea of enactment in accordance with the will of an individual or institution, plus a coercive power employed by that will, in order to enforce obedience or to inflict pains and penalties upon its transgressor. In such general idea, the Decalogue, Acts of Parliament, and the laws of this Hospital agree. In the other category there is no bringing forward of this idea; all that the law contains is, it is affirmed, a statement of the conditions under which certain phenomena occur. Apparently, there has been some endeavour to get rid of the idea of will or coercion from the statement of such laws; and in such negation are practically allied to one another, the laws of Kepler, those of the Statistical Society, of Von Bär, and Auguste Comte.

This distinction is not one in regard merely of the supposed origin of the laws referred to; the ideas of will and coercion are essential to the very conception of law in the one class, and not only inessential, but, in the judgment of many, absolutely foreign to the idea of law in the other class. This difference, however, is, in my belief, apparent, but not real; on the contrary, that which is common to all the uses of the word, and which justifies the employment of the term "law" to any of them, is, in my judgment, the presence of those very ideas which it seems now to be the wish of some to exclude. Whensoever the word law is used, there is in it the suggestion or conception of that which is other than the phenomena, and greater than the mere fact of the order in which they occur. There is inherent in the idea of law the suggestion of a plan; and it is only to such generalisations of sequence as contain this suggestion that the word is correctly applied.

What, then, is the difference—for one plainly exists—between the two groups of laws that I have quoted? It is this:

that, in the one group there is merely a command,—by a visible or admitted lawgiver,—which may be disobeyed, the penalties resulting from transgression being the fulfilment of the law; in the other, there is no expressed command;—the lawgiver, though unseen, being necessarily assumed,—but there is no place for disobedience. To the one kind of law man's will may be opposed; he can obey, or he can resist: to the other kind the will of man may be opposed or not, but he has no power of resistance; obedience is a matter of choice in the one case—in the other, no choice is allowed. Thus, a man may violate every moral principle—he may break every social tie—he may transgress his country's laws, but he cannot escape from or resist the law of gravitation: he will sink in the water if he makes no effort to escape; he cannot resist the vital law of respiration; he must breathe air, or die. Moral and social enactments may be so inextricably interwoven with laws, of which they form but a part, that their violation involves, and that of necessity, ulterior evil; but physical laws cannot be violated; man cannot choose between obedience and penalty. The association of suffering with sin is, in my belief, as real as that between air-breathing and human life; but the relation, in the former case, is matter of inference from other data than mere experience; in the latter, it is matter of actual observation. If, then, not only the idea, but the fact of coercion, is more evident in the one group of laws than in the other, it is in that very group from which the modern philosopher would exclude it. So familiar has man become with the transgression of moral and social law—with the determination to run the risk of penalty rather than simply to obey—that, because he sees physical laws maintained inviolate, he loses sight of their common nature, begins to think of them differently, and tries to denote them by different words. And surely, if the difference that is supposed to exist does pertain between them, some difference of terminology is needed.

An ingenious and learned writer has proposed, in order to rid our minds of the "intrusive and delusive meanings" with which the word law is "clogged," that it should be omitted altogether from our scientific glossary; and he substitutes in its place—though, it must be allowed, with some misgiving—the term "method," or "path." The so-called laws of nature are, in his improved phraseology, "the *paths* along which the activities of nature travel to results (phenomena.)"

But, let me ask, what is the meaning of a path? Surely there is, in the idea of a "*path*," something besides, other

than, and anterior to, the "activities" which travel along that path! If it mean anything, it must mean a distinct and necessary line or direction, along which this travelling must be accomplished; a something, outside of which the "activities of nature" cannot "travel to results!" In such idea, that of coercion is as distinctly present as it is in the term law. The philosopher of to-day has taken the "strait-waistcoat" from off nature, but he has not set her free—he has placed her in a "padded room!" Surely, such treatment is not a true example of a system of "non-restraint!" The hunter, in his loose box, has more liberty than when tethered to his stall, but he is not free. Restraint is still present in the philosophy, the asylum, or the stables of the nineteenth century; although it may be in a more pleasant form, than in the words, the cords, or stalls of a bygone day.

It may be said that the supposition of will or coercion in the conception of law is an attempt to explain how the laws were framed, and whence they came, not what they are; and, that in making such supposition, we at once pass beyond the limits of scientific inquiry, and enter the domain of either theology or metaphysics. If an attempt to explain what is meant by, and included in, the terms vital function, secretion, respiration, and the like, is a departure from natural science, or from the science of physiology, and is an entrance into the region of etymology, chemistry, or mechanics, then the endeavour to say what is meant by the term "vital law," is a departure from the science of physiology; but it is so only in a similar manner, and to an equal degree. You cannot advance one step in the science of life unless you know the meaning of the terms you employ, *i. e.*, unless you are already acquainted, on the one hand, with so much of chemistry, physics, and syntax as to enable you to comprehend and refer to something more general than the vital functions you are studying, the elements and processes before you; neither can you advance towards the appreciation of a scientific law, unless, on the other hand, you have some notion of the meaning of that term, and unless your idea of law is of more general extent than that of the particular example you are studying.

We must, then, endeavour to know what we mean by "law;" and, if we would grasp that meaning, we must seek to discover it by other means than mere verbal translations of the term and its idea. If by "law," it is said, is intended a generalisation of the "conditions" under which certain

phenomena occur, let me ask what is meant by "conditions?" Do we, by such phraseology, get rid of the ideas of coercion or of will? By no means; these are still present, only under another name. The word implies something more than the facts and their relation—something greater than, because the source of, their order of occurrence. Mere sequence is expressed by the words "do" or "did;" law, by the terms "shall" or "must."

For these reasons, then, I would insist upon it that—no matter with what eagerness some may endeavour to find in natural law nothing but a generalisation of the conditions under which phenomena occur—there is invariably and inevitably some supposition besides and beyond the facts, which supposition does, in reality, give meaning to their words and thoughts. If there is no immediate recognition of God, there is some mystical conception of Nature, or some personification of Order; and to the will, or action of one or the other of these the law is referred. It is not that, by law, we have merely expressed what is, or the mode in which what is appears to us, but the method in which God, Nature, or Order operates. Remove, as completely as you can, all metaphysical entities from the idea of law, and there is yet underlying it such a supposition of order and uniformity, of independence and unchangingness, which are attributes of a something that directs, pervades, and embraces all, that the most so-called "positive philosopher," in his very endeavours to escape, only binds himself hand and foot more closely by the ideas of coercion and of will.

Inasmuch as in the possibility to appreciate facts there is the proof that a relation subsists between our minds and external nature, so in the capacity to advance to the idea of law there is witness to a still deeper harmony. "The laws of nature (as it has been well said) are at the same time laws of reason; . . . the geometry and mechanics which are practically applied in the works of nature, are precisely the same as those which reason perceives as necessary truth; so that in scientific observation we are really viewing the laws of our own reason operating around us in the objective world." But the laws of our own reason, and the laws of nature, in thus testifying to their common origin, bear witness to that which is greater than nature and higher than man; and whatever may be the application of the word, there is, I believe, always, as part of the idea of law, the conception of a source far higher than itself—the suggestion of a purpose

and a plan, of which the facts and laws are but the fragments of a revelation.

This is true of every form of law. Those ten great utterances of man's moral and social responsibility, by their nature-like simplicity, depth, extent, and universal application. proclaim their source to be that from which nature itself arose: those "great commandments," the conception of which, as kept inviolate, is that of a perfected humanity; those expressions of what man should be, so lofty, and so far above his actual life, and yet so consonant with all the ideas, so far as he can frame them, of what his life was meant to be, are such that he cannot regard them as laws of his own making, but as having their source in that which is far higher than himself, their origin in Him, who is not only the Creator of the material world around him, and of his own material body, but the Author of his intellectual power, and of that which is the highest element in his being, viz., his own moral nature. The laws of a Country or an Institution have authority and stability, because in conformity with responsibilities wider and laws profounder than those of the Country or the Institution from which they issue and to which they are applied. The laws of Nature,—whether seen in the tiny cell of some microscopic monad, in the mimic whirlpool that a rotifer may set up, in a drop of water, itself almost invisible to the naked eye; or seen in the perturbation of some planet's satellites, so distant from our world that thought is baffled with the attempt to imagine its extent, so vast that the mind trembles and sinks under the effort to conceive its grandeur,—wherever these laws are traced, there is, in them, the witness to that which is beneath, above, and around them all; witness to that which is still deeper than our most microscopic search, still vaster than our most extended gaze; to that which transcends all our knowledge, but which is yet the light of all our seeing, and the ground of all we know,—

> "Whose dwelling is the light of setting suns,
> And the round ocean, and the living air,
> And the blue sky, and in the mind of man:
> A motion and a spirit, that impels
> All thinking things, all objects of all thought,
> And rolls through all things."

And, as we trace the harmony between our own thoughts and the facts of the objective world, we are moved, as the soul of Kepler was when he saw the planets obeying the

order which his reason had marked out for them, to exclaim, "O God, I think Thy thoughts after Thee!" In thus viewing law as the expression of the Divine thought, the mode in which God has been pleased to act, we arrive at its truest and deepest meaning, and find in it, not only the key to unlock nature's profoundest mysteries, but the key note to which our efforts must be attuned if we would understand her mighty harmony, and take our part therein.

In order to arrive at the discovery of law, there are certain fallacies to be avoided. Do not mistake for law mere sequence of events, although apparently invariable; do not confound therewith either accidental coincidence, mere speculation, or numerical hints of the direction in which law may lie.

There is no need that much more should be said on the confusion of mere *sequence* with law. Illustrations of a "*post hoc, ergo propter hoc*" mode of reasoning are too common to require further remark than that the real test of such fallacy is the discovery of a third element, previously unconsidered. Because when the sun arrives due south the chimes of a neighbouring tower perform a certain tune, and a large bell tolls twelve times, there is not to be inferred any necessary connection between that position of the sun and the particular tune that falls upon the ear. Certain clock-makers and bell-makers have come in between the sun and the sounds; and they could, if they chose, so arrange their apparatus that the particular sounds in question should occur at some other time, and that when the sun arrives due south the clock should strike one, or a quarter past. If, instead of coming to such unexpected grief, "Big Ben" and his satellites should have still kept true for centuries to come, until the well-known "traveller from New Zealand" commenced his sketch from a "broken arch of London Bridge," such long-tried and invariable association would not demonstrate the existence of a law to the effect that a certain position of the sun was necessarily associated with a certain chiming of those bells. Yet such laws as these, and many based upon evidence not one hundredth-part as good, are current in the science and pseudo-science of the day. We frequently leave out of the account our clock-makers and bell-makers, our men who wind up and regulate the works; and we fancy that the fore-established artifices, such as division into certain and particular hours, are parts of nature's plan. Absolutely invariable sequence is the strongest evidence you can obtain of the existence of some

law; but a mere expression of that sequence is not a statement of the law: nor is the latter statement a possibility, until all the elements of the sequence are understood.

The "traveller from New Zealand," if he came before his appointed time and took his stand within this hospital, might see one-half of the out-patients entering one room, and the other half another room; he might discover that the first group suffered from complaints of external organs, and that the second were the victims of internal disease; he might go further, and (waiving occasional mistakes or transgressions, and their penalties) might find that the individual by whom they were seen in one room was a surgeon, and in the other a physician;—he might generalise the conditions under which this sequence of phenomena occurred; but such generalisation would not be a "law." He might, from its regularity, infer the existence of a law; but if he did so, he would at once refer it to something other than the mere fact of the particular order of events I have described That which he would infer to be the law, and that which is alone worthy of that name, is the expression of the mode in which this Institution has resolved to act.

Accidental coincidence, a fortiori, is not law, although sometimes it is more striking than invariable order. Moreover, it does not afford evidence that any law of the kind supposed is in existence. Yet the annals of science contain many illustrations of the one having been confounded with the other. Eclipses have coincided with abortions, comets with pestilences, and the changes of the moon with epileptic fits; but it has yet to be shown that the relation between them is deeper than that of mere coincidence.

Not detaining you with any endeavour to show that *speculation* is not law, I pass on to the other fallacy, viz., that of confounding *statistical results* therewith. The "numerical method" of estimating facts has been, and is, of great utility; it has pointed out the direction in which truth lies; but the statement of a percentage is not the utterance of a natural law. At the best, we know but fractions of the truth; the numerical method enables us to state what those fractions are: as a method of inquiry, it is invaluable; but, if its results are regarded as final, it is useless, or worse than useless.

If in one hundred cases of a certain disease, say "continued fever," there are sixty which present a particular form of eruption, twenty which do not, and twenty which

exhibit another, a statement of percentage to that effect may be valuable, proving either that the name was wrong, for it included too much, or that the eruptions alluded to were neither of them essential to the idea of fever. If, however, inquiry stops at the enumeration of these facts, it is of little service. If the statement of these facts, and the proportion in which they occur, is regarded as a law of "continued fever," and the conclusion is that the name is correctly applied and that the facts vary, then the numerical method is worse than useless; but if the mind advances, and eventually demonstrates by this method that there are essential differences between the cases referred to, then the method is of great utility, in eliminating error, and pointing out the direction in which an important truth previously lay concealed. But the truth, or the law, is not expressed by percentage. The two or three forms of fever are absolutely distinct; and the statistical statement was but the sign-post, showing whither the inquiry should be carried in order to prove their distinctness.

Again: the difference between a law of nature and a so-called statistical law is further seen in this, that the former is true at all magnitudes, times, and distances, whereas the latter is true only at such magnitude, time, or distance, as shall be sufficient to lose or hide the individual in the multitude. For example: the law of gravitation is true of two grains of sand, or of two planets in the solar system; the law of atomic proportion is such that the relative quantities of calcium, oxygen, and carbon are the same in the faintest line of a chalk drawing and in the mountain range; the process of assimilation is the same in the infusorial cell and in the brain of man; the seedling oak contains the same elements as the giant trees of the forest from which the acorn fell. But, try a so-called statistical law by such test, and it is at once proved unworthy of the name. If twenty thousand out-patients are relieved annually by this Hospital, and the number varies but little from year to year, it is not equal, but varies widely, from day to day. It is affected by the weather as much as by disease! The annual average mortality of a particular ward will not bear such division that it can be applied as a law to the individuals now lying therein. The law, so-called, that shows the chance of a railway accident to be an almost inappreciable fraction in the particular journey that you are about to take, affords little consolation to you if you are jammed up in a tunnel; nor will it—although you

ought to lose only a fraction of your finger-nail as the result of a collision—save you from losing your head, if, as unfortunately happens so often, the appropriate conditions are provided.

If the statement is—and it may be quite true of the multitude—that of ten thousand individuals, one hundred must suffer something, be it accident, disease, or death, neither this statement, nor the proportion, can be true of a less number than one hundred, for the same proportion, carried out, would with a less number affect only the fraction of an individual. If the statement is that of one hundred cases of a particular disease, such a symptom should be present in twenty-five, that law applied to four individuals is, that the symptom should occur in one; but, in a given individual, only a quarter of the symptom should occur. We say, in such instance, that the "chances" are three to one against its appearance; but, if the moment these statements have to be applied to the individual we have to change our phrascology, and speak of "*chance*," what has become of our so-called law?

To illustrate this principle still further, let me employ some statistics furnished by the Royal Humane Society in the *Times* of September 5. From these it appears that during thirteen years 1,015,853 persons skated in the Regent's Park, and that during those thirteen years, 274 individuals met with accidents of different kinds: the general proportion, or numerical law, therefore, is, that 1 in 3,707 individuals should be victimised during this amusement. Now, this proportion is so near the truth that it agrees, almost precisely with that furnished by two periods, each of six years' experience, in the same locality. We divide the statistical law by about two, and find that of 498,283 skaters, 129 met with accident, the proportion being 1 in 3,862. Again: the numbers injured in periods of three years are very nearly identical, being 1 in 3,909, and 1 in 3,836; or, omitting the two last figures for the purpose of easier recollection, the proportions are—for thirteen years, 1 in 3,700; for six, 1 in 3,800; in the former three years, 1 in 3,900; in the latter, 1 in 3,800.

But, if we divide still further, the statistical result becomes variable, and the so-called law defunct; for in particular years—to say nothing of days—the proportions vary from 1 in 1,237 to 1 in 37,191. From this it is evident that a statistical result approximates the truth in proportion to the distance

to which it is carried from the particular fact with regard to which the statement is made. So long as the number of skaters ranges between tens of thousands and hundreds of thousands, the results are unequal, but when the numbers are between hundreds of thousands and millions, the results become nearly uniform. It is, then, interesting to observe, that although in consecutive years the proportions are so various as to range from 1 in 1,200 to 1 in 37,000, yet that in triennial periods these very inequalities are reduced to an inconsiderable amount. Hence, though the variations are great, these variations have limits. Herein is the treachery, and also the value, of statistics. It is not until the numbers accumulated are so large as to include all sources of variation that the result is true to nature, and therefore uniform: then it is so, but it loses its direct utility and applicability for the individual; and that which is a law for the multitude becomes, so far as its application is concerned, a *chance* for the unit or the hundred.

It seems that in periods of three years, and in about a quarter of a million skaters, every variety of folly, hardihood, and clumsiness on the part of the skaters; every variety of blunder, carelessness, and officiousness on the part of the icemen; and every treachery of ice, in its early brittleness and commencing thaw, must be included; the limits of variation are reached, and the results are equal:—1 in 3,751 is (not killed, but) visited with accident. But if, when this next winter comes, two of you start for the Regent's Park—not to any other park, for the proportions differ—persuaded that the chances are 3,751 to 1 that you will not go through: and if 3,749 individuals visit the same park, with the same persuasion, and one which is warranted by the facts, and applies equally to all, what becomes of the statistical law when one of you two receives a ducking? It amounts to this, that the chances were not equal for the two, but that it was from utter ignorance you thought they were: they differed as widely as they possibly could; and therefore, that the statistical statement is not worthy of the name of law. Further: if the statistical law could be accurately applied to the individual, so that his particular chances could be known beforehand, the practical utility of the law of probabilities would be annihilated. A moment's reflection upon the principle of life insurance will at once prove this. If, so far as human intelligence extends, it were not a mere chance that A and B, whose ages are the same, should have the

same expectancy of life; but if, on the contrary, it could be known that A would live thirty years, and B would die in three, the former would not now insure his life, and the office would refuse the latter. It is because statistical results are true only of the multitude, and uniform only in regard of large numbers, that the individual can derive advantage from them. It is because the so-called law for the many becomes a chance for the unit, that the law of probabilities has a practical utility.

Perhaps the most unwarrantable conclusion which has been drawn from the employment of the statistical method is to the effect, that because events occur in such order that their numerical frequency may be calculated beforehand, therefore neither God nor man, neither Divine Providence nor human will, are operative in the world. Because there is a certain average number of suicides, for example, per annum, individual choice and general Providence have had nothing to do with the matter.

To arrive at such a principle as this, at the conclusion of a survey of the history of civilisation, would be, in my judgment, to conclude in opposition to both historical evidence and true reason; but to start with such proposition, and to employ it as a method of investigation, is one of the most extreme examples with which I am acquainted of a "*petitio principii*."

Allow, for a moment, that the principle is correct—viz., that the existence of uniform averages would exclude the idea of will, either Divine or human—the past history of the world, and its present history, are such as to show that these averages have not always existed—do not now exist. The ordinary course of events has been suspended, or superseded, by the extraordinary or the supernatural, and the true basis of all nature has been revealed in such suspensions. But, further than to enter protest against such interpretation of history as this, is not my object now: it is to show that, even if the averages exist, they do not warrant a belief in the non-existence of will, either Divine or human. The proposition is this, that, because out of 10,000 individuals, say 100, in the course of twelve months, will do such a particular thing, therefore there was no will in the individuals who did that thing, or in those who did not. The argument in such proposition is erroneous; it is a conclusion with regard to the individual, from observation on the mass or multitude; and I have already shown that until the individual has been lost sight

of in the multitude—*i. e.*, until the accumulation is so great that it includes every possibility of action and every variety of condition, the statistical result is demonstrably untrue: the statistical law loses all force, and has to be termed a chance, when applied to the individual, *i. e.*, to the very thing or being supposed to exert the will.

Beyond the fact of almost universal human assent to its existence, I know of nothing which proves more cogently the "freedom" of human will than does the application of the numerical method to the results of human action. Such application shows the presence of disturbing causes, quite out of the range of explanation by the mere crossing or combination of natural laws. No matter how complicated is the object to be examined, so long as it is under the dominion of physical laws alone, the net result of its action is soon arrived at, and may be stated in comparatively low ranges of number; but, immediately that you have for examination that to which moral and social laws apply, you have to derive your results from tens and hundreds of thousands. Two healthy individuals of equal age, weight, and height, affect the physical atmosphere of this room in the same manner and to the same degree. Here you may soon calculate all the possibilities of variation, and state them in numerical terms; but between two individuals, side by side, how vast is the difference, when regarded in their capacity to affect the moral and social atmosphere! Alike in the laws they cannot transgress, the net result of their lives physically—so far, that is, as mere physical elements have been employed—is almost identical; but the difference between them is almost infinite when they are viewed in relation to the laws they can disobey. The fact that human actions, as studied in large multitudes, result in tolerably uniform averages, is evidence that man is under the dominion of some laws which cannot be broken. The fact that such large multitudes are required in order to furnish uniform results, is evidence that man has a disturbing power; that he is able to set himself in opposition to certain other laws, interwoven with these: choosing to disobey their expressed commands, and to fulfil the law, by suffering the penalty attached to disobedience.

The possibility of calculating the frequency of some human actions shows that the power and the variations of the human will are, in regard of action, limited, and that by physical conditions. Man cannot will himself out of life, for example: he must, in order to commit suicide, throw himself under the

dominion and operation of a physical law, which he cannot break, but which can destroy him: he must choose poison, or gunpowder, or drowning, or what not. Moreover, the implements from which he shall make his selection are not innumerable; poisons and possibilities of destruction have their limits; and thus the resulting actions of this hundred thousand individuals, and of that, may be nearly numerically identical in regard of suicide; but the very largeness of these numbers is evidence of the presence of disturbing causes, which, in extent and variety of influence, will bear no comparison to any physical agencies.

It is by removing yourself to a great distance from the actual facts that the numerical statement of these facts exhibits uniformity. To the naked eye the moon's outline is an even curve—there are no inequalities, no changes in its form; but lessen the distance, or enlarge your power of vision, and then mountain ranges, valleys, and extinct volcanoes, break the line. And so it is with man. Go far enough from the individual soul; lose his personality in the thousand or ten thousand that surround him,—and the net result of this ten thousand, and of that, may be the same. But there are in him individual features—heights of aspiration, and depths of despair, angry passions, and Divine helps;—and these, seen in the unit, but lost in the many, are the real moving forces, the determining causes of all his action.

In thus surveying the "aim" which, as students of the medical profession, is placed before you, I have shown that the "subject-matter" of your study, LIFE, is two-fold—mind and body; material and immaterial; temporary, tangible, and seen; unseen, intangible, and eternal: and further, that the gulf between these two is such that no science has yet bridged it over, and no speculation, launched from either shore, has done other than find shipwreck in the abyss. Yet, that each is true, is near to us, and within us all;—that our life-study will be but one-sided unless it embraces both, and imperfect unless it finds in each the hints and proofs that they come from the same source, obey the same laws, and work out but different parts of the same great purpose.

I have urged upon you, that although you know not, and may never know, the essence of life, yet that you may learn 'FACTS' about it; that the object of your study, then, is truth, for the acquisition of which all your care and all your industry are required; that you must arrive at truth by

avoiding the mistakes of confounding facts with either fancies, hypotheses, opinions, or half-truths; and that, by using these facts and this knowledge as your alphabet, you advance to the study of LAW; that in it you find the suggestion of something beyond the mere facts themselves—the conception and revelation of a higher power, and of a plan which gives harmony and meaning to the diverse phenomena you witness; and you trace a purpose which the processes of nature are gradually, but surely, developing. I have urged upon you the differences, to be constantly remembered, between law and generalisation of sequence or coincidence, and between natural law and numerical results of law.

Thus, your aim is to learn the truths or facts of a two-fold life, and to find in its deepest laws the revelation of that which is greater than itself. Life, life of two kinds. Truths about it, not untruths; and the reference of it all to a higher source.

For the reconsideration of your aim no time is more appropriate than the commencement of a new period of your work; but if you advance in this work—and in proportion to the rate of your progress—will it become every year more important that you should take this review. For at every step in our upward progress towards some height of science, the horizon widens, and with new wonders and new beauties there are new elements of confusion, and we may be baffled by the very largeness of our view. Pressing onward, we see that beyond some clear and hitherto limiting line there are depths into which our vision cannot penetrate, further than to see that they contain, in lavish profusion, wonders and beauties equal to those around us. The summit of yesterday's is but the starting-point of to-day's exertion; and as the circle enlarges, it is at its circumference that the increase lies The rate of increase is accelerated, and the objects that surround us seem infinite and overwhelming, by their largeness, their number, and the marvellous intricacy of their combination. Life is too short to see them all. We must choose our path: we must leave much unseen, still more unthought of, and an infinitude unknown. For as we ascend still higher, we catch glimpses of the far-off sea—the infinite, unfathomable, Unknown—that mighty ocean of unknown truth that lies beyond, around, and beneath all our knowings, which confounds and wrecks every venturous barque that launches forth upon its mighty waves—that vast waste of water, sometimes dreary and bodeful as the grave, but

sometimes bright and beaming as the sky; sometimes blazing with the sunlight, and again silent and sorrowful as the pale light of stars. It is only when we have attained, and can look out from some height of science, that we begin to see and know how vast is the unknown; and how, compared with it, dwindles into nothingness the little that our powers have yet taken in. The mightiest of philosophers felt that he was but as a child gathering pebbles upon the shore of that boundless sea! But we have to come down from these heights to every-day life and work: to know how to advance, and rejoice in the vision that each day's scientific progress yields; but, at the same time, to find among the wide fields of scientific fact those principles which shall guide us in the prosecution of our real life-work—the alleviation of the sorrows and sufferings of humanity. These principles you must learn by means besides those of chemical experiment and dissection: you must learn them in the hearts and lives of your fellow-men. You must come to know the meaning of the infant's look—to understand the tone and teaching of its cry. You must appreciate the patience and long-suffering of woman, and the hardihood and rough exterior of man: you must look, and see beneath these, if you would measure the true degree of their affliction. You must learn to feel with and understand the feebleness of age, and gain all that is required for your ministration to its wants, from those hints that come to you through the failing powers and closing avenues of a soul that has for a long while battled with a world too rough. True sympathy will reveal much to you that science cannot see. Wherever there is life, there is your field of study—wherever there is suffering, there is your field of work; and truly to relieve its burden your spirit must be that of Him, whose life was the perfect life—whose presence and word brought peace and health, and into whose work in this world it is our highest aim and highest dignity to enter.

But in such exercise—in loving your neighbour as yourself—you do but fulfil the duty incumbent on you as a man. As a medical practitioner, there is for you other work, and work which is peculiar to our profession. The medical practitioner has to educate—by converse, by life, and practice—those whose education is less specially scientific than his own. He is the exponent of the real method of arriving at scientific truth; and it is his part to show that in action he can lose sight of minor differences in scientific creed, and can combine, with those who differ from him, to employ common

truths for the advantage of his fellow-men. The man of science is the apostle of the true spirit of inquiry—he is the humble investigator of nature's plan. Submitting himself, with confidence and entireness, to the truth as taught by nature, he is the abject slave of neither institution, party, clique, nor self: he utters his protest alike against servile dependence upon authority, and against the rabid independence and the folly which would rely entirely upon itself. He sees that, to ignore the teaching of the past, is to cut off from himself the rich entail of scientific treasure, and to impoverish the present—that to yield no honour to institutions is to oppose the common sense of humanity, and to do away with all standard in regard of truth; but he also sees that, to receive the teaching of the past, without question and without addition, is to introduce retrogression or stagnation into science, and to render the future even worse than the present day; and that, to despise individual exertion and conviction, is to rob institutions of that which supplies the conditions of their existence, and gives authority to their words. If there were nothing to which we could appeal, it would matter little which guide we might choose to follow; mere temperament would decide whether we should rest contented with the dogmata of the past, or should follow the speculations of the present; and in either case, our science would be but an empty dream—our action would be but a delusion and a sham—our profession a mere guild, maintaining itself upon the superstitious fears of those not quite so knowing as itself. But the Court of Appeal exists, and is ever open: nature is open before you, as it was before the fathers of our science; and to it, with as much confidence as they, you can apply for instruction. Common sense, educated by both the wisdom and the follies of the past, and common honesty, universally applied, will enable you to ask questions and receive their answer; but you need more than these—you need a simple, child-like trust, to free you from preconceived opinion on the one hand, and from self-confidence on the other, and to make you as open to learn as nature is to teach her lessons.

And finally, amidst the petty jealousies, the party bickerings, the presumptuous ignorances, the pharisaic prides, and mock humilities that do so fetter the limbs of men, and bind them up into such little bundles of humanity, that they can do nothing in this world but harm, unless they may do it precisely in the way their own fancy, their great grandfather's fancy, or their party's fancy dictates;—against all these littlenesses, that make us almost ashamed of the race to which we belong—it is the part of the man of science to utter his strongest protest, the protest of example. To show that though he and his brother may chance to differ about the nature of ozone, they can yet clasp hands heartily and join in works of science and of charity. To show that his "aim" is such, that even if the method his brother adopts to "learn the facts and laws of life" does differ from his own, yet that he has so learned—from a higher source—the duties incumbent on him as "the true man of science, of art, and charity," that he can join that brother in "utilising his knowledge in every way, and to the highest degree for his fellow men."

LONDON: PRINTED BY S. GEOGHEGAN, 6, BOUVERIE STREET, FLEET STREET.

E. A. Parkes M.D.
with the author's kind regards.

[*From the* Proceedings *of the* Royal Society *for February* 17, 1859.]

"On the Influence of White Light, of the different Coloured Rays and of Darkness, on the Development, Growth, and Nutrition of Animals." By Horace Dobell, M.D., Licentiate of the Royal College of Physicians, &c. &c. Communicated by James Paget, Esq. Received January 10, 1859.

(Abstract.)

In this communication the author laid before the Society the particulars of a series of experiments, having for their object to discover what influence is exerted by ordinary light, by the different coloured rays, and by darkness on the development, growth, and nutrition of animals.

After referring to the experiments of Edwards, Higginbottom, E. Forbes, Morren, Wöhler, Hannon, Moleschott, and Béclard, the results of which were shown to be somewhat contradictory, the author described the precautions taken by himself to avoid sources of fallacy.

The original experiments detailed in this Paper were conducted in the years 1855, 1856, 1857, 1858. The subjects selected were the Ova and Larvæ of the Silkworm (*Bombyx mori*) and of the Frog (*Rana temporaria*). A comparative experiment in the vegetable kingdom was also made on the Sweet Pea (*Lathyrus odoratus*). An apparatus contrived for the experiments on Tadpoles was described and figured; it secured the following desiderata:—

1. That each of six compartments or cells should be supplied with water from the same source, at the same time, subject to the same changes, and capable of being refreshed without interfering with the cells.

2. That each of the cells should be placed in the same condition with respect to the supply of air and of food.

3. That during exposure for examination of the animals, the whole series should be opened the same length of time and to the same extent.

4. That each cell should receive no light but that transmitted by its proper cover.

One of these six cells was open to the air and to light; one was covered with ordinary white glass; one was made completely dark by a covering of blackened opaque glass; one was covered with blue, one with greenish yellow, and one with red glass. The transmitting and absorbing powers of these glasses were detailed from experiments made upon them by Mr. Cornelius Hanbury, jun., and by the author.

The apparatus used for the Silkworms was, in all essential particulars, the same as that for the Tadpoles, only without water.

A tabulated analysis of the daily journal kept during the experiments was given, and its separate items compared and discussed; after which the author concluded his Paper with the following *résumé*.

If we may venture to reason on so small a number of observations, so far as the results of these experiments are concerned, the following propositions may be advanced.

All other conditions being the same, (1.) *The Ova of Insects* are not directly influenced in their development by white light, by the different coloured rays, or by darkness.

(2.) *The Larvæ of Insects* are not directly influenced in their development, growth, nutrition, or metamorphoses by white light, by the different coloured rays, or by darkness.

(3.) *The Larvæ of Batrachian Reptiles* are not directly influenced in their development, growth, nutrition, or metamorphoses by white light, by the different coloured rays, or by darkness.

4.) *The Materials necessary to the Colour of Insects and Reptiles* are prepared equally under the influence of white light, of the different coloured rays, and of darkness.

These results are so opposed to preconceived ideas upon the subject to which they relate, that they cannot fail to excite some surprise and incredulity; when, however, they are carefully considered, they assume a theoretical probability, which assists us in believing that the practical results are without fallacy.

(*a.*) With regard to the *development of the ovum*, when we consider the unity of plan which appears to preside over the germs of the simplest and of the most complicated forms, and the infinite variety of external conditions in which these germs are placed throughout the animal kingdom, we are led to the conclusion that their development must be so arranged as to be independent of the direct influence of light.

(*b.*) That *after emerging from the ovum* the animal is not directly influenced by light, is more difficult, at first, to believe, because experience seems to have taught us that "to live without light is to live without health;" but this familiar fact may be at once disposed of in the argument and explained by its coincident, that, under ordinary circumstances, the admission of light is inseparably connected with,

α. The regulation of external temperature.

β. The free circulation of a respiratory medium.

γ. Those processes of vegetable life and of inorganic change upon which the proper condition of the respiratory medium depends.

Speaking generally then, it must be admitted that light is essential to the development, growth, and nutrition of animals, but only *indirectly*. In the foregoing experiments, the usual coincidents of light, a proper supply of food, a due aëration of the respiratory medium, a properly regulated external temperature, &c. having been provided in each cell, the direct influence of light only being changed, no corresponding change occurred in the animal life.

In the vegetable kingdom the case is quite different, and the experiments on *Lathyrus odoratus* recorded in this paper demonstrate again, what has been shown by numerous other experimenters, that *light as a direct agent* is essential to the nutritive processes of plants. An interesting exception occurs, however, in the vegetable kingdom, which serves to strengthen the probability that the conclusions arrived at concerning animals are correct, viz. that fungi—which derive their nutriment, like animals, from organic compounds already prepared for them—perform their vital functions without dependence on the influence of light.

Under the head of colour, it would seem that the familiar phenomenon of *etiolation* witnessed in plants which have been deprived of light, has led to erroneous anticipations as to the effect which alterations of light would produce upon the development of the colouring materials in animals.

In the experiments here recorded, it is seen that neither white light, nor the different coloured rays, nor darkness altered the development of those materials necessary to the exhibition of colour, when the animal was seen in ordinary light. The experiments of Dr. Gladstone, on plants, also show that the development of colouring matter in the petals of flowers is independent of the influence of light; that flowers raised under the different coloured rays and in darkness have the same colour in their petals as when raised in ordinary light. Thus, even in vegetables, *etiolation* is confined to

those parts of the plant which depend for their colour upon the condition of the chlorophyl, to the green appearance of which some portion of the solar beam is evidently essential.

Although, therefore, at first sight, the results of my experiments under the head of colour may appear questionable, I think we must rather throw the question upon the correctness of our preconceived notions on the subject; and the facts elicited by Prof. Edward Forbes (referred to in the Paper), while retaining all their value and interest as assistants in determining the depths of primeval seas, cannot, I think, be taken as evidence against the correctness of my observations. On the other hand, the results of my experiments may be found to put a new construction upon the facts observed by Prof. Forbes. He discovered that increased depth of sea corresponds with diminished light, and that both of these conditions again correspond with peculiar changes in colour, and ultimately with loss of colour in the shells inhabiting these depths; but there is no evidence to show that these colourless shells have developed any materials capable of manifesting colour after exposure to the influence of light; whereas my own and other experiments show that the *etiolated* stalks and leaves of plants speedily manifest the characteristic colour of the chlorophyl if placed in the sun's rays.

So far, therefore, as our present knowledge on the subject justifies any conclusion, the varieties of colour and the absence of colour in the mollusks are physiologically separated from the phenomena of etiolation in plants, and may be placed in the same category as the varieties of colour and the absence of colour in the corollas of flowers, which depend upon the development of materials having certain optical properties.

The beautiful facts observed by Prof. Forbes, instead of being regarded as the consequence of imperfect exposure to light, must, I think, take rank with the phenomena of coloration observed throughout the animal kingdom, such as the peculiar markings of reptiles, birds, and wild animals, according to their different habitats and modes of life; the colours of the upper and lower surfaces of fish, and the like; which cannot be shown to depend upon the exposure or non-exposure to light with which they frequently, but not always, coincide. These facts appear only to form a part of the vast and perfect plan of creation, in which everything that exists is suited in every particular to the conditions of its existence; thus, those mollusks which are designed to inhabit depths scarcely permeable to light, can have no need, and hence have no provision, for elements, to the manifestation of which light is an essential condition.

ON THE COAGULATION OF THE BLOOD

IN THE VENOUS SYSTEM DURING LIFE.

BY THE SAME AUTHOR.

Medium 8vo. cloth, price £1. 8s.

I.

A TREATISE

ON THE

HUMAN SKELETON,

(INCLUDING THE JOINTS),

With Two Hundred and Sixty Illustrations drawn from Nature.

DUBLIN MEDICAL QUARTERLY.

"It does not consist of a mere detailed account of *dry* bones, but a beautifully written description of their development, growth, structure, union by joint, position in the human economy, varieties in the lower animals, and uses in standing, walking and running, and leads gradually on to the last most interesting chapter on the Homology."

GLASGOW MEDICAL JOURNAL.

"Mr Humphry's work does not come into competition with any other in our language, and the medical literature of our country has been enriched by him with a book which has long been an unsupplied desideratum—a *readable* treatise on Osteology....It is written in an attractive style, and contains sound anatomical information conveyed in a useful way."

II.

In the Press.

AN ESSAY

ON THE LIMBS OF VERTEBRATE ANIMALS.

III.

ON THE HUMAN FOOT.

A Popular Lecture.

From the Author

ON THE

COAGULATION OF THE BLOOD

IN THE

VENOUS SYSTEM DURING LIFE.

(A THESIS FOR A MEDICAL ACT IN THE UNIVERSITY OF CAMBRIDGE.)

BY

GEORGE MURRAY HUMPHRY, M.D. F.R.S.

SURGEON TO ADDENBROOKE'S HOSPITAL; LECTURER ON ANATOMY IN THE CAMBRIDGE UNIVERSITY MEDICAL SCHOOL.

(*From the British Medical Journal, with additions.*)

MACMILLAN AND CO.

Cambridge:

AND 23, HENRIETTA STREET, COVENT GARDEN, LONDON.

1859.

Cambridge:
PRINTED BY C. J. CLAY, M.A.
AT THE UNIVERSITY PRESS.

ON THE

COAGULATION OF THE BLOOD.

I.—Clots in the Veins.

The obstruction of the veins, by clots forming in their interior, has, of late years, been the subject of investigation by several pathologists, who have pointed out clearly the conditions under which it most frequently occurs, and the changes which take place in consequence. Still, doubts appear to exist respecting the causes of the phenomenon, and the starting point of the mischief; and, though the affection is one of very frequent occurrence, and may usually be diagnosed with facility, it certainly has not attracted the attention of practical men so much as it deserves. Very commonly it is suffered to pass unnoticed during life; and, after death, the vessels concerned are seldom examined to a sufficient extent, and with sufficient care, to enable the observer to form a correct opinion upon the matter.

Case I. My own attention was first particularly directed to this disease, in 1843, by its occurrence in a member of our own profession, a tall, thin, delicate person, who, when just beginning to recover from an attack of pleurisy on the right side, by which he had been much prostrated, experienced an uneasiness and a stiffness in the left groin and side of the

pelvis. For a day or two he did not pay much attention to the symptom, thinking he had probably strained himself by lying in some awkward position, though there was in reality no ground for such an idea. Soon, however, the uneasiness extended down the thigh into the ham, in the direction of the great vessels, and was accompanied by tenderness in the same part, with swelling on the inner side of the thigh, and enlargement of the superficial veins. It was consequently inferred that there was some affection of the femoral and popliteal veins; and this was rendered quite clear by the extension of the disease to the posterior tibial and lesser saphena veins; the latter of which formed a firm tender cord under the skin. The whole limb also became œdematous. A few leeches were placed over the femoral vein; but it was remarked that very little blood flowed from their bites. Poultices and fomentations were applied to the limb. Gradually the affection subsided, and the limb was restored to its natural condition. When the patient had so far recovered from these attacks as to be able to sit in a chair, he used to wheel himself about the room by means of the right leg, and in this way may have strained the part in some slight degree. At any rate, he began to perceive sensations in the right ham, which excited suspicions of the commencement of an affection there, corresponding with that which had just subsided in the opposite extremity. These suspicions were soon verified by the implication of the lesser saphena vein, and by the gradual extension of the uneasiness and tenderness up the thigh, in the course of the femoral vein, as far as the junction of the saphena major. There was also swelling of the limb. A few leeches were applied; but again very little blood flowed from the bites, in spite of diligent fomentation. The symptoms gradually subsided; and the patient quite recovered. Some œdema of the limbs remained for several months. This entirely disappeared in course of time; the superficial veins resumed their former dimensions; and there was every reason to suppose that the current was re-established through the deep veins. It is worthy of remark that the same patient exhibited symptoms of similar disturbance in the popliteal and femoral veins after each of two subsequent illnesses; viz. after an attack of typhoid fever in 1846, and of pleurisy on the left side in 1851. On the latter occasion, the upper part of the posterior tibial vein and the lesser saphena in the left limb, were involved, as well as the popliteal and femoral. The symptoms were slighter and of shorter duration than in the first attack, and were not followed by any evidences of permanent obstruction to the circulation, or impairment of the limbs.

The following cases occurred soon afterwards.

Case II. A man, aged 30, not much emaciated, died of phthisis with bronchitis and much prostration. Œdema of the right leg and thigh, with mottled blue and white patches on the skin, like those resulting from ecchymosis, had existed for some days; but had not attracted much attention. The external and internal iliac veins, and part of the common iliac, as well as the femoral, profunda, and saphena major, were distended with coagula, of a dark colour, irregularly intermixed with lighter fibrinous portions, which gave them a mottled aspect. Near the junction of the saphena, the clot was of more uniform colour, presenting the appearance of reddish, soft, dirty lymph, or fibrine, and was adherent to the inner coat of the vein. The latter had lost its polish, was rather soft, and admitted of being peeled off from the outer tunics of the vessel. The cellular tissue immediately surrounding the vein was, especially near the saphena, infiltrated with serum and lymph. The clot terminated abruptly in a thick rounded end, about an inch below the junction of the left common iliac vein.

Case III. A pale anæmic girl, æt. 19, was in the hospital on account of excessive debility, and was being dressed

by her friends for the purpose of removing her, without regard to the warning which had been given of the danger of such a proceeding, when she fell, fainting, and quickly died. She had been observed, during her stay in the hospital, to lie with the right arm hanging out of bed, and with her head inclined towards the right shoulder; not because she was in pain, but she preferred that position, and could not tell why. In the last few days the arm had swollen. We found the internal organs bloodless, but healthy, with the exception of slight emphysema of the lung. The right innominate, subclavian, and internal jugular veins, were obstructed and distended by a large clot, which, in the innominate vein, was of light or buff colour, scarcely tinged with red, and firm. It was adherent to the coats of the vessel, though easily separated from them; and the internal surface of the vein had lost its polish. In the jugular vein, the interior of the clot was softened to about the consistence of cream, and looked like a mixture of blood and pus. This semifluid portion of the clot was, on all sides, surrounded, and separated from the coats of the vein by a layer of firmer coagulum, which stretched, but was only very loosely adherent to, the vein. The coats of the several veins were natural; but the tissue on their exterior was indurated and infiltrated by serum and lymph.

Case IV. In a man, æt. 45, who died of phthisis, the right upper extremity having, for some days, been œdematous, and the superficial veins of the arm and shoulder distended, we found the subclavian vein, near its junction with the internal jugular, blocked up by a tough brown clot, which was adherent to the interior of the vein. The latter presented no unnatural appearance, except a loss of polish. There was some induration of the tissues on the exterior of the vein. The rest of the subclavian vein, the axillary and the internal jugular, were distended with dark clots; but were in other respects natural. The external jugular was free. There were large, dark, soft clots in the heart; also evidences of recent

pleurisy on the right side; and numerous tubercles, and recently formed cavities, in the lungs.

I have met with many similar cases; but it is needless to detail them, because they presented no great differences from those just given, and from others which are to be found in various writings since the time of Morgagni. In some, the deep veins of the calf only were implicated; their condition being evidenced by swelling of the limb, with uneasiness and tenderness in the course of the posterior tibial vessels, and an enlarged, firm, and tender state of the saphena minor. This has occurred lately in two cases, in which I had laid open the tunica vaginalis testis, for the purpose of inducing suppurative inflammation in that membrane. Both the patients were young subjects; one of them was in a debilitated state. In both of them the affection was on the side opposite to that on which the operation had been performed, and subsided spontaneously.

In all the cases that I have seen, with the exception of one of the two just mentioned, the patients have been in a feeble state, most of them having been previously reduced by some other disease. The most frequent causes of the accompanying debility were: some chronic disease, such as phthisis, or a discharging abscess; old age; low fever; or an acute inflammatory affection, more particularly of the serous membranes or of the lungs. In no instance has the condition of the veins appeared to be the cause of death, either directly or indirectly; though in many cases the patients died of the diseases which preceded that condition, and the state of the blood which was induced appeared in some to accelerate the fatal result. It is, moreover, a very important fact, that in no case, which has occurred within my observation or reading, has this affection been productive of any of those alarming and much to be dreaded symptoms, which attend occasionally upon traumatic inflammation of the veins, and occur under other circumstances, and which are supposed to

depend upon the admixture of purulent, or other morbid fluids, with the circulating blood. In some instances, as in Case I., the affection is attended with uneasiness, or pain, in the early stages. More commonly, it comes on insidiously and does not attract attention till the swelling of the limb is observed, when some tenderness in the course of the vein may generally be found. Not unfrequently, we are called upon to treat an œdematous state of one of the lower extremities, which commenced during an attack of fever, or some other illness, and which may be traced to an obstruction of the vein that had escaped notice. In several cases the first suspicion of any obstruction to the circulation has been excited by the observation, after death, that one of the limbs was swollen; this has led to an examination of the veins, and to the discovery in them of clots, which must have existed many days.

The circumstances under which the disease occurs, and the fact that it often affects several parts of the circulatory system at the same time, or consecutively, in the same person, are quite in accordance with the supposition that it depends, primarily, not upon a morbid condition of the vessels, but upon a preternatural tendency to coagulation in the fibrine; and this view derives confirmation from several of the following phenomena, which may be observed in the origin and progress of the malady.

Thus, the obstruction most frequently commences in the parts of the venous system which are most favourable to the coagulation of the blood; viz. in the great veins, particularly those of the lower extremities, where the current is more feeble than in other regions. The points of selection in the lower limbs are: first, at or near the junction of two large veins, as the external and internal iliacs, the superficial and deep femorals, the anterior and posterior tibials; the projecting angles between the confluent trunks, furnishing favourable spots for the settling of the blood: secondly, in the neighbourhood of the valves. These present loose, free edges,

to which the fibrine may readily adhere; and each has also the effect of shutting off from the circulating current the small quantity of blood which lies above it, included in the retiring angle, between the upper surface of the valve and the adjacent wall of the vein. The blood so situated must be almost at rest when the circulation is feeble and the limbs are kept quiet, because the valves will be then only partially opened; and, being at rest, it has a favourable opportunity to coagulate and become the nucleus of a larger clot. That this is no imaginary cause is proved by a case in which I found small dry clots lying above—that is, under shelter of—the valves of the femoral vein; the remainder of the vein being free from clots, or nearly so. Moreover, the veins just above the valves often present slight bulgings, or dilatations. Their walls are here a little thicker than at other parts, and they exhibit a faintly reticulated appearance upon the internal surface[1].

It is to be remarked, that the valves are more numerous in the lower limbs than in the upper, and in the deep veins than in the superficial[2]. They are also often placed in the main veins near the points of junction of large branches; so that a number of causes combine to facilitate the coagulation of the blood in these situations.

Thirdly, the formation of the clot often begins in the popliteal vein. This has relation, not merely to the fact that the trunks of the anterior and posterior tibial veins and the saphena minor are here united, but also to the fact that the internal surface of the popliteal vein is often remarkably uneven, presenting quite a reticulated appearance from the

[1] In a man, aged 76, who died of senile gangrene, I found a reddish brown clot, which was evidently of many days standing, closely adherent to the valves of the femoral vein, near the junction of the profunda. The rest of the veins, in both lower extremities, were healthy, and contained no peculiar clots.

[2] I have found the distances at which the valves are placed in the *superficial* veins of the lower limbs to be about equal to those at which they are placed in the *deep* veins of the upper limbs.

interlacement of opaque strengthening bands which form projections in the interior.

In the upper part of the body the clots form most frequently at, or near, the junction of the jugular and subclavian veins, where there are always large valves, and in the cerebral sinuses[1]. In the latter, the peculiar construction of their walls prevents much variation in their calibre, so that there must be considerable variations in the rate at which the blood traverses them in different states of the circulation; and they present, at the points of junction of the branches, many and marked projecting angles favourable to the settling of the fibrine.

The clots form not unfrequently in the venous plexuses around the prostate[2], and in the hæmorrhoidal veins.

It appears that, as a general rule, the formation of the clot commences on the outside, that is, near to the coats of the vein, where the current must be somewhat slower than in the axis of the tube; and the first stage in the process seems to be the settling of a patch or layer of fibrine upon the inner surface of the vein. This is probably increased by the addition of successive layers upon the interior, whereby the channel for the blood is diminished. Soon the tube is completely obstructed; this result being commonly accelerated, more or less, by the clotting of the blood, in addition to the settling of the fibrine. The two processes,—fibrinous deposit and blood-clotting,—which differ, probably, only in the circumstance that the greater rapidity of the latter causes the entanglement of the red globules with the fibrine, go on somewhat irregularly, whence the mottled appearance of the coagula; but, as a general rule, the clots are firmer and more fibrinous near the exterior, softer and darker in the middle. In a young woman, who died of fever, with peritonitis, excited by approaching perforation of the ilium, we were led to examine the veins by observing some œdema about the left ankle, and found the external and internal iliacs, at and near their junction, on both sides, occupied by coagula, which, on the left side, extended down below the popliteal vein, and, on the right, terminated in an ordinary clot at Poupart's ligament. Sections of these clots showed them to consist of laminated fibrinous tubes, moderately firm, and enclosing central cylinders of dark soft blood. The thickness of the fibrinous tubes varied. In some places, more particularly near the junction of the iliacs, where we judged the affection had commenced, it was so great as to leave little space for the dark central portion of the clot. In other parts, the fibrinous layer was thin; and in one place it was separated from the internal surface of the vein by a layer of soft dark clotted blood, resembling an ordinary recently-formed coagulum in consistence and appearance. This was probably formed from blood, which had insinuated itself between the clot and the vessel, and had coagulated shortly before, or possibly after, death. A transverse section of one of these clots showed very clearly the central dark soft coagulum surrounded by a circle, or tube, of laminated fibrine, which again was enclosed by a more recent dark external layer.

The exterior of the clot is usually smooth; sometimes it has quite a polished appearance, except at the points where it has become adherent to the sides of the vein. These adhesions are not usually very extensive; they are most commonly found where the clot began to form, and vary in their firmness with the period of their duration. The smooth character of the external surface of the clot is important,

[1] They were found by Virchow, *Froriepa Notizen*, xxxvii. 30, in the cerebral sinuses, in 6 cases out of 18.

In a remarkable case related by Mr Hulke, *Opkthalmic Reports*, April, 1859, an obstruction of the left cavernous and adjacent sinuses by coagula was attended with all the symptoms of aneurism by anastomosis of the orbit, and the carotid artery was tied by Mr Bowman.

[2] In a man, aged 67, who died with sloughing of the nates, after fracture of the thigh, I found several short thick firm clots, with stunted branches, in the veins near the prostate. They were smooth, quite unadherent, and tumbled out from the divided vessels. A section of each showed a central cavity containing red fluid, surrounded by a wall composed of tough, laminated, reddish or mottled fibrine. The fluid exhibited red corpuscles and a great number of pale nucleated cells.

inasmuch as it rather militates against the view so much advocated by Virchow and some other pathologists, that portions of the clot are very liable to be detached, and to be carried along in the blood-current, till they cause obstruction and give rise to secondary coagula in distant vessels[1].

The extension of the clot in the direction of the heart is usually limited by the junction of some large vein which is sufficient to maintain the current in the main trunk. Often the clot does not reach quite so far as this, but ends on the *peripheral* side of the junction. Thus, when the clot commences at the junction of the iliacs, on one side, it commonly extends about half way up the common iliac; and, in a case of cancer of the uterus, in which the iliacs, on both sides, were obstructed, the clot extended up the vena cava nearly as high as the renal veins. Sometimes the clot reaches further, and terminates in a round or conical end on the *cardiac* side of the point of junction of some large trunk. In the *peripheral* direction it is prolonged to a variable extent into the tributary branches, but does not usually reach the small veins: indeed, the latter are very rarely obstructed, either primarily or secondarily, in this affection.

The clot not only fills and chokes up the vessel, so as to prevent the passage of blood through it, but also distends or stretches it; and this distension, together with a certain amount of irritation resulting from the presence of a solid body in its interior, soon produces an effect upon the walls of the vein, the results of which are exhibited chiefly, or almost exclusively, on the *exterior* of the vessel. Thus we soon find that there is inflammation of the investing cellular tissue, causing an effusion of serum, lymph, or pus; whereas, in the *interior*, there is commonly little change beyond a removal of the epithelium from the lining membrane, and more or less intimate adhesion of the clot to it. There may be also an

[1] Mr Hewitt, *Medico-Chirurgical Transactions*, xxviii. 74, found the clot in one case "enveloped in a perfectly distinct, transparent, smooth, polished membrane, presenting the appearances of serous tissue, with arborescent vessels in its structure."

increase of redness at some parts, which is evidently due to staining by the contiguous blood, inasmuch as it is commonly proportionate to the colour of the contained clot, being deepest where the clot is darkest, and less marked, or quite absent where the clot is composed chiefly of fibrine. I have never seen lymph or pus, or any inflammatory product, formed from the interior of the vein. This proves that the inner coats of veins are by no means easily excited to inflammation, and is quite in accordance with the results of experiments made upon the veins of animals by Lee[1], Mackenzie[1], and Virchow[2]. It accords also with the general results of my experience, which by no means indicate a liability to inflammation in the inner coats of veins. I have, in many instances, applied a ligature to the chief vein of a limb after amputation, without any ill result in a single case; and I have never seen any mischief caused by the ligature of a varicose vein or a hæmorrhoidal tumour, though I have employed that method of treatment very often. It is not improbable that where unfavourable symptoms have ensued in cases of this kind, they have been caused, not so much by inflammation of the vein itself, as by suppuration in the surrounding cellular tissue.

When examining a vein which is plugged by a tough and adhering coagulum, one can scarcely be persuaded that the circulation could ever have been re-established through it, if the patient had survived; yet there can be no doubt that this does take place, and that a vessel may, in process of time, resume its functions, and be restored nearly, if not entirely, to its natural condition, after its channel has been completely, or

[1] *Medico-Chirurgical Transactions*, xxxv. and xxxvi. Dr Mackenzie infers, from the results of numerous experiments on the venous system, that the origin of *obstructive* phlebitis is to be sought for in a vitiated state of the blood, that this causes an irritation of the lining membrane of the veins at various points, which, in turn, leads to coagulation of the blood. I do not, however, discover sufficient evidence of this irritation of the lining membrane of the veins, and think there are many reasons against admitting that it is a necessary, or even the ordinary, intermediate link between the vitiated and the coagulated condition of the blood.

[2] *Handbuch der Specielle Pathologie und Therapie*, i. 161.

to a considerable extent, blocked up by a clot. The perfect restoration of the limbs in Case I., and in several other instances, assured me of this; and it is in accordance with the great difficulty which I have experienced in effecting the permanent obliteration of varicose veins by temporary ligatures, or by other means which had for their object the formation of coagula in the vessels. It appears that the blood is almost sure to revert to its natural channel, in process of time, unless the vein be completely destroyed[1]. The dissection in the following case illustrated the condition to which the clots become reduced. A man, aged 63, died, of erysipelas and pleuro-pneumonia, ten days after resection of one ramus of the lower jaw, performed on account of extensive necrosis and suppuration, which had continued for several months, and had reduced him to a very low state. His health had long been bad; but he did not mention that he had suffered any particular affection of the lower limbs. I was led to examine the veins in consequence of the condition of the pulmonary arteries, presently to be described. In dissecting out the femoral and popliteal vein of the left side, I remarked that the investing layer of cellular tissue, usually so delicate, was more coarse, tough, and closely adherent, than natural. With this exception, there was nothing to attract attention on the exterior of the vessels, or in the structure of their walls. In the interior (see Plate, figs. 1 and 2) were numerous delicate, but tough, white bands or strings, extending across or along the vessels; some were adherent in their whole length, and others only at their ends; also small firm lumps of pale yellow, or gravel, or golden colour, smooth on the surface, and more or less adherent to the inside of the vein. In some places there were merely yellowish stains in the lining membrane of the vein. The nature of these stains would have been scarcely recognisable,

[1] Hence in the treatment of varix, where it seems desirable to resort to operative procedure, my practice is to pass a needle or silver wire beneath the vein, and to allow the metal to find its way out by ulceration through the vessel and the superjacent skin.

had they not been in most instances continuous with the threads or some other evident remains of the clots. These veins contained also coagula, which appeared to have been formed recently, probably after death. The popliteal and lower part of the femoral vein, on the right side, presented appearances similar to those on the left. The upper part of the femoral vein was occupied by a firm, dry, mottled clot; and the profunda was tightly plugged by a continuation of the same, of white colour. Above the junction of the profunda, the vein was distended by a clot of comparatively recent formation, which, in the centre, was semifluid and of dirty cream colour. This soft part contained red corpuscles, and larger pale cells having indistinct nuclei.

It is no uncommon thing for the middle part of the clot to be, as in this instance, softened and converted into a dirty pultaceous or creamy substance, in which corpuscles are found, varying in size and shape, less regularly formed than pus-cells, and having less distinct nuclei. These are intermixed with oil-globules and red corpuscles; the latter may be natural in appearance, or more or less misshapen and granulated, and in various stages of dissolution. The changes which the blood thus undergoes are, as it would appear from the experiments of Mr Gulliver[1], similar to those which take place when it is subjected to concoction after its removal from the body. They seem most frequently to occur when the clot has been quickly formed.

In all the cases that I have seen, except one, the soft central part of the clot was walled in by the firmer exterior portion, so that there was no opportunity for any of the *débris* to enter the circulating current.

The exceptional instance was that of a sickly lad who died in a state of extreme emaciation, with bed-sores, &c. after amputation of the right knee. The left femoral vein was blocked up and distended by a clot which was slightly

[1] *Medico-Chirurgical Transactions*, xxii. 138.

adherent to the interior of the vessel and presented the appearances usual in such cases. It extended into the external iliac and terminated, near the upper end of that vessel, in a soft semifluid dirty substance, which had been free, so far as we could discover, to mix with the fluid blood in the common iliac and vena cava. If any barrier had existed it was broken down, before, or after, death, and had disappeared. The lad had suffered under inflammation and swelling of several joints, attended with perspirations and rigors, subsequent to the amputation; and a small abscess had formed near the left ankle. These, however, had all subsided before the commencement of that swelling in the leg, which indicated an obstruction in the vein; and there were no symptoms in the latter part of his illness, and no appearances after death, to justify the supposition that the blood had suffered from the admixture of morbid matter.

The right femoral vein in this same patient was contracted and converted into a firm white cord. Its canal was impervious to fluid; but I succeeded in pushing a probe along it, and, on cutting it open, found it occupied by a tenacious yellowish white substance not unlike putty. This, which I judged to be altered fibrine or blood, was seen under the microscope to be composed of granules of various sizes. The external and common iliac veins were also impervious. The outline of the former was not very distinct, its track being occupied by a quantity of yellowish white substance similar to that in the femoral vein; and some of this substance was in contact with the fibres of the psoas muscle, the coats of the vessel having disappeared. Doubtless these veins had, at a former period, been in a condition more or less similar to those on the left side; and they offered an example of the changes which may take place after the clot has been so softened and connected with the walls of the vein as to cause the obliteration of the vessel[1].

[1] There was a similar condition of the veins in a case described by Dr R. Lee, *Med.-Ch. Trans.* xv. 137. Specimen 1733 in the Museum of the College

It seems probable that the softening of these clots may sometimes give rise to the formation of abscesses. But I have never been able to prove that it does so. We not unfrequently meet with abscesses containing pus and blood in the course of the superficial veins of the lower extremities in persons who are in a low or disordered state of health; and such abscesses are supposed to originate in the vessels, and to be due to the softening of coagula found in them. In one

of Surgeons offers an example of a further result of the same process. It is thus described in the Catalogue: "The right iliac veins with the surrounding parts. Parts of them appear to have been completely obstructed by clots in which bone-like matter has been formed." "The whole length of the left external iliac vein is obliterated and contracted, and its coats are slightly distended, thickened and indurated. In its interior there is an appearance of the coagulum of blood by which it was probably at first obstructed, and which has now lost its colour and become firm and completely adherent to the inner surface of the vein." They were taken from the Earl of Liverpool, who had a swelling of the left leg and thigh, and a varicose state of the veins from the ankle to the groin, for several years before death.

Cases of obliteration of the veins, some of which were probably from similar causes, are given and referred to in Hodgson's *Treatise on the Arteries and Veins*, p. 525.

The appearances in the following case are interesting; though it may not be clear how far they are to be connected with one another. A young woman died of phthisis. There was nothing to create suspicion of any disease in the veins. On the inner surface of the lower part of the vena cava was an opake-white slightly raised patch, tolerably well defined, of the size of a fourpenny-piece. The surrounding interior of the vein presented radiating lines indicative of some contraction having taken place. The patch was quite smooth, of tough structure, and consisted of fibres, some of which resembled those of elastic tissue, others were straight and parallel, and others, rather thicker and less clearly defined, were nucleated. At the junction of the iliacs, on the left side, the lining membrane was rather thickened, of opake-white or yellowish colour, and slightly reticulated, forming a patch resembling that in the vena cava, but larger and less well-defined. There was also a similar appearance of radiation, in the interior of the vessel, around it. Though the blood was fluid in most other parts there was a large dark clot in this situation, having the appearance of a clot formed after death. In the right femoral vein, lying under the shelter of one of the valves, between it and the side of the vessel, was a small, dry, firm, mottled clot just fitted to the niche which it occupied. It had evidently been formed some time before death, and it was slightly adherent to the side of the vein. While the vessels were being removed a firm cylindrical clot fell out from the profunda vein. It was about two inches long, conical at the ends, dry, with a smooth surface, and was composed of dark and white portions alternating irregularly.

case, however, of the kind the patient died; and I dissected carefully without being able to discover any communication, or connection, between the several abscesses and sinuses and the trunk or branches of the saphena vein over or near which they were situated; neither was there any evidence of clots having formed in these vessels.

We come next to inquire what are the conditions of the blood which predispose it thus to coagulate in the veins during life. It has been already remarked, that the clots are most liable to form in persons who are in an enfeebled and cachectic state. Now, in this state, it is well known that the fibrine of the blood exceeds its normal proportions; and it appears that its tendency to coagulation is increased by there being also in the blood an excess of water, which dilutes the saline or ammoniacal elements, and thereby renders them less able to hold the fibrine in solution. Nevertheless, persons often remain in cachectic and anæmic states for a great length of time; and they may, in addition, suffer several and prolonged attacks of syncope, without any coagulation of the blood taking place. Indeed, the cases in which the latter occurs are quite the exception; and we therefore search for some other cause to explain the phenomenon in these exceptional instances. It is most frequent when the cachexia has been induced by some inflammatory affection; and we know that the effect of inflammation, more particularly when it attacks the serous membrance, is to increase the amount and the coagulability of the fibrine. The parturient state, which, especially in the early period of lactation, is productive of a similar effect upon the blood, is also marked by a great tendency to clotting of the blood in the venous system. The researches of Dr Richardson[1], continued with great assiduity and care through a long period, give strong reason for his view that the fibrine is held in solution by the presence of ammonia, and that its tendency to coagulate in the body is increased, and its coagulation out of the body is accelerated, by a diminution of the volatile alkali of the blood;

[1] *The Cause of the Coagulation of the Blood*, 1858.

and it is quite probable that, in the cases which we are discussing, an insufficient quantity of this solvent medium is one of the proximate causes leading to the clotting of the blood in the vessels[1]. There may be, in addition, some alteration in those relations of the corpuscles to one another and to the walls of the vessels, which, though not well understood, have an important influence in facilitating the circulation of the blood[2]. Certainly there seems no good reason to attribute the affection to the introduction of pus or other morbid fluid into the circulating current.

The diagnosis of the disease is easy. Œdema of the limb is often the first symptom. This may be attended with, or preceded by, uneasiness in the course of the affected vessels; and there is enlargement of the superficial veins, with, perhaps, induration of some of them. When the femoral vein is affected, the inner side of the thigh is sometimes swollen in a marked manner. The integuments usually remain white; occasionally they are inflamed in patches, or in a more diffused manner; and occasionally they are mottled by purplish spots, like petechial spots, or like those resulting from ecchymosis. Mortification rarely or never results from this cause alone[3].

I have already said that the affection is rarely attended

[1] The researches of Scherer and Lehmann (*Physiological Chemistry*, i. 97) show that the blood sometimes exhibits an acid reaction in the puerperal state; the acid present is supposed to be the lactic. This may be associated with the fact that the skin may be often observed to be remarkably dry in cases in which the blood coagulates in the vessels during life. An incipient decomposition of the blood is regarded by Zimmermann to be one of the chief causes of its coagulation.

[2] The commencement of the clots must be attributed to influences affecting the fibrine, rather than the corpuscles, because at the parts where they are first formed they are usually composed almost entirely of fibrine. Nevertheless, it is most probable that changes in one of the constituents of the blood are attended with corresponding alterations in the others; and the analogy that may be drawn from what is observed in inflammation and in pregnancy, suggests that an increase in the coagulating tendency of the fibrine is accompanied by an increase in the adhesive qualities of the corpuscles, which would materially contribute to the formation of clots, and which may, indeed, sometimes be the immediately originating cause of them.

[3] Virchow, *Handbuch der Speciellen Pathologie und Therapie*, i. 171.

with any serious consequences. It does not commonly seem much to aggravate the patient's condition, or to diminish his chance of recovery: indeed, I have sometimes observed an amelioration in the general condition of the patient to be coincident with the swelling of a limb which indicated an obstruction in the great vein; as though the general mass of the blood had become thereby relieved of a certain quantity of its redundant fibrine, and was consequently better fitted to minister to the healthy nutrition of the body. If the obstruction of a vein occur during the course of an inflammatory disease, it generally takes place when the disease is subsiding: it may, therefore, be regarded as an attendant on recovery, though it is an evidence of a low or cachectic state of system. I may again remark, that it seems to have no relation to the malady called "pyæmia;" and though necropsies prove that it is sometimes associated with the formation of clots in the pulmonary arteries, I have not in any case had clinical evidence of its being followed by that formidable affection.

Nevertheless, it is a dispiriting, tedious malady; it retards the restoration of the patient, keeps him confined to his bed, and causes much annoyance and apprehension. The liability to its occurrence is to be borne in mind as a reason against having recourse to depletion or purgation, or other measures which may exhaust the patient, or draw away the saline and watery ingredients of the blood, in the latter stages of an inflammatory or febrile affection. I have given ammonia in some cases in which I thought there might be a predisposition to the formation of clots; and, where the general condition of the patient is likely to be benefited by the use of such a medicine, we should not ignore the evidences which have been afforded of its influence in retarding coagulation of the fibrine. If given in a pure form, it is probable that some of it will enter the blood; and that it will operate in the living vessels, more or less, in the same manner as it is found to do when mixed with blood which has been removed from the body. When the blood has begun to clot in the veins of a limb

I do not think that much good results from any particular local treatment. The affection runs a certain course, and, if the disease upon which it is an attendant do not prove fatal, will gradually subside spontaneously; and the veins usually become clear again. Considering the nature and cause of the malady, we should be unwilling to resort to the use of leeches. Nevertheless, I have known decided relief follow their application when the pain and inflammation around the vein was considerable. Fomentations are sometimes attended with comfort.

Of the instances which I have seen, the greater number have been in males[1]. Nevertheless, it appears to be the same condition of the veins which in women, after delivery, usually constitutes the disease known by the name of "phlegmasia dolens." The cause of the disease, and the results disclosed by dissection, appear to correspond very closely, whether it be developed after parturition, or in the course of an illness; and the name "phlegmasia dolens in the male" has, accordingly, been applied to it by Sir H. Halford[2] and others. Phlegmasia dolens most frequently occurs *after* parturition, at that time when the fibrine of the blood is found to be most abundant; viz. during the early period of lactation; and it is most common in women who are weak at the time of delivery, or whose strength has been reduced by flooding, and especially in those in whom there has been, in addition, peritonitis or some inflammatory affection of the chest. Moreover, when a fatal result has followed, it has commonly resulted, not from the condition of the veins, but from some other cause[3]. It must, however, be observed that, in phleg-

[1] Of forty non-puerperal cases tabulated by Dr Mackenzie (*Medico-Chirurgical Transactions*, xxxvi. 235), about two-thirds were females, and one-third males.

[2] *Medical Gazette*, x. 172.

[3] See paper by Dr Davis, who first pointed out the true nature of this disease, *Medico-Chirurgical Transactions*, vol. xii.; also papers by other writers in the same *Transactions*, and in various medical journals. Dr Simpson, in his lectures recently published in the *Medical Times and Gazette*, calls attention to the fact that in some well-marked cases of phlegmasia dolens the veins have been found quite healthy.

masia dolens, the pain is usually more severe, and the disease, on the whole, is of a more acute nature than in the ordinary obstruction of the veins: it seems sometimes to originate in a morbid condition of the uterine veins, and is sometimes attended with, or productive of, those inflammatory and suppurative affections in distant parts which are attributed to a morbid condition of the blood.

Indeed, phlegmasia dolens would seem to occupy an intermediate position between the simple and comparatively innocent obstruction of the veins, which we have been considering, and the more severe and dangerous affection of the veins, which has been described by Arnott[1], and others, as an occasional attendant upon wounds and injuries. The changes which occur in the veins, and in the blood contained in them, seem to be much the same in the three classes of cases, except that in the traumatic variety the inflammatory symptoms are commonly more severe. And there is the further important difference, that in it we are liable to encounter that alarming, and commonly fatal, train of symptoms which is supposed to depend upon a purulent infection of the blood; whereas, in the ordinary obstruction of the veins, as I have already said, such concomitants are little to be apprehended.

The purport of the foregoing remarks may be condensed into the following summary:

1. The great veins are very liable to become obstructed by clots forming in them when the patient is greatly debilitated, and when the circulation is enfeebled—by inflammatory affections, by discharging abscesses, difficult labours, and other causes.

[1] *Medico-Chirurgical Transactions*, xv. 46. Mr Arnott remarks, and I think he was the first to call attention to the fact, that the inflammatory changes "are usually limited by the passage of a current of blood; where a trunk is concerned, the boundary line being the entrance of a branch, and where a branch is concerned, the boundary being the junction of this with the trunk."

2. The clots result from an altered state of the blood, disposing the fibrine to solidify; and are found in those parts of the veins which offer the greatest facilities for its so doing.

3. The inflammation of the veins is a consequence of the presence of the clot, and is chiefly confined to their outer coats, and to the surrounding cellular tissue.

4. The clots may soften and become intimately connected with the walls of the vessels, and may lead to the complete and permanent obliteration of their canals: more commonly, however, they are removed, or shrink into delicate bands or fibres, which offer little or no obstruction to the circulation.

5. The affection rarely leads to any serious result. It may be associated with so-called pyæmia; but has no necessary or frequent connection with it.

II.—Clots in the Pulmonary Artery.

The following examples of this affection have come under my notice.

Case I. A thin, delicate lady, aged about 35, was confined with her third child, and had so quick and easy a time that the medical attendant, who lived close by, was not summoned. No unfavourable symptoms followed till the fourteenth day, when, having been nursing her infant, she went into an adjoining room, and whilst in the act of standing up to pour out tea, she suddenly fell back upon the sofa, as if faint, and died. The pulmonary arteries, on both sides, where they enter the lungs, were plugged with dark clots of moderately firm consistence. These were not of uniform colour, some parts being darker than others; and they did not appear to have formed long before death. They extended into the second divisions of the arteries, and were only slightly adherent to the walls of the vessels. The latter were quite healthy. The lungs, heart, and other great organs, and the blood in them and in other parts of the body, presented nothing remarkable.

Case II. An exceedingly fat woman, aged 54, had been confined to her room three weeks by a sore leg, and in the last few days had suffered three attacks of dyspnœa and faintness, for which ammonia had been given by her medical attendant. One of these occurred on the 21st of the month, and she felt very ill all that day. On the 22nd she was better. On the 23rd, being in a neighbouring house, I was summoned to her in consequence of a sudden seizure, and was in time only to see her die. There were a few inspirations, but I could not feel the pulse. When first attacked, a few minutes before her death, she was quite sensible, and said, "It's of no use; I'm dying." The main branches of the pulmonary artery, on both sides, were distended with firm, dark, mottled clots, which extended into the second and third divisions of the artery. The interior of the vessel was discoloured, but for the most part it preserved its polish. In a few places, where the clots were slightly adherent, it had lost somewhat of its natural smoothness. In other respects it was quite healthy. The substance of the lungs was natural. The walls of the heart were flabby and thin; but the muscular structure and the valves were healthy. The right auricle and ventricle were distended with clots which were large, firm, and fibrinous in their upper parts.

Case III. A girl, aged about 18, had been in service in the town, in a hard place, under a hard mistress, had been ailing for several days, had one or two fainting fits, and in the last few days, had suffered from shortness of breath, and other symptoms which excited suspicion of inflammation of the lungs. When she was admitted into the hospital she had an anæmic appearance, her breathing was quick and hurried, and her manner was suggestive of hysteria, rather than real disease, to the gentleman who then saw her. Next day a faint abnormal sound was thought to be audible about the base of the heart; ten minims of tincture of digitalis were prescribed. At six o'clock that day, just after the bowels

had been relieved in bed, the bed-pan being used, she fainted, and died before the house-surgeon could reach the ward. The left pulmonary artery contained a tough clot, tightly impacted in the main trunk, with prolongations extending into the third and fourth branches. It was party-coloured, with alternating black and white portions, quite free and smooth in the smaller branches of the vessel; but in the first and second divisions it adhered at some points to the lining membrane so as to require slight pressure with the handle of the scalpel to detach it. The vessel appeared to have lost its epithelium at these points, and presented some patches of red. The right pulmonary artery was still more tightly plugged with a tough, dry, party-coloured clot, which was adherent to about the first and second divisions of the vessel, and was at this part softened in its middle to a considerable extent. The heart and the large vessels of the trunk and extremities were healthy. The clots in the venous system were rather larger and firmer than usual, but appeared to have been all formed after death. The contiguous parts of the upper and lower lobes of the right lung were consolidated by pneumonia, and covered with lymph.

Case IV. An emaciated young man had for several weeks suffered under symptoms of abdominal and pulmonary phthisis, with feverishness and diarrhœa. He was reduced to a very low state, and was slowly dying for some days. There were tubercles in the peritoneum, and in both lungs. In the pulmonary artery of one lung was a large, firm, fibrinous clot, nearly filling the vessel, with prolongations extending into its third and fourth divisions. It was slightly adherent about the point where the artery first divides; and the interior of the vessel, at this part, was rather rough and reddish when the clot had been removed. At the corresponding situation in the other pulmonary artery was a mottled clot of darker colour, and apparently more recent. It was slightly adherent to the vessel, and its prolongations extended

into the large branches. The left lower extremity was œdematous, and the femoral and profunda veins and their tributaries were distended with dark, mottled clots, which were slightly adherent about the neighbourhood of the valves. Neither the coats of the vessels, nor the investing tissue, had undergone any change. In the profunda, two valves were adherent together; and a narrow, tough bridle, of yellowish colour, passed from them to the adjacent side of the vein. It looked as if it were the remnant of a clot which had formed at some previous period.

Case V. A lad, aged 12, died, much emaciated, with disease of the hip and discharging abscesses. The left pulmonary artery was extensively occupied by clots. These, in the larger branches, were light coloured and soft; in the smaller branches they were darker, more mottled, tougher and drier; in neither were they adherent to the lining membrane, except in one situation where a vessel looked as if converted into a long narrow abscess. There was no other disease in the lungs. Well formed coagula, of the usual character, and some fluid blood, were found in the cavities of the heart. The lower part of one external iliac vein, with the femoral and profunda veins, were blocked up by coagula, which were closely adherent in the vicinity of the valves.

Case VI. An old man died exhausted, after suffering retention of urine for some time, which required the frequent use of the catheter. The left lower extremity was swollen; and the great veins were distended with clots from the junction with the right iliac, where the clot terminated abruptly, to low down in the leg. They appeared to have commenced where the profunda joins the femoral. In the left pulmonary artery, where it divides into its primary branches, was a small, toughish, slightly mottled clot, adhering to the lining membrane, which was very slightly altered.

Case VII. In the patient who died after removal of a portion of the lower jaw, followed by erysipelas and pneumonia, whose case has been before referred to (page 14), the trunk and primary branches of the right pulmonary artery (fig. 3) were nearly filled up by a firm, yellowish white, fibrinous clot, which was for the most part loosely adherent to the vessels, but in some places was so firmly attached that the lining membrane was torn up in the attempts to detach it. In the smaller branches of the artery the clot was mottled with red and white portions, was less adherent, but was firm and dry, and filled up the canal. Its ramifications extended as far as I could follow them. The lower lobe of the lung was hepatised; and there was sero-purulent fluid in the pleural cavity. The trunk and primary branches of the left pulmonary artery (fig. 4) were occupied by a still firmer and older clot, which was white, quite closely adherent to one side of the vessel, so as to form a part of its wall; and the smooth surface of the clot was continuous with that of the artery. Though the new structure stood out in strong relief, and occupied a considerable space of the canal, there was still room enough for a large column of blood. The ramification of this clot in the secondary and tertiary branches of the artery were closely adherent to the sides of the vessel, so as to form mere ridge-like projections, which would not have much interfered with the current of blood. They were marked with dark lines and spots, as if from deposit of pigment in them. In some of the branches of the artery the only remaining trace of the clot was a staining of the lining membrane with delicate dark lines. These stains were continuous with the clots which occupied the larger branches. There was no apparent disease of the vessel itself. The lower lobe of this lung was in a pneumonic state.

There can, I think, be little doubt that the formation of the clots in the pulmonary arteries, in these, and other like cases[1],

[1] Baron, *Archives Générales de Médecine*, ii. p. 1, appears first to have directed attention to the subject. Paget, *Medico-Chirurgical Transactions*,

is due to the same causes as the formation of the clots in the great veins; that is to say, it is due, primarily, to an increase in the coagulative tendency of the fibrine of the blood, and, secondarily, to some facilities which the vessel offers for that coagulation to take place. With regard to the primary cause: the pulmonary clots have been found chiefly, if not exclusively, in cases where the vital powers have been lowered by some other disease; in short, in cachectic states, especially where an inflammatory affection was superadded; and after confinements; that is, in precisely the same conditions as the venous clots. Moreover, the clots are often found in both situations in the same patient, as in Cases IV. V. VI. and VII.[1]

With regard to the secondary, or immediately inducing cause: it has not been satisfactorily shown in any one instance that the clot was preceded by, or attributable to, disease in the coats of the vessel. The discoloration and roughening, where they were present, was evidently due, as in the case of the veins, to the presence of the clot, and were not the cause of it[2].

It appears, from the above related cases, and others which have been recorded, that the clots begin to form, in some instances, in the smaller branches of the vessels; and that in these instances there is often some obstruction to the circulation in the artery, caused by inflammation, pulmonary apoplexy, or other cause, which must tend to promote the settling of the fibrine. In other cases the affection commences in the

ix. and x. Ormerod, *Medical Gazette*. Virchow, *Archiv*, x. 225. (In the case related here, the blood in the heart was fluid), and *Handbuch der Speciellen Pathologie*, Bd. i.

[1] Virchow, *Froriepa Notizen*, xxxviii. 35, remarks that in only one case out of eleven, in which clots were found in the pulmonary arteries, did he fail to discover them in some other part of the venous system.

[2] Kidd, *Dublin Journal of Medical Science*, xxii. 376, attributes the affection to inflammation of the pulmonary artery. Baron alludes to the absence of an inflammatory condition of the vessel in the case described by him. Nevertheless, in some other cases, to which he refers, he conceives that inflammation of the wall of the vessel led to the formation of the clots.

larger branches, or in the main trunk; and in these the spot at which the first deposit takes place is usually at or near the root of the lung. In this situation the pulmonary artery breaks up at once into a number of branches, which radiate from it, at different angles, to the several parts of the lung. Consequently, a large extent of surface is presented to the blood, and there are numerous angular projections into the current; both which conditions are calculated to induce the coagulation of the fibrine. It must also be remembered that the rate at which the blood travels through the pulmonary arteries is subject to considerable variations, depending, partly upon the alternating contraction and repose, and the varying force of the contraction, of the right ventricle, and partly upon the vicissitudes of respiration; and both these sources of disturbance become more marked in those enfeebled states, with tendency to fainting, in which we have found that the fibrine has a peculiar disposition to settle. In such states the circulation in the vessel must always be feeble; and, probably, the current is sometimes absolutely suspended in certain portions of the artery, or in the main trunk, for short periods. Moreover, the venous blood seems to exhibit an increasing disposition to coagulate in the body as it approaches the lungs, and therefore may be presumed to acquire the property in the greatest degree in the pulmonary arteries.

At any rate, the clots which we find in ordinary *post-mortem* examinations are more common and larger in these vessels, and in the right cavities of the heart, than in any other parts of the body. Frequently they are of firm consistence, while the blood in the other veins, and in the left side of the heart, is quite fluid.

Virchow attributes the formation of these plugs in the pulmonary arteries to the lodgment there of small clots, or fragments of clots ("emboli") which have been formed in the veins, and have been wafted, with the blood, through the right cavities of the heart, towards the lungs. These fragments, he thinks, become detached from the ends of the clots

which project into the great venous trunks; thus, in any case, where the end of a clot, formed in one iliac vein, projected into the vena cava, a portion may be washed off by the blood flowing against it from the other iliac vein, and, being carried into the pulmonary artery, may lodge upon one of the projecting angles of the vessel, and constitute a nucleus for the formation of a plug. It is not improbable that this may sometimes occur. It must, however, be remembered that the surface of the venous clots is usually quite smooth, and therefore not very likely to be disintegrated by the slowly flowing current of blood; secondly, that in many cases, as in No. I. II. III. there was no reason to suppose that the pulmonary clots were preceded by clots in the veins; and thirdly, that the effects of a preternatural tendency of the venous blood to coagulate are, for the reasons just given, likely to be exhibited in the pulmonary arteries as well as in other parts of the system.

The pulmonary clots undergo the same changes as those in the veins, provided the patient survives. They may soften (Cases III. and V.)[1] or become firmly adherent to the vessel and disappear, leaving scarcely a trace behind (as in Case VII.); or be converted into threads or bands, stretching from one part of the tube to another. I do not know an instance in which they have caused obliteration of any of the pulmonary vessels; though it is probable that this may take place occasionally in the smaller branches.

The plugging with coagula does not appear to induce inflammation on the exterior of the pulmonary arteries so easily as it does in the case of the veins of the limbs. It is not usually attended with pain or uneasiness, or any symptoms which lead, with certainty, to a diagnosis. Hurried, oppressed breathing, with faintness, occurring without any other obvious cause, would make us suspicious of this affection, and should induce us to auscultate in the situations in which a bruit,

[1] In the case related by Dr Kidd, *loc. cit.*, one of the clots was softened in the middle, and the tissues around the artery, on one side, were condensed and indurated.

originating in the pulmonary arteries, would be most likely to be distinguished. I am not aware that a bruit, produced in this manner, has yet been recognised, though it probably would have been discovered had attention been directed to the point during the life of any of the patients.

It is, indeed, a remarkable feature in the affection that the pulmonary arteries, one or both, in the main trunks, or in the larger branches, may be blocked up to a considerable extent without causing any sign of obstruction to the circulation, or of affection of the lungs, or, indeed, without producing any symptom whatever. In Case VII. it was clear, from the size of its remains, that a clot must at some time have occupied nearly the entire calibre of the main trunk of each of the pulmonary arteries; yet there had been no symptom of such condition observed during the life of the patient. In Case I. the patient appeared to be in her usual health till the moment of the fatal seizure. In this, and other parallel cases that have been recorded, there can be no doubt that the clots were forming for some time before death, and that sufficient blood found its way by the side of them into the lungs to maintain the circulation and supply the wants of the system.

The sudden death is probably caused by a slight exertion following a period of repose. During the repose we may judge that the clots are increasing; and the ensuing exertion, by causing a greater demand for oxygenated blood than can be supplied through the impeded pulmonary vessels, induces fainting, which is fatal. The extreme suddenness of the fatal seizure in these cases has suggested the idea that it may have been caused by some displacement of the clots, producing more complete occlusion of the vessels; but this is opposed by the fact that the clots are usually more or less adherent to the walls of the vessels, and show no sign of such displacement having taken place.

III.—Clots in the Cerebral Sinuses.

A girl, æt. 15, in whom the catamenia had never appeared, suffered headache for several days, and had been attended by a medical man, who thought the affection to be of hysterical nature. I saw her on March 7th. She had a dull heavy expression and an obstinate manner. The mouth was firmly closed; and she could not, or would not, put out her tongue or answer questions; yet she was evidently conscious of what passed and showed a certain amount of cunning. She did not appear to be able to use the left arm or leg, and when out of bed did not put the left leg to the ground; but when the soles were tickled she drew up both legs alike. The breathing was noisy. Head to be shaved; Aperient given and Hydr. Chlor. gr. ij every four hours. On the 8th the countenance was more dull and heavy, the eyes vacantly staring; when left alone she fell into a heavy sleep. Urine passed into the bed. P. 110 rather sharp.—Acetum Canth: and Empl: Canth: capiti. On the 9th she was rather less drowsy and took some notice, still did not move left arm or leg. After this the drowsiness increased to stupor; the pupils became rather dilated, and the optic axes ceased to converge; a slight tremor occasionally pervaded the frame; she became weaker, and the pulse became quicker. She died on the 11th.

Examination next day. Dura mater and arachnoid natural. Pacchionian glands larger and more grey than usual. On the right side of brain the convolutions were rather compressed and dry: on the left there was more than the usual amount of fluid in the pia mater. The ventricles contained a considerable quantity of fluid, which had evidently been recently effused, for the septum lucidum was torn through, and the lining of the cavities was in other places ragged. The interior of both ventricles was, moreover, studded with

numerous red spots, averaging about the size of a pin's head. These were most abundant in the right ventricle, and especially on the surface of the optic thalamus. An incision into that body disclosed a red patch in its middle, of about the size of a nutmeg, composed of a vast number of minute points of congestion or extravasation, so closely set, especially near the centre, as to give it almost the look of an apoplectic clot. We could not tell whether the appearance was entirely due to distension of the vessels, or whether there was extravasation in addition. Towards the circumference the red points were more separate. In the corresponding part of the left optic thalamus was a similar, but smaller and less deeply coloured, patch. A more or less ecchymosed appearance pervaded the remainder of the optic thalami, the corpora striata, and the adjacent substance of the brain. It was also present to some extent all over the grey matter of the right side of the cerebrum and the cerebellum, and in some parts of the left side; and there were spots of the same in various parts of the white substance of both hemispheres. We were struck by the appearance of the venæ Galeni, which were greatly distended with firm, mottled, slightly adhering clots. The same condition existed in the straight sinus and in the right cavernous, petrosal, and lateral sinuses. It was in some places difficult to detach the clots; and the lining membrane of the sinuses had, in these situations, lost its polish and was excoriated. Some of the veins on the surface of the hemisphere were blocked up with mottled coagula. The right jugular vein was healthy. The lateral and other sinuses on the left side and the longitudinal sinus were healthy.

It seems most probable that the blocking up of the sinuses was the earliest of the morbid phenomena, and that the others were dependent upon it; but what should have caused a deposition of fibrine to take place in these vessels is not clear[1].

[1] A similar case is related by Dr Bright, *Medical Reports*, ii. 57. Patient was a child, æt. 20 months, who had recently had pneumonia. Also cases by Dr J. W. Ogle, *Transactions of Pathological Society*, vi. 31, and in the last

IV.—On the Clotting of the Blood in the Cavities of the Heart.

In the following case the evidences of the formation of clots in the cavities of the heart and in the blood-vessels were unusually clear; and there can be little doubt that death was due to this cause.

A delicate girl, aged 11, was received into the hospital, March 27th, 1859, with a burn upon the outer surface of the right thigh, and on the palmar aspect of the right forearm. The skin was destroyed over a considerable extent; but it was hoped that she would recover, and, for several days, the wounds proceeded favourably, under the application of flour. About April 8th, without any apparent cause, she became rather feverish and refused her food. The slight feverishness subsided, but left her very feeble, with small pulse, dryish tongue, and shrivelled, harsh, scaling skin, the finger-ends feeling as after scarlet fever. Wine and nutritious diet were administered, and some improvement took place. On the morning of the 17th she was very low, but enjoyed her meals more than on previous days; and the nurse thought her better. About four in the afternoon she was somewhat uneasy, and begged the nurse to sit by her; but nothing remarkable was observed till about two hours afterwards, when she asked to be raised up in the bed. As soon as this request had been complied with, it was perceived that she was dying, and in a few minutes she was dead. No marked difficulty of breathing or other especial symptoms were noticed.

Fifteen hours after death we found the right cavities of the heart, the left ventricle, and all the vessels connected with these cavities, filled with fibrinous clots. The left auricle also con-

volume. In one the fibrinous clot in the sinus was attributed to asthenia, and in a second to disease of the internal ear; in a third it was associated with suppurative pneumonia.

tained a clot, but was not filled by it. The central and greater parts of the clots presented the ordinary characters of the fibrinous masses often found in the heart, though they were tougher and firmer than usual. They were not laminated. Under the microscope they exhibited very fine linear fibres, disposed in an irregular network. The exterior of the clots presented, however, in many places, quite a different appearance. It had a more opaque white, or cream colour, something like lymph in the neighbourhood of an abscess, and was composed, apparently throughout, of well formed cells, larger, clearer, and less granulated than pus-cells, with distinct nuclei. This cream-coloured portion of the clot was, in the right auricle, separated by a defined line from the remaining buff-coloured part, and could easily be peeled off from it. It had not quite a smooth external surface, and adhered slightly to the interior of the cavity, particularly in the appendix; nevertheless, it remained with the clot when the latter was turned out of the auricle. In the ventricles it adhered to the lining membrane of the heart more closely than in the auricles, especially about the edges of the valves, and near the apices of the cavities, prolongations of it being intertwined with the carneæ columnæ. Where the clots extended into the pulmonary artery and aorta, they were deeply impressed by the sigmoid valves; but were not adherent to them.

Near the bifurcation of the pulmonary artery the whole thickness of the clot had a dull cream colour, and this was the case in the primary divisions of the vessel. In no place were the clots adherent to the walls of the pulmonary artery. Fibrinous strings, presenting the usual appearance, extended into the smaller branches of the artery.

In the inferior cava and the iliac veins was some fluid blood, which coagulated on exposure to the air. There were mingled with it numerous small clots, coloured in different degrees: some of these were particoloured. They appeared to be free in the fluid blood, or, if adherent to the walls of the vessels, their connexion was very slight. In the great veins which converge

to the superior cava, the clots were more numerous, larger, and more fibrinous, but still loose in the tubes, or very slightly connected with their walls. The left jugular and subclavian veins, at and near their junction, were almost filled by yellowish or cream-coloured clots, having a peculiar coiled or wrinkled exterior (fig. 5), which must have been caused by their being subject to some movements during the flow of the blood, or to their having been formed, or increased, by smaller clots carried in the current from other parts, and intercepted there. None of the vessels were distended by the clots.

In all the arteries which I examined, except the aorta and pulmonary arteries, the blood was fluid. The heart itself was quite sound, and there was no disease of the internal organs.

I have seen other cases more or less similar. In none of them, however, did the symptoms and the appearances after death so clearly indicate that death had been caused by the clotting of the blood in the cavities of the heart and in the adjacent great vessels. Such cases form a highly important class, to which the attention of pathologists and practical men is being more and more closely directed, and for the clear appreciation and discrimination of which much information as to the causes influencing and regulating the coagulation of the blood within the body, before and after death, has yet to be sought. At present, I think we are unable to explain many of the appearances we are in the habit of observing, and of deciding with certainty whether they are to be referred to *post-mortem* changes, or whether they commenced during the life of the patients. For instance, we often find the cavities of the heart (the right auricle and ventricle more particularly, the left ventricle not unfrequently, the left auricle more rarely) filled with fibrinous clots, and the prolongations of the clots extending into the adjacent vessels and descending into the most dependent branches of the pulmonary arteries; so that the absence of colour in them does not admit of explanation upon the ordinarily received hypothesis of the subsidence of the red corpuscles during the slow coagulation which goes on in the

recently dead body, because the lowermost portions of the clots in the several cavities, and the most dependent portions in the pulmonary arteries, are as devoid of colour as the uppermost parts. Nevertheless, it seems pretty certain that clots of this kind are, for the most part, formed after death. Coincident with these fibrinous clots is sometimes a variable amount of fluid blood, which may be in the same cavities as the clots; commonly there is some fluid blood in the left auricle. Not unfrequently the clots extending from the ventricles into the pulmonary artery and the aorta exhibit well marked impressions of the semilunar valves, showing clearly that they were moulded upon the valves; and this is regarded as evidence of their having been formed before death[1]. Yet these clots are almost invariably found not adherent to the valves, but quite free from them and smooth. Moreover, each one commonly bears the impression of each of the three valves in an equal degree; and this often happens on both sides of the heart in the same body. If, therefore, these clots had formed, as is supposed, or had commenced, before death, upon the valves, they must have lined the whole of the interior of the vessels at and near their connexion with the heart, and must have completely covered and impeded the action of all the semilunar valves in such a manner, and to such an extent, that it would scarcely have been possible for the circulation of the blood to have been continued at all.

I think that additional observations upon the coagulation of the blood in the dead body, under varying circumstances and after various diseases, must be made before we can agree with the author of the excellent work *On the Coagulation of the Blood*, that either the tubular or the laminated structure of a clot, or the fact of a clot consisting of a fibrinous tube containing red blood in its interior, is conclusive evidence of the coagulation having begun before death. Lamination is a singularly prevalent and remarkable feature in the inorganic, as well

[1] Richardson, *On the Coagulation of the Blood*, 407 and 420.

as in the organic world; and the circumstances under which it occurs and the causes which induce it would prove a fertile subject for study and experiment. It is doubtless more commonly observed in those clots which, there is good reason to believe, have formed before death, than in those which have commenced subsequently. It would seem to depend upon the deposition of the fibrine in successive layers; though that is not certain, inasmuch as it may depend upon some peculiarity in the manner of the solidification of the fibrine, the particles becoming disposed in plates and layers, just as we know they have a disposition to be arranged in fibres; and it is no unreasonable supposition that this may, under favourable circumstances, take place after death. With regard to the tubular form of a clot, it is by no means improbable that, in certain peculiar conditions of the blood, the tendency of the fibrine to separate itself and settle upon the walls of the vessels, on the valves, and on other structures, may be manifested after death, in the same way that it often is before death; and may thus lead to peculiarities in the appearance and structure of the clot which render it very difficult, or impossible, to distinguish whether they are *ante* or *post-mortem* formations.

The mode in which the *fibrinous* and the *corpuscular* varieties of the fibrine are combined in the same heart, or even in different parts of the same clot, are difficult of explanation. Thus in a lad, who died with cerebral symptoms after amputation, I found in the right auricle and ventricle a firm white clot which presented, under the microscope, a distinctly fibrous structure, the fibres being clear and delicate, but strong; and in the left ventricle were small reddish clots, containing an unusual quantity of pale nucleated corpuscles. When the two varieties coexist in the same clot, the fibres generally preponderate at the circumference, and the corpuscles nearer the centre: the reverse, however, was found in the clots occupying the heart in the case above related.

I am not aware that the presence of small fibrinous clots dispersed throughout the venous blood, as in the case just

related, has been observed by other pathologists, and it is not very easily explained. I recently found them under the following circumstances.

An unhealthy man, who had encountered many hardships, and had long been a sufferer from various complaints, was admitted into Addenbrooke's Hospital on account of a cutaneous affection of peculiar character. It consisted of inflamed and swollen patches of skin, upon some of which the surface became scabbed or blistered, whereas in the others the portion of skin first affected resumed its natural appearance, and the disease spread, ring-like, in the circumference. He had also laryngitis, and was in a feeble state, with difficult breathing and small pulse. He became quickly more prostrate and restless; the difficulty of breathing increased, and after being twelve hours in a dying state, he sank on the sixth day from his admission. The treatment consisted in the administration of small dozes of mercury with Ipecacuanha and Quina and such nutritious diet as he was able to take. On examination twenty-four hours after death, we found interstitial suppuration in the glands and cellular tissue of the loins and pelvis; defined white masses caused, apparently by lymph-deposit, in the kidneys; a swollen and superficially ulcerated state of the mucous membrane of the larynx; a thin layer of recent lymph upon the pericardium and upon many parts of the endocardium; and a granulated state of the inner surface of the right auricle in the neighbourhood of the ventricular opening.

The clots in the right cavities of the heart, and in the trunk and large branches of the pulmonary artery, were large and dark, but presented nothing which attracted attention. They were, however, removed hastily; and any slight peculiarity might have escaped notice. In the left ventricle the greater part of the clot was also of uniform dark colour and consistence. Prolongations of it, however, which branched between the carneæ columnæ at the apex, were rather firmer,

and were composed of white and dark portions intermixed; they were only slightly adherent to the lining membrane. I examined the blood in the venæ cavæ, in the iliac, subclavian, axillary and azygos veins, and in the cerebral sinuses. In all these it was very dark; in some parts it was fluid; in others it was in soft black clots; and in the substance of many of the clots were small white or yellowish-white irregularly shaped masses of fibrine. These masses were nearly of the same consistence as the other portions of the clots, and were continuous with them; in some instances they were near the surface, so as to catch the eye directly the clots were drawn out of the vessels; in others they were more deeply imbedded. They presented under the microscope a remarkably distinct fibrillated structure. I did not find any of them in the fluid blood. There were thin continuous coloured clots in the smaller branches of the pulmonary artery, and in some of the other arteries; they presented a natural appearance, and there were none of the pale fibrinous masses which were so common in the veins.

It is, I think, pretty certain that these peculiar fibrinous masses were formed either after, or very shortly before, death, during, or just antecedent to, the setting of the blood, and that they had not moved from the position in which they were first formed. Had it been otherwise, they must have accumulated in the small branches of the pulmonary arteries, and caused obstruction there; whereas those vessels were quite free from them. Moreover, the fibrinous masses shewed no signs of having been wafted along in the blood-current, but looked as if they had, from the first, formed parts of the clots in which they were found. Neither is there any reason to suppose that they were fragments—"emboli"—detached from a clot laid down in any part of the venous system. There was no evidence of existence of any such clot: the fibrinous masses were found in each of the veins above-mentioned; and to have reached these several situations, they must, some of

them at least, if they were all derived from the same source, have gone the round of the circulation, and have traversed the pulmonary and systemic capillaries.

Perhaps an explanation of these, and of some other phenomena of a similar kind which are more frequent, may be given in the following manner. It is generally admitted that in certain conditions, in the *living* and in the *dying* body, the fibrine acquires the property of separating from the other constituents of the blood, and of forming clots which are more or less devoid of colour, or which are particoloured in consequence of the red corpuscles being only here and there entangled in the setting fibrine. The theory of the obstruction of the veins and pulmonary arteries given in the preceding pages is based upon this hypothesis. But is it not probable that the same thing takes place *after death*, and with even greater rapidity than during life? The influences which retained the fibrine in solution being withdrawn, any preternatural tendency to separation, or any tendency to separation and coagulation in an irregular manner, which had existed, and perhaps manifested themselves, during life, are likely to be evinced in a more marked manner after death. It seems not only probable, but almost certain, that this must be the case; and if it be so, we must cease to regard irregular intermingling of fibrinous and coloured masses, giving a pied appearance, as the evidence that a clot has been formed before death; we must look for some additional proof, such as adhesions of the clot to the interior of the vessel, or a choking up —a distension—of the vessel, or a corrugation of the clot, as in the case of the burnt girl above narrated, or some other sign of the influence of the blood-current upon it.

In the case of the man, which has just been described, I infer that the colourless portions of the clots were caused by the hasty separation and consolidation of the fibrine, at certain points, after death; and that the fibrinous masses thus irregularly formed were included in the fluid blood, and formed parts of the coagula which were formed in the ordinary way

from the blood. Probably, also, in the burnt girl some, possibly the greater part, of the clots were *post-mortem* productions, though, for reasons previously given, it may be presumed that some of them were formed during life.

It may be further assumed, that the tendency to separate coagulation of the fibrine will produce its effects after death in much the same manner, and in the same positions, as during life; and that it will, thus, lead to the formation of fibrinous clots upon and near the valves, upon the interior of the vessels, and between the carneæ columnæ of the heart. In this manner a tubular lining of fibrine may be laid down in the interior of the pulmonary artery after death, and be moulded upon the sigmoid valves while the remainder of the blood in the vessel is fluid. This remaining blood subsequently coagulating would form a soft dark cylindrical clot enclosed in, and continuous with, the fibrinous tube. Still later the contraction of the whole clot, more particularly of the outer fibrinous tube, would draw it away from the sides of the vessel, and leave it free in the interior or nearly so. Thus by a sequel of intelligible *post-mortem* processes we may have produced those tubular fibrinous clots containing red clotted blood in their interior, as well as those fibrinous or particoloured strings between the carneæ columnæ, which have been described by Dr Richardson and others, and which are commonly thought to be dependent upon changes going on during life.

I do not mean to say that such clots are always formed after death, but that they may be so, and that we must be careful ere we admit these and other similar appearances to be evidence that the coagulation of the fibrine has taken place during life.

CAMBRIDGE: PRINTED BY C. J. CLAY, M.A. AT THE UNIVERSITY PRESS.

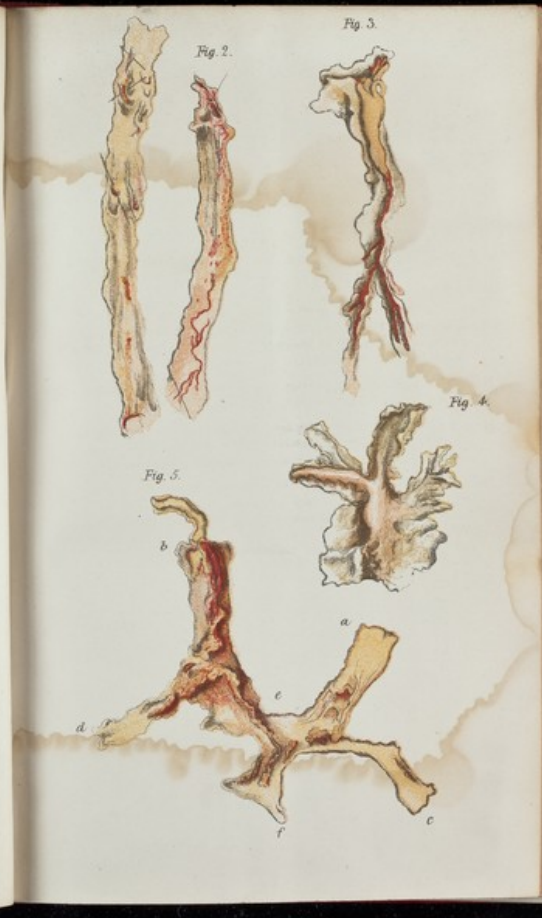
DESCRIPTION OF PLATE.

Fig. 1. The femoral vein, and Fig. 2, the popliteal vein. There are small coloured fibrinous lumps, also strings and bands and pigmentary stains, and other traces of the former occupation of the vessels by clots. (See page 14.)

Fig. 3. The right pulmonary artery from the same patient, containing a firm fibrinous and mottled clot.

Fig. 4. The left pulmonary artery from the same patient, containing a very firm and closely adherent clot. There are also black stains, continuous with the clot, in the lining membrane. (See page 27.)

Fig. 5. Wrinkled fibrinous and coloured clots at, and near, the junction of the large veins of the neck and arms: *a*, right jugular vein; *b*, left ditto; *c*, right subclavian vein; *d*, left ditto; *e*, innominate vein.



ON THE

NATURE OF THE SUBSTANCE

FOUND IN

THE AMYLOID DEGENERATION

OF VARIOUS ORGANS OF

THE HUMAN BODY.

(A THESIS FOR A MEDICAL ACT IN THE UNIVERSITY OF CAMBRIDGE.)

BY

FRANCIS HARRIS, M.D. (CANTAB),

DEMONSTRATOR OF PATHOLOGICAL ANATOMY AT ST. BARTHOLOMEW'S HOSPITAL;
ASSISTANT PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN, ETC.

Westminster:

PRINTED BY THOMAS BRETTELL, RUPERT STREET, HAYMARKET.

1860.

The occurrence in the animal kingdom either of Starch itself, or of substances exhibiting properties similar to those possessed by the different substances of the amylaceous group (which were at one time supposed to belong exclusively to the vegetable kingdom), is at the present moment a subject of considerable interest, both in its bearing on Physiology, and still more on Pathology, inasmuch as it is calculated to elucidate the nature of some of those degenerations with which at present we are only imperfectly acquainted.

The general characteristics of the Starch group of the vegetable kingdom may be summed up as follows:—

First,—These substances are non-nitrogenous,—simple compounds of oxygen, hydrogen, and carbon.

Secondly,—Under certain circumstances they are capable of appropriating to themselves oxygen and hydrogen, and so become converted into sugar.

Thirdly,—With iodine alone, or with iodine and sulphuric acid, they exhibit certain changes of colour—certain characteristic reactions.

The composition of Starch is $C_{12} . H_9 . O_9 + HO$.
" " of Cellulose $C_{12} . H_{10} . O_{10}$.
" " of Grape Sugar $C_{12} . H_{14} . O_{14}$.

All these substances, therefore, possess this peculiarity, that they contain 12 eq. of carbon, and amounts of oxygen and hydrogen in the proportions to form water.

The conversion of these substances into glucose, or grape sugar, by prolonged boiling with dilute mineral acids, is a fact which requires only a passing mention.

The characteristic blue colour, which is formed when free iodine comes into contact with starch, is also so well known as to require no description.

With cellulose, however, iodine alone produces little change of colour, and it is only when sulphuric acid is added that a blue colour is perceptible. If, for instance, a solution of iodine be

applied to cotton wool, the individual fibres are seen under the microscope to assume a faint pinkish brown tinge, and this tinge is readily removed by washing them in a stream of water; if, however, sulphuric acid be also added, the fibres swell up, take a deep blue colour, and ultimately dissolve;—even this colour vanishes after a few hours' exposure.

To shew, then, that any substance which is found in the animal kingdom is strictly analogous to this amylaceous group, it would be necessary not only to shew that it formed with iodine colours more or less resembling those produced by starch, but that also it was a non-nitrogenous substance; nor would these two characters alone be sufficient, as another substance possesses both these peculiarities,—Cholesterine is a non-nitrogenous body,—a compound of carbon, oxygen, and hydrogen alone—its formula being C_{27} H_{22} O . and with iodine and sulphuric acid it exhibits a blue colour, much resembling that which cellulose shews under the same circumstances. So that it is also necessary, in order to prove the amylaceous nature of any animal substance, that it should, in addition to these two properties, be also convertible into sugar.

Quite recently the investigations of M. Berthelot* have shewn that there exist in the animal kingdom two substances capable of fulfilling all these conditions. The one of these, Chitine, has been long known; for the other M. Berthelot proposes the name of Tunicine It had, however, already been described as animal cellulose, by Schmidt in 1845, by Löwig and Kölliker in 1846, and by Professor Huxley in 1853.

The organic matter of the envelopes and external skeletons of certain invertebrate animals differs from the organic matter of the bone of vertebrate animals, by its insolubility in solutions of caustic potash; in this respect it presents a remarkable analogy to certain vegetable tissues.

From the envelopes of certain of the Tunicata (the Ascidians were used in the experiments) M. Berthelot obtained the organic principle, for which he proposes the name of Tunicine, in the following manner:—

The envelopes were first boiled in concentrated hydrochloric acid for some hours, in order to free them from any earthy substance; they were then washed frequently with water, till all trace of the acid had disappeared. Afterwards they were boiled

* Brown-Séquard's *Journal de la Physiologie*, No. VII. p. 577. Oct. 1859.

with a solution of caustic potash, and again washed, till they became perfectly neutral.

There was then left a white opaque substance, of almost horny consistence, and non-crystalline in structure. When moist it was soft, supple, and tenacious. Under the microscope it presented a fibrous structure, like animal tissues, and quite different in this respect from vegetable cellulose.

By the ultimate organic analysis of this substance, he found that 100 parts yielded 44.6 of carbon, and 6.1 of hydrogen.

The formula C_{12} H_{10} O_{10}, the formula of cellulose, requires 44.4 of carbon, and 6.2 of hydrogen, so that tunicine may be considered isomeric with cellulose.

This analysis coincides very closely with two analyses, given by Schlossberger*; one by Schmidt, which gives

Carbon	45.38
Hydrogen . . .	6.47
Oxygen	48.15

The other, by Löwig, gives

Carbon	43.40
Hydrogen . . .	5.68
Oxygen	51.32

A mere identity in the ultimate chemical composition would, however, prove but little, unless some of the properties of this substance resembled those of the vegetable tissue.

M. Berthelot found that, with iodine and concentrated sulphuric acid, this substance assumed a very pale blue colour. The colour, he states, resembled rather that which cholesterine produced, than that of cellulose.

Schlossberger†, however, and Professor Huxley‡, both found that it coloured in a manner precisely similar to that of vegetable cellulose. This substance, however, differs from cellulose by its far greater resistance to acids. M. Berthelot found that it could be boiled with dilute acids for several weeks, without any change into sugar taking place in it.

Its resistance was so great, that it was necessary to employ re-agents of so strong a nature, that they were calculated not to produce sugar, but to destroy it, if it had already been formed.

Yet, after many failures, he was completely successful, in the following manner:—

* *Thier-Chemie*, Vol. I. p. 255. 1856.

† Op. cit.

‡ *Microscopical Journal*, Vol. I. p. 22. 1853.

Dried tunicine was mixed with cold concentrated sulphuric acid. By degrees it dissolved without becoming coloured. The fluid was then poured, drop by drop, into a hundred times its weight of boiling water. The mixture was then kept boiling for an hour. It was then saturated with chalk, and, after having been filtered, was evaporated to a syrupy consistence.

This syrupy fluid was a mixture of sugar with some substance of an indeterminate nature. It reduced energetically the potassio-tartrate of copper, and assumed a dark brown colour when boiled with caustic potash.

To a mixture of this syrup with water, yeast was added, and fermentation was readily set up, with the production of carbonic acid and alcohol.

The conversion of this substance into sugar was therefore fully proved.

The other substance, chitine, is the organic basis of the external skeleton of crustacea arachnida, insecta, &c. It may be obtained by a method similar to that employed for the preparation of tunicine. As early as 1823, M. Odier* considered it to be a substance approaching in its nature to cellulose. It differs from tunicine in one very important particular—it has not yet been obtained free from nitrogen.

M. Berthelot states that he boiled the chitine, which he obtained from the shells of lobsters, for several weeks with dilute sulphuric acid; and that the mixture was then left for a period of fourteen months at the ordinary temperature of the air, but that after this time the chitine was found almost intact, and still contained 5 to 7 per cent. of nitrogen.

Various other analyses of it have been made at different times. In one by Messrs. Children and Daniell† its composition is stated to be

Carbon	46.08
Hydrogen . . .	5.96
Nitrogen . . .	10.29
Oxygen	37.41

Schmidt found it to contain‡

Carbon	46.69
Hydrogen . . .	6.69
Nitrogen . . .	6.33

but found in it no trace of phosphorus or sulphur.

* *Mémoires de la Société d'Histoire Nat. de Paris*, Tome I. p. 29. 1823.
† *Todd's Cyclop. of Anat.* Vol. II. p. 882.
‡ *Zur vergleich. Physiologie der Wirbellosen*, p. 39. 1845.

Frémy* however considered it a non-nitrogenous substance isomeric with cellulose.

But on the other hand Lehmann and Schlossberger† both found in it 6.5 of nitrogen.

Frémy was unable to obtain sugar from it, nor could he form a detonating mixture with it and the elements of nitric acid, and stated therefore that it had the composition, but not the properties, of cellulose.

Schmidt formulated‡ it C_{17} H_{14} N O_{11}, and considered it a union of two substances; the one a proteine compound C_6 H_6 N O_2; the other a hydro-carbon C_9 H_8 O_8.

He obtained this latter formula by subtracting the formula for the muscular tissue of the crab, C_6 H_6 N O_2 from the formula for chitine above stated.

M. Berthelot inclines to this latter hypothesis in so far that he considers it a union of two substances, the one non-nitrogenous and the other analogous to horny matter. By means of a process exactly similar to that employed with the tunicine he was enabled to obtain a syrup which reduced the potassio-tartrate of copper, and with yeast fermented with the production of carbonic acid and alcohol.

The existence in the animal kingdom of these two substances which seem capable of fulfilling in every respect the conditions necessary to establish their perfect analogy with the group of amylaceous substances is a most interesting fact, and a very important link in the chain of evidence which tends to point out the starchy nature of the substances found in certain degenerations of the human tissues, of which I will presently speak.

To Virchow§ we are indebted for a knowledge of the fact, that certain structures in the human body when brought into contact either with iodine alone, or with iodine and sulphuric acid, exhibit changes of colour similar to those produced under the same circumstances with starch and cellulose.

* *Annales de Chemie*, Tome XLIII. p. 95. 1855.
† *Thier-Chemie*, Vol. I. p. 228.
‡ Op. cit. p. 50.
§ *Arch. für Patholog. Anatom.* Vol. VI. p. 135.

In 1854 he first shewed that certain of the round concentric bodies found in the so-called lining membrane of the ventricles of the brain (ependyma ventriculorum) exhibit a pale blue colour on the addition of iodine, and on the further addition of sulphuric acid, a beautiful violet. This reaction separated these from certain other forms of concentric granules (Brain-sand), and to the class thus distinguished he restricted the name "Corpora Amylacea." He considered that this reaction alone was sufficient to indicate their cellulose nature; for though he was unable to isolate them in sufficient quantity to subject them to chemical analysis, he considered their cellulose nature beyond doubt, as there was no other known substance in the whole range of animal tissues which produced similar reactions.

These observations of Virchow's were repeated by Mr. Busk*, who, however, arrived at the conclusion that these bodies were not cellulose but genuine starch, since they "possessed all the "structural, chemical, and optical properties of starch as it "occurs in plants." They exhibited concentric markings,—they coloured blue with iodine,—they acted upon polarized light in the same manner as starch does. Other observers, however, have not been able to convince themselves of the actual starch-nature of these bodies†. Schmidt while distinctly expressing his opinion that the covering of the tunicata is actual cellulose, still thought that the cellulose nature of the corpora amylacea had not at that time been determined.

Until, then, these bodies have been shewn to be convertible into sugar, some doubt must exist as to their nature.

The occurrence of starch itself, as starch in the human body, has been maintained by Mr. Carter‡. In endeavouring to make out the structure of a tumor of the optic nerve, he found a large number of bodies (corresponding to the corpora amylacea of Virchow), which, when brought into contact with iodine, passed through various shades of purple to the densest black. He afterwards examined a long series of tissues, some healthy, others variously diseased, both in the human subject, and in the

* *Quarterly Journal of Microscopical Science*, Vol. II. p. 105. 1853.

† *Thier Chemie*, p. 255.

‡ *Edinburgh Medical Journal*, Vol. I. p. 133. 1855. And *Ibid*, March 1858.

sheep and ox; and he found in the cerebrum, cerebellum, liver, lungs, spleen, kidneys, striped muscular, and connective tissues, bodies which in every way corresponded with starch—in their size and shape and concentric marking,—in their colouring with iodine, and in the phenomena they exhibited when viewed by polarized light. Indeed, no doubt can exist that these bodies were actually starch; the only doubt that remains is, as to how their presence is to be accounted for.

Were they really formed in the tissue? or, were they only accidentally present in the microscopical preparation?

Mr. Carter has no doubt that they were really formed in the tissue. He states that two varieties of them existed; the one resembling wheat starch, the other, the rarer kind, "corres-"ponding in every particular with the starch derived from "potatoes." He most frequently found them diffused through the tissues, either isolated, or aggregated and united by interposed homogeneous matter, but in some exceptional cases occupying the cavity of flaccid cells. This last is certainly a strong point in favour of their actual formation in the tissue.

On the other hand, M. Rouget has recently investigated the subject, and, from the number and extent of his experiments, many curious facts have been brought to light.

In examining the epithelial glycogenic papillæ of the Amnion of Ruminants, he found, when he had crushed the pulpy substance between his fingers, a considerable number of grains of starch; but he found that these grains were always on the surface, never in the substance of the epithelial layers. He suspected, from this circumstance, their origin; and he found that, even after repeated washings, the fingers deposited grains of starch on all surfaces, especially when they were moistened. M. Balbiani found, too, that the most frequent washings could not remove the starch grains from the hands. He therefore washed one of his hands in a solution of potash, and then covered this hand with a glove: at the end of eight hours he could not discover a single grain on the hand which had been thus protected, whilst on his other hand he found the starch grains as numerous as before. M. Rouget found starch grains also in the dust on the outside of windows, on roofs, on stones, in the layer of dust deposited daily in his laboratory, on glass slides exposed to the air; in

* Brown-Séquard's *Journal de la Physiologie*, No. V. p. 83.

short, on almost everything to which the ordinary air had access.

My own more limited experience tends to confirm that of M. Rouget: for I have certainly found starch granules in the dust on books, on glass slides, and in the scrapings of my hands, but in far less numbers than I was led to suppose from M. Rouget's description. Perhaps the rarity of corn mills in London may account for the difference. In microscopical preparations, too, I have occasionally found a few starch grains, but I never could persuade myself that their presence was other than accidental.

On the whole, I think it must be concluded that the presence of starch, as starch, in the human tissues, is a matter at present "not proven."

The formation in the healthy liver of a substance which is converted during life into sugar, even in animals fed exclusively on meat, is a fact which renders it probable that in certain states of disordered nutrition a substance analagous to starch may be accumulated in the liver and other organs. This substance, which has been named by M. Bernard*, Glycogène, exists in the hepatic cells, and may be obtained from them by pounding in a mortar the liver-substance of recently killed animals, and then treating it with boiling water, and adding to the expressed fluid four or five times its volume of alcohol; a precipitate instantly falls, and when this is boiled with a solution of caustic potash to free it from any nitrogenous matter and again purified by alcohol, there is at last obtained a white amorphous powder, of neutral reaction, without colour or taste, which becomes blue when brought into contact with iodine, but does not reduce the potassio-tartrate of copper nor ferment on the addition of yeast. This powder is soluble in warm water but not in alcohol, and is readily converted into sugar by all the agents which produce this transformation in starch and cellulose.

M. Rouget† has shewn that this substance is produced by other tissues as well as by the liver; that it is not produced by special glycogenic cells but by epithelial cells in general; he found it in epithelial cells of the skin, of the palate, of the tongue,

* *Nysten, Dictionnaire de Médecine*, Article Glycogène.

† *Gazette Hebdomadaire*, 1859, p. 266, and *Journal de la Physiologie*, No. VI. p. 312.

stomach, and intestines. From these and other facts he concluded that the presence of this matter must not be attributed to a special function of the elements of any tissue in particular, but that it must be regarded as a peculiarity in the constitution of the elements themselves, thus establishing one analogy more between the animal and the vegetable tissues.

Dr. Pavey* has recently endeavoured to shew that the conversion of this glycogenic matter into sugar does not actually occur during life, but that the sugar which is found in the blood of the right side of the heart is really due to a post-mortem change, for in the blood that he obtained from the right ventricle of living animals, by passing a catheter down the jugular vein into the right side of the heart, he found only a trace of sugar. His conclusions have however been disputed by later observers.

These facts shew that the formation of an amyloid substance in the human body is not only extremely probable, but that such a substance is really physiologically produced.

The proof of the actual existence however of an amyloid matter as a pathological phenomenon must rest upon other evidence.

Certain pathological conditions of the liver, spleen and kidneys, in which these organs became infiltrated with a peculiar wax-like substance, had been long known, and had been described under the name of Waxy, Lardaceous, or Albuminous Infiltrations; but neither the nature of the substance to which this change was due nor the exact anatomical seat of this deposit had been clearly defined until Virchow pushed his investigations in pursuit of animal cellulose into the regions of pathology.

In 1854† he shewed that this waxy degeneration of the spleen commenced in its malpighian bodies, that while the whole spleen became enlarged, indurated, and anæmic, it was at the same time studded through with dull, gelatinous-looking bodies, varying in size from a pin's head to a hempseed, more or less resembling in general appearance little granules of boiled sago; and that these bodies were due to a deposit within the malpighian follicles of roundish or somewhat angular granules, rather larger

* *Guy's Hospital Reports*, Vol. IV. p. 291. 1858.

† *Arch. für patholog. Anatom*, Vol. VI. p. 268.

than a lymph cell; and that moreover when a solution of iodine was applied to them their colour changed to a dark yellowish red, which on the addition of sulphuric acid passed into a deep violet. From these changes in colour he was led to infer that the substance was of a similar nature to that which he had described as cellulose in the corpora amylacea of the brain. Shortly* after this time he pointed out that changes of a similar nature occurred in many other organs and tissues, namely, in the kidney, liver, cartilages, intestinal canal, &c.

In his lectures on "Cellular Pathology†," he has given his last opinion as to the nature of the substance, its anatomical seat, and the character of the constitutional symptoms which accompany it.

His views are as follows:—

Almost all parts of the body are capable of undergoing this process of degeneration. The affected parts become enlarged, somewhat indurated and anæmic; the cut surface is semi-transparent, but dull; the natural colour of the parts is lost, but the colour of the neighbouring parts and vessels being seen through, gives them a yellowish or brownish tinge. The coats of the small arteries are the most frequent primary seat of this infiltration, and from them it spreads to the parenchyma of the organs; the walls of the arteries become thickened, and their calibre reduced, and hence the anæmic condition of the organs. The muscular fibres of the middle coat are the parts first affected. In the place of each muscular cell a compact homogeneous body is seen, in which, in the earlier stages, the centre of the nucleus appears as a hole; this afterwards disappears, so that a kind of spindle-shaped body remains, from which all trace of cell structure has vanished, no distinction being left between cell-wall, contents and nucleus. When the infiltration has reached this point, it commences to invade the parenchyma of the organs. In the liver, the cells in the immediate neighbourhood of the hepatic arteries are first affected; the liver-cells gradually become homogeneous; nucleus and cell-wall gradually disappear; and at last nothing is left but an absolutely homogeneous shining body; the cells are thus converted into a kind of corpora amylacea. In the kidney, the vessels of the malpighian bodies and the afferent

* *Arch. für patholog. Anatomie*, Vol. VI. p. 410; and Vol. VIII. pp. 140 and 364.

† *Die Cellularpathologie*. Berlin, 1858, p. 331.

arteries first undergo this change. In the earlier stages, but little alteration is perceptible to the naked eye; the kidney appears merely indurated and anæmic, and only when a solution of iodine has been applied to it, does the change it has undergone become apparent; then, throughout the cortex, numerous fine red dots appear, corresponding in their size and position to the glomeruli, and fine red streaks, running from them, indicate the afferent arteries.

The disease is constitutional; one organ alone is rarely affected; the only spot, where as yet an independent development of this change had been remarked, was in the permanent cartilages. The organs thus affected cease to discharge their functions. The patients assume a cachectic appearance, and gradually waste away. Dropsy frequently supervenes. Sometimes, too, the whole digestive tract is affected by this degeneration. During life this is rendered manifest by continual diarrhœa, and by diminished powers of absorption.

During the last four months I have in all met with four cases of this degeneration, two of them were affected by chronic albuminuria, and in these the kidney was the seat of the change; in the other two the liver was the organ chiefly affected.

I will now relate them,—

Sarah Ann P , æt. 24, was admitted into Hope Ward of St. Bartholomew's Hospital on November 14th. She was a servant, and had been in good health till about two years before her admission, at which time she had met with a fall and struck her back severely; this was followed by a severe illness attended with great pain in the abdomen; an abscess had formed internally, had burst, and had continued to discharge till about June last. About the beginning of last year she noticed that her feet began to swell towards evening; the swelling gradually increased and gained her legs and thighs. She had, it seems, never suffered from any acute renal attack, though she stated that some time ago the urine had become scanty, but she was certain that it had never looked bloody. She stated also that she had been more swollen a few days previously.

When admitted she had a fatigued expression, her face was pale and sallow, somewhat puffed under the eyes, pupils dilated, skin cool and dry, pulse 120, very weak, tongue red, raw-looking with aphthous ulceration on the sides; no appetite. Bowels open many times a day with watery motions; this diarrhœa had continued for a long time. She had no pain in the loins. She complained of no particular pain anywhere, but only of excessive weakness.

On the following day I examined the urine and found it copious, of a light amber colour, clear, depositing scarcely any sediment, of specific gravity 1008. On boiling and adding nitric acid a well marked cloud of albumen formed, but the quantity of albumen was small. On examining the sediment under the microscope, only a few kidney-epithelium cells, containing minute oil globules, were found, but no casts or blood-cells. The epithelium-cells presented no unusual appearances on the addition of iodine.

The diarrhœa persisted in spite of treatment. On the 29th a considerable amount of blood passed with each motion. She gradually sank, and on November 29th she died.

I made the post-mortem examination on the following day.

There was still a moderate amount of fat in the subcutaneous tissue.

The brain and its membranes were anæmic, but otherwise healthy.

Heart was small (5½ oz. avoird.); its substance pale, its valves quite healthy; in all its cavities very firm pale clots were found.

In the right pleura there were some old and very firm adhesions about the apex of lung, and in this part the lung tissue was found condensed around a smooth-walled cavity of about the size of a walnut. This cavity opened at its inferior and inner angle into the superior branch of the right bronchus, No recent tubercular deposit was found anywhere.

The liver was larger than usual (wt. 3lb 8 oz.), pale mottled, greasy, of diminished consistence, under the microscope found to be generally fattily degenerated. No reactions were obtained with iodine and sulphuric acid.

Spleen, 6 oz., apparently quite normal; no appearance of sago-like bodies.

The intestines were all matted together by old and firm adhesions, in the form of thin layers of connective tissue.

The walls of the intestines were thin and very pale; with the

exception of this exceeding paleness no change was remarkable in them by the naked eye until the lower part of the colon was reached; here there were a few small circular superficial ulcers: these increased in number as the rectum was approached. All the pelvic organs were matted together into a firm mass, and at the same time were united firmly to the walls of the pelvis—no doubt this was caused by an attack of pelvic cellulitis at the time of the formation of the abscess mentioned in the patient's history.

On brushing a solution of iodine over the mucous membrane of the intestines, innumerable little dark red dots, corresponding with the villi, appeared, and on placing on the part thus changed a drop of dilute sulphuric acid, the whole surface previously coloured by the iodine changed to bluish-steel colour.

The right kidney weighed 7¾ oz., the left 6¼ oz. The right measured 5 inches in length, 2½ in breadth, 1½ in thickness.

The capsules stripped off readily, leaving the surface smooth, and not torn. The surface was of a pale yellowish white colour, here and there irregularly depressed, the depressions giving the surface an uneven lobular appearance. Little, very opaque, white specks and streaks indicated fatty degeneration of the contents of the tubuli. The depressed portions appeared somewhat darker; in colour of a pale pink, and semi-translucent. A few stellate veins stood out on the surface of the kidney. On the cut surface—the pyramids and cortex were very pale. From the apex of a central pyramid to the circumference of the kidney measured $\frac{1}{2}\frac{6}{0}$ inch along a line drawn perpendicular to the base of the pyramid. Of this the pyramid measured $\frac{7}{1\,0}$; the cortex $\frac{9}{1\,0}$. The cortical substance was of a pale yellowish white colour—the portions occupied by the straight tubes of the cortex being semi-translucent: the intermediate portions occupied by the convolutions of these tubes were opaque white, with little dots and streaks similar to those seen on the surface of the organ. The malpighian bodies were scarcely apparent, but on brushing the cut surface over with a solution of iodine, numerous dark red dots instantly appeared, which by their size and position were recognizable as malpighian bodies.

On a microscopical examination of a thin section, those parts which to the naked eye had appeared most opaque (namely, the convolutions of the cortical tubes), appeared black. This black deposit was contained within the tubules, many of them being considerably swollen out by it. With a higher power this deposit was seen to consist of granular matter, set through with innu-

merable minute oil globules. Between the convolutions of the tubules the malpighian bodies were seen pale and semi-translucent, about $\frac{1}{70}$ of an inch in diameter, their capsules not thickened, but in parts dotted with oil globules. On examining with a higher power isolated malpighian bodies, which had become shelled out of their capsules, they appeared confused, translucent, colourless, and refracted the light very strongly, in this respect presenting an appearance very similar to minute calcified arteries of the brain; no nuclei were visible in them. (See fig. 2.)

On applying a solution* of iodine, the malpighian bodies changed to a bright transparent carmine (see fig. 3) colour, that is when seen by transmitted light (by daylight there was a tinge of orange in the red). The colour in some instances was seen to extend for a short distance into the afferent arteries of the bodies; and a few streaks, of a similar colour, were visible among the straight tubes of the pyramids. The other parts of the kidney were stained only of a pale brownish colour.

On the addition of a drop of dilute sulphuric acid, the colour of the malpighian bodies instantly began to change, becoming more of a port-wine colour, which gradually deepened till they assumed an indigo black. Those bodies on which the acid had acted less, still, however, retained a shade of violet. (See fig. 4.)

These colours were evanescent, and after a few days disappeared, leaving the kidney structure just as at first; but a fresh application of iodine reproduced the colours, but not so vivid as before. The same thing occurs with vegetable cellulose; if the preparation is left uncovered, the colours quickly fade away, and may be reproduced in a similar manner.

The second case that I met with was in the beginning of this year†. The patient, a gentleman twenty-seven years of age, had been known to be passing albumen in his urine for five years. Three years previously to this he had suffered from a slight attack of syphilis. Two years before his death his sight became troubled, and almost complete blindness followed. After a time, however, the sight of one eye was regained. Some months before his death he had a series of severe epileptiform convulsions, followed by coma. These convulsions recurred a fortnight before

* The strength of the solutions and method employed were fully described by me in *The Lancet*, December 24, 1859.

† For the particulars of the case during life I am indebted to Dr. Baly, whom I assisted in making the *post-mortem* examination.

his death. During his whole illness he had never had any dropsy, nor had he had any pain in the loins. The urine was abundant, contained a large quantity of albumen ($\frac{1}{3}$ to $\frac{1}{4}$ the test tube); and an examination of the sediment showed numerous pale, transparent, smooth casts.

On examination after death, the skull was found much increased in thickness, the frontal bone, measuring $\frac{1}{2}$ inch in thickness, very hard and heavy. The optic nerves were thin, and apparently atrophied. Some adhesions were found on the under surface of the anterior lobes of the cerebrum, uniting them to the opposite layer of the arachnoid. The brain was otherwise normal.

The right pleural cavity contained some bloody serum, and there were some recent adhesions at the base of the right lung.

The pericardium, too, contained about 3 oz. of bloody serum.

The coils of the intestines were slightly matted together by layers of soft, yellowish, somewhat puriform lymph.

The liver was considerably increased in size and weight; its surface was everywhere drawn into large globular nodules, varying in size from a nutmeg to a hen's egg. On cutting into the fissure between these, a dense cicatrix tissue was found. The interior of the nodules themselves was composed of ordinary liver tissue, somewhat softened, and here and there fattily degenerated. Scattered indifferently through the substance of the liver were numerous small abscesses (the smallest of them apparently limited to a single lobule), filled with dirty sanious puriform matter. No reactions were obtained with iodine.

Spleen was large, its capsule thickened, at its upper edge were two wedge-shaped purulent deposits.

Kidneys were very large and soft. On stripping off the capsule the surface was left extremely uneven, a thin layer of kidney tissue being torn off with the capsule. The surface was of a yellowish colour, mottled with dark red. The numerous little prominences on its surface were white and opaque. On section the cortex was found much thickened of an opaque yellowish white colour, here and there mottled with red; this opaque deposit appeared to extend some little distance between the tubuli of the pyramids. The kidney tissue was very soft and pliable, and when cut into a considerable quantity of bloody serum drained away.

On microscopical examination the intertubular structure of the cortex was everywhere greatly thickened, by a transparent homogeneous substance in which nuclei were present in great

numbers, and here and there minute oil globules. The convoluted tubuli appeared compressed, much reduced in diameter, and in parts so constricted, that some of them presented little varicose bulgings; their epithelium was changed into a mass of oil globules, in which no trace of cell structure was any longer visible. In parts the intertubular substance appeared distinctly organised into connective tissue.

The malpighian bodies appeared solid, compact, and homogeneous, and refracted the light strongly, and on the addition of iodine, first alone, and afterwards with dilute sulphuric acid, they exhibited changes of colour exactly similar to those described in the last case, with this difference, however, that the change extended for a much greater distance into the arteries. Arteries even of considerable size had very thick homogeneous walls, quite equal in thickness to the calibre of the artery; the whole thickness of the walls became coloured by the iodine, so that under the microscope they appeared as if injected with some red preparation.

The third case was a young man, æt. 21, who about eleven months before his death was admitted into John Ward of St. Bartholomew's Hospital, under the care of Dr. Jeaffreson.

At the time of his admission his right pleural cavity was full of fluid. After some time a large quantity of pus was let out by paracentesis, and from time to time a discharge of pus issued from the opening thus made.

He slowly sank, and on December 21st he died.

I made the post-mortem examination on the following day.

The right pleural cavity was distended by about two pints of pus.

No trace of tubercle was found in either lung.

The liver was large, weighed 4lb. 10 oz. was considerably displaced to the left by the accumulation of fluid in the right pleural cavity, and was firmly adherent to the diaphragm on the right side, to the abdominal parietes anteriorly, and to the right kidney inferiorly, its peritoneal covering in these situations being much thickened.

Its edges were rounded. On section it was found very firm, cutting crisp and brawny. The colour of the cut surface was pale, pinkish, and semi-transparent, the lobules were distinctly marked out by white lines. The very centre of the lobules too,

immediately around the hepatic veins, exhibited little white opaque dots and streaks, the intermediate zone being semi-transparent. On the convex surface of the liver, the liver substance was opaque, white, soft, and greasy; this condition extended for about an inch into the substance of the liver, and terminated by a very irregular line.

A solution of iodine brushed over the cut surface instantly stained the transparent portions a deep reddish brown, leaving the opaque white parts unaltered. Some of the lymphatic glands in the gastro-hepatic omentum gave similar colours with iodine, but the intestinal mucous membrane and the kidneys remained unaltered.

The spleen too appeared perfectly normal.

A microscopical examination shewed that the opaque white lines surrounding the lobules and the specks in the centre were caused by a great accumulation of oil globules in the hepatic cells. The transparent portions yielded white translucent cells, strongly refracting the light, many appeared quite homogeneous and solid, other faintly granular, while some contained a few oil globules, and a nucleus. These changed to a carmine red with iodine, but the violet blue, with sulphuric acid, was not well marked, as the dilute acid produced but little change, and the strong acid acted too energetically.

The fourth case was a little boy æt. 10, one of my out-patients at the Children's Hospital. When brought, he looked very ill and pale. His mother told me that his illness began about ten months previously with an attack of rheumatic fever, and that since that time he had never regained his health. I admitted him into the hospital.

He complained of vague pains in his limbs, his pulse was quick and feeble, his tongue furred.

He gradually became feebler; symptoms of peritonitis manifested themselves; his breathing became hurried and oppressed. The left arm became suddenly œdematous; he began to vomit dark fluid, like coffee grounds, and ultimately blood,—and so died.

I made the post-mortem examination fourteen hours after death. There was no post-mortem rigidity. The left brachial vein was distended by a firm dark clot, to which it was firmly adherent for a distance of about two inches.

There was a considerable quantity of turbid serous fluid in the left pleural cavity.

The apices of both lungs were solid and dark-coloured. The pericardium presented no sign of inflammation. The heart was large; its valves, with the exception of the mitral, on which there were some small firm granulations, quite healthy.

Some recent, yellowish, puriform lymph was smeared over the serous surfaces of the intestines. Spleen was firm and dark, but not enlarged; its malpighian bodies were very perceptible, but unaltered by iodine. The liver was large, weighed 2lb. 9 oz. (avoird.); the substance firmer than natural; on section there was seen throughout it little white semi-transparent streaks, not bounding, or limited to, the circumference of the lobules: these transparent parts changed to a deep red brown colour on the application of iodine; the other parts remained unaltered.

The kidneys were pale and firm; with iodine they gave no change of colour.

The stomach and intestines were full of dark discoloured blood; no lesion of their mucous surface was perceptible.

These cases are very far too few in number to admit of any general deductions being drawn from them. But it is remarkable that in all of them there had been a long debilitating illness, in three of them attended with a constant draining discharge. This coincides with the experience of Professor Virchow*, who pointed out the frequent coincidences of this degeneration with diseases of the bones; and with that of Dr. Bennett†, who has shewn its frequent connection with phthisis.

Another remarkable circumstance is that in both the cases of the kidney affection, although the malpighian bodies and the arteries leading to them were the seat of this degeneration, there was an almost constant excretion of a large amount of watery urine. If in a more extended experience this should be found to be invariably the case, might it not lead to some modification in our views of the functions of these organs?

In both the kidney cases, too, this degeneration was accompanied by extensive alterations in the tissue of the other parts of the kidney.

Another circumstance, which, however, may be only an accidental coincidence, is, that in two of these four cases there was found after death those general inflammations of the serous membranes, in one accompanied by secondary purulent deposits, in the other with obstructions of the veins, which indicate the existence of blood poisoning,—the so-called pyæmia.

* *Arch. für patholog. Anat.* Vol. VIII. p. 364.

† *Principles and Practice of Medicine.* Edinburgh, 1858. 2nd edit. p. 760.

In considering, then, the nature of the substance to which these appearances are due, it is evident that we have to deal either with two substances or with two modifications of the same substance. The one, the material which forms the corpora amylacea of the brain, and the very similar prostatic concretions —for in the prostate gland there are very frequently found little oval or round concentric bodies, which assume a blue colour on the addition of iodine alone, just as the corpora amylacea do; and the other, the substance which is met with in the so-called waxy degeneration of the different organs.

If we inquire, then, how far these substances are capable of fulfilling the three conditions necessary to establish their perfect analogy with the substances of the amylaceous group, we are at once met by the difficulty that, owing to mechanical obstacles, we are not yet in possession of any exact analysis of their ultimate chemical composition.

Schmidt* has indeed attempted their analysis, and the results he has obtained are not favourable to the view that they are of an amylaceous nature. He made two analyses: the one of a choroid plexus rich in corpora amylacea, and the other of a waxily degenerated spleen. In the first he admits that, owing to the small size of these morphological elements, their mechanical separation from the surrounding tissues was impossible, and their chemical separation could be only very imperfectly attained, so that an elementary analysis could only give a partial explanation.

The argument which he follows may be thus briefly stated:—

Albuminous matters, such as albumen, fibrine, caseine, epithelial, and muscular tissue, &c. contain

Of Carbon,	from 50.	to 54.	per cent.
„ Hydrogen,	„ 5.7	„ 7.3	„
„ Nitrogen,	„ 15.	„ 18.	„

* Ueber das sogenannte thierische Amyloid.—*Annalen der Chemie, Sogenannte* Vol. CX. p. 250, 1859.

Gum, starch, and cellulose, the formula for which is $C_{12} H_{20} O_{10}$, contain

Of Carbon, . . . 44.4 per cent.
„ Hydrogen, . . . 6.1 „

If, then, in any mixture of these amyloid substances with albuminoid matter, the proportion of carbon sinks below 50 per cent., of hydrogen below 6.7, and of nitrogen below 15 per cent., and at the same time glucose is formed by treatment with sulphuric acid, these amyloid substances would belong to the second or starch group. If, however, the proportion of these elements rises above this per centage, and especially if the nitrogen rises above 15 per cent. these substances will belong to the albuminous class.

In the choroid plexus, after boiling it for half an hour with dilute sulphuric acid, he obtained no trace of sugar, either by the copper test or by yeast. He subsequently treated the plexus with concentrated sulphuric acid, in a manner similar to that described in the conversion of chitine, but without obtaining any traces of sugar.

From the spleen, too, he was unable to obtain any evidence of the presence of sugar. In an analysis of this spleen substance he found that the per centage of nitrogen = 15.56.

He concluded, therefore, that these substances were nitrogenous substances, to be classed with albuminous matters, and not with the non-nitrogenous hydrocarbons.

Virchow, too, admits that he has been unable to form sugar from either class of these amyloid matters.

The analysis above detailed is far from being completely satisfactory, and until a more extended series of experiments has been made, it will not be possible to arrive at any definite conclusion as to the ultimate composition of these substances.

In the absence of a complete analysis, are there any other relations of these bodies which may help us to form an opinion as to their nature?

In the *First* place, they possess a great resistance to putrid decomposition. Virchow found that after subjecting a spleen thus degenerated to a stream of water for three weeks, the amyloid granules were apparently unchanged.

Secondly,—In warm water the corpora amylacea of the brain swell up, and ultimately dissolve.

Thirdly,—They are insoluble in æther and alcohol.

Fourthly,—They offer to the actions of the alkalies and acids a far greater resistance than albuminous matters do; though, indeed, a less than is exhibited by vegetable cellulose.

And, *Lastly*,—They exhibit certain colours, when brought into contact with iodine and sulphuric acid.

The colour produced by iodine alone on organs which are the seat of this pathological process is perfectly distinctive. When, for example, a solution of iodine is brushed over a liver which has undergone this change, the affected parts in a few minutes assume a deep red brown colour, very different from the colour produced by iodine on organs in any other condition,—once seen it cannot be mistaken. In the last five months I have made or superintended more than sixty post-mortem examinations. In by far the greater majority of these I have applied iodine to the different organs; indeed, it has been my rule to do so, whether this degeneration has been suspected or not—(only by inadvertence have I omitted it)—and only in the four cases related have I seen this peculiar colour. In these cases too the colour was precisely similar to the colour which Professor Virchow showed me at Berlin in the summer of 1858.

One cause of confusion however exists. Two, if not more very different pathological states have been confounded together under the name of waxy degeneration.

Not infrequently the malpighian bodies of the spleen are seen enlarged, forming small round white bodies, varying in size from a pin's head to a hemp seed, these are not so semi-transparent —not so sago-like—as the amyloid-degenerated corpuscles. Occasionally too the spleen is found occupied by numerous largish opaque white, or whitish yellow, irregularly-shaped, hardish masses. In two cases which have recently come under my observation this condition of the spleen was associated with great general enlargement of the lymphatic glands; both of these cases were boys of the ages of eight and twelve respectively; in one there was a considerable increase of the white corpuscles of the blood, in the other the blood, owing to an accident, was not examined. In neither case was any change of colour in the affected organs perceptible on the addition of iodine.

When to parts which have become thus reddened by iodine sulphuric acid is added, a change to a bluish red, or violet red, or deep blue purple, or even to an indigo black, speedily commences; in some cases this colour quickly passes into a deep reddish brown. In the malpighian bodies and arteries of the kidney the bluish coloration is most marked, and in these the

dilute acid is sufficient to produce it, in the liver stronger acid is necessary and the colour is obtained with greater difficulty.

This blue coloration is so distinctive of amylaceous matter that up to the present time only one other substance has been found to exhibit it. Cholesterine when treated with strong sulphuric acid and iodine shows a very similar blue colour.

From this Meckel* was led to conclude that the substances in question were cholesterine or modifications of it.

From cholesterine they differ however, as Virchow has shewn, in many important particulars.

First,—Cholesterine is unchanged in colour by iodine alone.

Secondly,—The corpora amylacea dissolve in warm or boiling water. Cholestine is insoluble in water.

Thirdly,—Cholesterine melts on the application of heat; the amyloid bodies do not melt but only dry, and still give the same reactions with iodine.

Fourthly,—Cholesterine dissolves into a brown fluid on the addition of concentrated sulphuric acid. These other bodies swell but do not dissolve with a change of colour.

Fifthly,—Cholesterine is soluble in ether, amyloid not.

That, therefore, these substances are not cholesterine is abundantly manifest. One striking peculiarity in this reaction of cholesterine is, that the blue colour is not produced until the cholesterine has begun to oxidize under the influence of the acid. The formula for cholesteric acid is $C_6\ H_4\ O_4 + HO$, a formula much resembling the formula of the starch group. Is it not possible that this exceptional instance may indeed become an additional proof?

Are, then, those bodies found in the brain starch?

The pale blue colour with iodine alone differs considerably from the colour which starch assumes. And though sometimes the phenomena they exhibit with polarized light much resembles those exhibited by starch, yet until these bodies have been shewn to be convertible into sugar, I think we must answer this question in the negative.

Is the substance found in these amyloid degenerations of the different organs cellulose?

It differs from cellulose in the red brown colour produced by iodine alone, and by its less resistance to the action of alkalies.

---

* *Annalen der Berliner Charité-Krankenhäuser*, reference from Virchow's *Arch.*

All attempts, too, to convert it into sugar have, up to the present time, been unsuccessful.

So that, I think, all that we are justified in concluding is,—

That the reactions of these substances with iodine and sulphuric acid indicate their analogy, not their perfect identity, with the substances of the amylaceous group.

DESCRIPTION OF THE PLATE.

Fig. 1.

Epithelial cells, from the cortex of the kidney of Case 1.

Fig. 2.

A malpighian body, from the same kidney, in a state of amyloid degeneration. (250 diameters.)

Fig. 3.

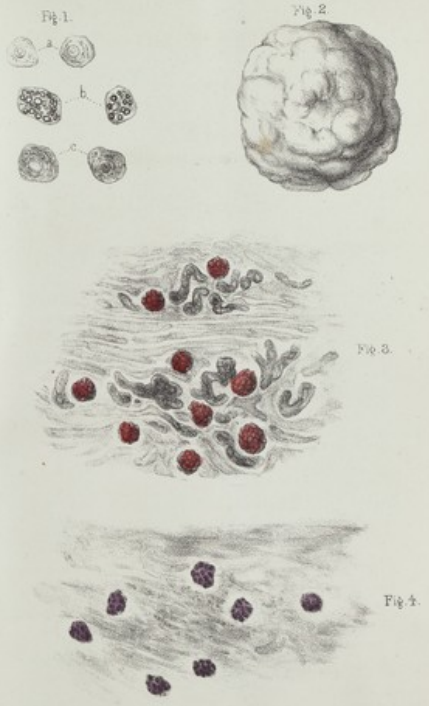
A thin section of the same kidney, treated with a solution of iodine. The malpighian bodies are seen of a bright red colour.

Fig. 4.

Another section, treated with iodine and sulphuric acid. (inch power.)

The drawings were made by Mr. Godart, from microscopical preparations.

WESTMINSTER:
PRINTED BY THOMAS BRETTELL, RUPERT STREET, HAYMARKET.



HABIT:

PHYSIOLOGICALLY CONSIDERED.

A Lecture,

DELIVERED MAY 9TH, 1853,

AT THE BRISTOL LITERARY AND PHILOSOPHICAL INSTITUTION,

BY

JOHN ADDINGTON SYMONDS, M.D.

CONSULTING PHYSICIAN TO THE BRISTOL
GENERAL HOSPITAL, ETC. ETC.

Reprinted from the PSYCHOLOGICAL JOURNAL *for July*, 1853.

LONDON:
JOHN CHURCHILL, PRINCES STREET, SOHO.
EVANS & ABBOTT, 29, CLARE STREET, BRISTOL.
1853.

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS STREET,
COVENT GARDEN.

A LECTURE,

ETC.

I PROPOSE on this occasion to invite your attention to a class of phenomena, generally designated by the word Habit. This word, as you are aware, is one of various meanings; and yet through them all you will find a thread of connexion. It denotes that which has been observed so often in a living organism, that it is regarded as having something of permanence or constancy in a being which is evermore liable to change. It sometimes implies a cause; as when we notice that a friend has become stout or thin, and our suspicion is put down by the remark—"Nay, but he is of a full or spare habit,"—which means, that there is something in his original constitution, whether derived from inheritance or noted in an early period of his history, which is considered to be the cause of what has become the ordinary condition of this person's frame as to plumpness or leanness. Or it simply expresses a mode of action, the final or ultimate explanation of which is simply that it has occurred so often as to be always looked for. A stamp of permanence has been given to what at first might have been regarded as casual and temporary—that is, begotten of the circumstances under which it first originated, and to be expected to recur only under like conditions. This reference to habit as something ultimate or final, springs from the tendency in the human mind to acquiesce in the customary. From the unreasoning peasant, who quietly tells some traveller, startled by what he conceives to be a foolish or even outrageous mode of action, *that it is the custom*,—to the philosopher, who, unable to discover any more remote antecedent to the chain of sequences under his survey, says, that such is the natural order of events, the mental tendency is the same. A thing familiar is customary, and what is customary is acquiesced in. Nay, there is a tendency to imitate it passively. And what was an individual trick or habit, becomes a fashion or social convention.

Habit is sometimes used in a more vague signification, as when we

speak of the habits of animals or their customary actions, and when it would be more correct to say, instinctive actions. But in this case, the habits are referred to as the effects of the instincts. And if the phrase "Instincts and Habits" of animals be used, it is intended to distinguish that mysterious principle called instinct, from the action which it prompts. Not, however, that habit in its strict sense may not be predicated of the lower animals; for as they are the subjects of volition and reasoning processes to a certain extent, so also they may be subject to habit.

It is not uncommon also, to speak of the habits of nations or races, meaning thereby, such modes of life and action as are usual to them. Perhaps in all the senses of the word, we shall find a connexion with its etymology, and that it bears reference to something which has been held (*habitus*) retained after being acquired; something added to the individual, and henceforth always associated with him. Again; *dress* is a *habit*, and *costume* passes into *costume*. But we have to treat of habit in a more restricted sense, or as I have expressed it in the title to this lecture—"Habit physiologically considered." As physiology is the science which treats of vital actions, so we have to investigate habit with reference to these, or at least to some of them. We shall consider it in reference to those which constitute the life of relations in man—those functions which place him in relation with the outward world—sensation, motion, and thought. But while each of these heads will have some discussion, the greater part of our survey will be devoted to habit in relation to motion.

Some of the most curious examples of actions resulting from habit are to be found among those which we generally associate in our minds with the prosecution of some purpose, the relief of some feeling, the expression of some emotion, and yet in these cases there is no such final cause discernible. "Why does Mr. Thunderton speak so loudly and angrily? does the occasion call for it?" "Oh, that's his way—it is a habit he has got." "What does Mr. Scrutator find so very remarkable in us? Do you see how he fixes his eyes upon us?" "Oh! that's nothing—he always does so—it is his habit." "Don't you pity that poor gentleman? he is obliged to cough between every clause of his sentences." "Oh, no, he is quite well. We call it his interjectional or ejaculatory cough; and very expressive it is, like that of one of the fictitional characters of the day; it is a habit he has fallen into." "That person must surely be very well—very giddy.—See how he sways about in his walk—he will surely fall." "Oh! no,—he always walks in that way—he got the habit early in life." "What is that gentleman in search of? Do you see how he examines everything on the table and mantle-piece? What is he looking for?" "That shows you don't know him! We never notice it. He has had that habit as long as we have known him, many years ago." These cases might be multiplied indefinitely. In each you observe that an action which appears to a stranger to be generated by a particular occasion or circumstance, is by one familiar with the subject of it explained, and seemingly quite to his satisfaction, as being an *habitual* eccentricity. That action, or manner, or mode of expression which strikes

us as odd and exceptional, has in the observation of others occurred so frequently as to be considered necessary to the individual, the result of his acquired bodily or mental constitution. It is, in common phrase, "second nature,"—habit.

We are not, however, about to spend much time upon these abnormal instances. For in the category of Habit must be placed a great number of our most ordinary and familiar actions. Such are those actions which have become secondarily instinctive, or automatic;—those wonderful acquirements which we call standing, walking, running, grasping and handling, speech and talking, and those less universal but still not uncommon accomplishments of swimming, climbing, dancing—all the manual arts or handicrafts, and the fine or æsthetical arts;—all these belong to habit, as a principle of action, no less than do those actions which we call mechanical, and those aberrant movements which are designated nervous tricks and fidgets.

Habit in Relation to Motion.

The function of muscles is, as you are aware, to move some part or the whole of the body, or to impress some motion on surrounding objects. A muscle is a bundle of fibres, which, in shortening themselves, bring their two ends nearer to each other. For various reasons, which we have not time to consider, several muscles act together in effecting particular movements. It is obvious, therefore, that they must act harmoniously, and to effect this they are under the governance of a central impulse. The muscular fibres are called into action by motor nerves, and the impulses in these several motor nerves are associated or derive a unity of origin from a nervous centre, as it is called, a part which receives an impression or imparts an impulse. Within the compass of the spinal cord and the brain, an immense number of these centres or ganglia are congregated, and so closely packed together as to seem to be one substance.

Some movements in the body are unaccompanied by sensation, and not instigated by the will;—these are often called the involuntary, and sometimes, instinctive movements. All the mechanism consists in the transmission of an impression by an afferent nerve to the ganglion which originates an impulse in the motor nerve. These movements may occur in an animal deprived of consciousness, or the susceptibility of feeling, as well as in one possessed of it. Thus, suppose the spine of a frog has been cut across the middle, if you pinch the extremity of one of the hinder limbs, the whole limb will be retracted. We are constantly the subjects of many such actions; one of the most striking examples is the series of muscular actions employed in breathing—a wonderful congeries of muscles which open the aperture of the windpipe, expand the chest, and so admit the fresh air, and afterwards expel the air which has yielded its oxygen to the wants of the blood; all these are put into action by afferent nerves, which convey an impression to the central respiratory ganglion, which sets in motion the requisite muscles. These movements are often accompanied by sensation, and may be altered, to a certain extent, by the will; but they are quite capable of going on, and in the healthy state they always do go on,

without the one or the other, as we know at the present moment. Were other proof needed, it would be enough to observe the breathing in a person profoundly asleep or rendered insensible by disease, or in an animal deprived of those parts of the nervous system which minister to sensation and volition. This class of automatic movements has been called of late years reflex, or excito-motor.

There is another set of movements which are prompted by sensation, and are yet independent of the will. They are designated by some physiologists as consensual. Dr. Carpenter uses the term sensori-motor. I shall employ the term sensational; and, further, I think it desirable to discriminate these movements into two subdivisions—the subjectively sensational, and the objectively sensational. Now what do we mean by subjective sensations as distinguished from objective? An objective sensation is one that gives us information of something in the outward world, something that does not involve our consciousness or remind us of our own individual existence. I see a green meadow, or hear the song of a bird, or smell the fragrance of a flower, without my mind being cognizant of anything but the objects which have excited the sensations of vision, hearing, and smell. These, then, are objective sensations. The subjective are those in which the consciousness is involved, the *ego*, the individual self, partially or exclusively. I touch a cold marble: while I recognise the marble as the cause of my feeling, and attribute to it a certain quality which gives me the feeling, my consciousness, or an affection of me, myself, as the subject of the outward operation, forms a part of the sensation. But this is a mixed example, and might be called an objective-subjective sensation. Take another example:—I feel *ill*. I do not know what is the matter with me,—I have no pain. I am surrounded by pleasant objects, and within reach of everything likely to make me happy; but I feel ill. This is an intensely *subjective sensation*. Take one more example:—I have a headache, *i. e.*, I feel pain and I refer the pain to my head. This is a mixed sensation;—it is subjective so far as the feeling of pain implicates the ego; it is objective with reference to the seat of the pain; that is, the part of the outer world constituted by the body. It may seem strange to talk of a person's own body as being external to his conscious identity. But if you consider the matter fully, you will find that the knowledge of our bodily organs is an acquirement, and not a part of the feeling of personal consciousness. A young child suffering pain in some part of the body, is often quite incapable of saying where the pain is seated. In disturbed states of mind, the separateness of subject and object is very well shown with reference to these bodily feelings. I have known a person in delirium say to a bystander, "You have a dreadful headache, sir." *He* was the sufferer, but his morbid intellect linked the pain with the image of the person before him, instead of with his own body.

These sensations may then, I have said, whether subjective or objective, have motions immediately related to them; responding to them, as directly as those which are instigated by the will—often coincident with, and controllable by the will—but still having a causation of their own.

As an example of subjectively sensational motions I may adduce the act of coughing. A tickling sensation is felt in the wind-pipe; a series of muscular actions ensue, vehemently forcing the air out of the chest, and often not to be restrained or prevented by the will. Winking the eyes is another example. The movements which preserve the equilibrium of the body are mainly prompted and guided by subjective sensations, with but little and often no assistance from voluntary efforts. This governing feeling of equilibrium is directly related with pressure on the nerves of the feet, or whatever parts are supporting the rest of the body. If that compression be removed, giddiness, or a feeling of loss of balance is the result; and in an instant such muscles are thrown into action as tend to restore the equilibrium.

Of the objectively sensational movements, no instances are more striking than those which are associated with vision. Close one eye of an infant, and mark the movement of the eyeball as you shift the light before it—then let both be exposed, and see the admirable concert of action between them—and when you are acquainted with the muscles and their nerves that effect these movements, how nicely they are adjusted and balanced against each other, and with what delicacy they mutually respond, your admiration is great indeed. Though these actions are thus immediately related with vision, they may be, and very frequently are, the servants of our will, as when they fulfil our wish of looking in particular directions, or at different objects. But, in these cases, motion does not enter into our consciousness. The object of our desire is the sight of something. We *will* the seeing of it—that is, we look at it, inspect, examine it, &c., but we are unconscious that, in order to this, certain movements of the visual organ must take place. Here then is a palpable distinction between this class of movements and the voluntary: for in the latter, whether we know anything or not of the muscular instrumentality, we are conscious that our wish is to be executed by a movement either of the whole, or of a part of our frame.

It is in this group of sensational motions that our actions are most closely analogous to those which subserve the instincts of animals. They are as *blind*, that is, as dependent on motives involving no foresight, no prearrangement of our own. They are as perfect—for we neither learn them nor teach them—they are the works of an art unteachable, untaught—and they were as complete in our first parent, when his eyes feasted on the natural glories of Eden, as in his latest descendant, inspecting the wonders of art in the Crystal Palace.

Another class of movements are those which give outward expression to the emotions. These also occur without any intention or volition, whether they consist in the play of the features, or in the gestures of the limbs; joy, sorrow, anger, complacency, fear, courage, confidence, distrust, speak in the quick turns and ever-shifting hues of the eyes, in the varying surface of the cheek, in the expansile nostril, the flexile lip, and the smooth or knitted brow. To this head also belong those vocal movements which give utterance to particular feelings, and are common to all ages of the human being, and to all animals possessed of vocal organs. The screams of pain, the shrieks of terror, the sigh of

grief, the yell of resentment, the shout of joy, are the instantaneous productions of these passions. The emotions which approach nearer to intellectual conditions are also related with actions altogether involuntary. Such are the perceptions of the beautiful, the sublime, the wonderful, and the ludicrous.

These emotional movements are interesting in a point which has not received much attention. The feelings prompt the movements; but it is no less true that the movements excite the feelings. And this, I think, is the key to some of those curious phenomena, often thrown into the vague category of sympathy. A sudden panic in a multitude, a burst of enthusiasm, will spread like lightning. The individuals look round and see countenances and gestures expressive of a particular emotion, of which, in the very process, they themselves become the subjects. But you may say this is nothing but the instinctive reading of nature's language of gestures and featural expression. The best instance is in those anomalous states of the nervous system, observed in persons subjected to mesmeric processes. Here the individual partially asleep, and with his will in abeyance, is at the mercy of any association suggested from without: close his fists and put his arms in a menacing attitude, and the emotions related with such movements are excited.* Passive imitation comprehends movements very analogous. Yawning is the expression of weariness. The sight of yawning engenders the related feeling, and our own muscles answer to it.

Our next class of movements would be those which are directly prompted (still without volition) by ideas; meaning by this term, remembered sensations and thoughts, as distinguished from those objects of consciousness which are immediately brought from the outer world. But as the best illustrations are derived from the group of allied movements which we shall have to consider as pertaining more particularly to the topics of this lecture—that is, the secondarily ideagenous—I shall not now adduce them. I must content myself with remarking, that in various kinds of passive imitation we have specimens both of sensational and of ideagenous movements. You know how readily some persons contract a particular tone of voice from association with others. Now as the individuals often wish to avoid the infection, and yet do not escape it, the imitation is clearly passive and mechanical—the vocal movements which give the particular tone respond instinctively to the sound which has been so often impressed on the ear. But take a case of unintentional mimicry: you must have observed that when any one is relating an anecdote respecting the sayings or doings of an individual who has marked personal peculiarities, the narrator's face, tones of voice, and gestures, will represent the individual, though the operator has no intention of so taking him off (as the phrase goes). The idea of the individual is so vividly before him,

* The explanation of some of these strange mesmeric phenomena is admirably set forth in Dr. Carpenter's "Human Physiology." The chapter on the Physiology of the Nervous System in the last edition, abounds in views highly original, and stated and illustrated with wonderful perspicuity and affluence. The members of this Institution should be proud of this production of one to whose voice these walls have so often echoed.

that he cannot help this imitation; and consequently his vocal muscles, those of featural expression, and those which govern the gestures, the gait, carriage, and demeanour—all obey the mental impression, without the intervention of any volitional action.

Let us now inquire into the distinction between automatic and volitional action. We have seen that in the several groups comprehended under the former, motion succeeds to impressions, without sensation or with sensation, to emotions, and to ideas; but that there is no indication in any of these instances of the self, the conscious ego, being the originator of the movements. The common statement is, that we will the action of certain muscles or groups of muscles, in order to execute our purposes. But this is an incorrect statement; for it is obvious that in childhood, and indeed at all ages, unless we have learned anatomy, we know nothing about the mechanism whereby we move our limbs. Nay, after we have acquired such knowledge, we cannot make use of it in the manner supposed. I cannot, by an effort of my will, cause the contraction of that muscle which is the chief agent in bending the elbow—yet that muscle may, in morbid spasm, be thrown into single and separate action. But, will the bending of the arm, and it is done. In many of what we call voluntary movements, we do not even will the action of particular limbs. Thus, in learning to walk, the infant has no notion of limbs, of planting the foot on the ground, keeping one leg stiff, rotating the body on the hip-joint, &c. It only wishes to move to some place or person in view. The motions ensue upon the desire, guided by the subjective sensations of equilibrium, and by the objective sensations of vision.

The more we consider the subject, the more plain will it appear that the exercise of volition consists in maintaining an association between certain ideas, sensations, and motions; the individual self being conscious of its relation as the cause of those actions, and of its power to increase, or lessen, or interrupt, or arrest them. If we wish to analyse the process in any given case, we must be careful, for reasons which will soon appear, to select one that is not an habitual or mechanical action. In learning to play upon a musical instrument, there are many movements to be acquired which are quite new, and would, perhaps, never be required at any other conjuncture in our lives. Or to take a less uncommon case. You have to lift the third finger alone, while the others are kept flat on the keys of the piano. The master tells you to practise it,—and what do you do in practising it? You keep your mind on the end in view,—the raising this finger so reluctant to act alone, vehemently desiring to be successful, and sooner or later, if you are free from any physical defect, your ambition is realized. This little achievement seems a simple affair, but it is really rather a complex one. It is not the mere lifting of the finger, it is keeping the other two down; for the finger in question is raised by a muscle with tripartite tendon, of which two divisions go to the second and third fingers, and therefore the tendency is for all three to move together when the muscle is set in action. To prevent this, the muscles that place the first two fingers are put into action so as to antagonize the extensor and keep them down, while the action of that extensor is allowed to tell on the third only.

But of all this the successful young lady is unconscious. She has only, by diligent application of her mind, kept the object steadily in view, and a wonderful association of muscles has done her bidding, she is unconscious how; she knows no more of the process than if she had called to her aid some of the fairies or genii of her nursery fables.

The process in volitional movement is well illustrated by what takes place in imitating an instinctive action—as in acting or forcing a cough. We conceive the idea of the act of coughing with the will of producing the act; and this follows with more or less success according to the vividness of the conception, and the readiness with which in the individual, ideagenous motions follow the idea. In one who has histrionic genius, there is, besides the higher mental faculties, a quickness of response in the muscles to the ideas in the mind, as well as to the emotions.

But perhaps this subject may be further elucidated by considering what takes place in volitional efforts of thought. Much of our thinking is done mechanically—automatically—one thought suggesting another by the laws of casual association. But what is implied in such phrases as mental effort, application, undivided attention, painful abstraction of thought? There is a close analogy between this process and muscular exertion. They are analogous in the circumstance that they for the time engross our consciousness, that we feel a strain, an exertion, which, but for some ulterior object, we should be inclined to relax, and that, after it is over, we may be conscious of fatigue or exhaustion.

If you watch a child who, under the stimulus of emulation, or the fear of punishment, has been laboriously conning a task but just within the reach of its faculties, you may mark in the countenance, the skin, the pulse, the whole demeanour, effects like those produced by excess of bodily exercise. In both cases there has been an undue consumption of force. Wherein consists this mental labour? It is the forcible bringing together and keeping together ideas or signs of ideas, which have no natural tendency to cohere, or are not helped to cohere, by any other process than volitional effort. Take the instance of a child's committing to memory a rule of grammar, the meaning of which is altogether dark to him. I remember one, who before he was five years old, sat down to learn off the Latin Accidence (prompted by ambition—an elder sister having just been initiated into its mysteries), and he worked away at that luminous sentence—"A noun is the name of whatsoever thing or being we see or discourse of." Plain as this seems to us, it was to him only a series of words which had to be impressed on his mind, and remembered in the order in which they followed each other. And how did he achieve this wonderful acquirement? He read them over and over again—sometimes in silence—and then those cabalistic verbal signs were associated as visual remembrances. He read them aloud, and certain tones were associated with them,—reproductive of each other. The words contracted relationship, not only with the part of the page on which they stood, but with the part of the room where he sat, with the furniture near him, and with the emotions of which he was the subject, as hope and fear fluctuated in his anxious ambitious bosom.

At last the lesson was learned; that is, the words would come back

into the mind in the series in which they had been seen in the book. They were no longer held together by mere sight and volitional attention, but the occurrence of the first brought the second, and the second the third, because they had now very often co-existed, or proximally succeeded each other; and they had come to be associated with the images of other visual things, the recurrence of which was easy, by virtue of the liveliness of the original impressions.

To *attend*, or perform the mental act of attention, is to keep before the consciousness, by an effort of the will, particular impressions or trains of thought, or muscular actions. As to sensation, impressions excite the consciousness, and become sensations when the consciousness is not too closely associated with some other impressions or thought, or when they are new or very vivid. It seems to be a law, that consciousness does not link itself with any impression frequently repeated, unless it be accompanied by a feeling of pleasure or pain, or associated with some strong emotion, or with a train of thought associated with such emotion, (in common language, some interesting subject). The first time you sit down in a room close to a railway, and hear the roaring of a train, you think that it will arrest your attention every time it occurs, but when the novelty has ceased, you cease to hear it. It is not associated with any emotion or interesting series of thought; it is the passing of a train and nothing more. Or suppose you are at a dinner party. There is an incessant clatter of knives and forks and changing dishes, tramp of servants, and hubbub of voices, and yet you hear nothing but the words of your friend beside you, who is engaging you in some interesting dialogue. The consciousness at that time coheres to your friend's speech, fastened by the attractive force of present emotions, and is not to be torn from it by mere sensory impressions, unless these come reinforced by the divellent affinities of stronger emotions, or unless a voice pitched in a higher key than the confused Babel around you compels attention by its novelty. But the laborious action of the will in attention consists in enforcing a coherence between the consciousness and certain objects which are not bound to it by emotional interest.

But if there be labour or difficulty in this volitional effort, there is still more in that which has for its object the conjunction of past sensations or ideas, to the exclusion of the objects of sense. Suppose a young person shut up in a room and set to write down his notion of the character of Alexander of Macedon, and suppose, if it be possible, that the youth has never taken any interest in that magnificent hero—to measure the efforts he has to make you must consider what objects would be most likely to present themselves to his consciousness were he not to endeavour to control his thoughts. The things in the room, furniture, prints, &c., or should there be nothing of interest within, then the view from the window;—escaped from these temptations he has to resist the attractions of memory, some sport, or entertainment, or curious book, or some prospective pleasure. He has to keep before his consciousness the image of Alexander, and all the cluster of actions and sayings associated with that idea through books, lectures, &c. No difficult task; but he has to arrange and parcel these recollections in con-

nexion with certain qualities of Alexander's mind, and clothe them in appropriate language. These connexions are not so spontaneous as the others, and require more volitional effort.

The effort in *recollection* consists in detaining before the consciousness some idea about which the wished for ideas will cluster by association. We cannot *will* their reproduction immediately, for, were they objects of volition, they would already be in the presence of the mind, and therefore need no summons. Do I wish to remember the name of some person whose image is in my mind's eye? I keep it there—my thoughts about him take the forms of place and time—each of these bringing a large cluster of associations—to some one or more of which the *name* so adheres that it at last is presented to my consciousness.

There is a great difference in the facility with which ideas are presented to the consciousness and that by which they are detained. Conjunction between ideas and the consciousness, is greatly *assisted by the emotions*. An illustration will bring this before you immediately. The first time you gathered a violet you were in company with a friend. Long years afterwards you come into a room perfumed with violets which you do not see. The fragrance recalls the flower, its size, colour, form, &c. Those perceptions coexisted in the first instance. The presence of one has recalled the rest by the mere law of coexistence. The same law brings before your mind at the same time the friend who was with you; but he is associated with so many emotions that his image remains long after the violets and their perfume have faded from your sense and memory. Nay, you are so occupied with those remembrances that you may fall into a fit of abstraction, that is, be unconscious of anything but the ideas and sentiments which have thus been summoned to your consciousness. Thus you see the ideas and remembrances associated with emotions are not only before the consciousness, but are detained there; while those remembered sensations which had no other connexion than that of coexistence and succession, passed away as soon as they appeared.

These remarks on attention will enable us to return to volitional motion. The will compels a conjunction between the idea of the movement to be executed and our consciousness. To the idea of the movement, conjoined with the wish to execute it, the nervo-muscular apparatus responds sooner or later, if it be within the compass of our organization. The action is much aided by the senses, and, in some cases, particularly by the muscular-sense. Indeed there is no voluntary action of which sensation does not form a link in the series of events. This is well seen in the act of prehension. In grasping a ball for the first time, my fingers obey my wish to close upon it. The degree of force with which they will close depends on the sensation which the ball gives to the nerves of touch, and the feeling of resistance afforded by the muscular sense,—that is, the nerves of sensation in the muscles. To hold the ball in my hand, I must either keep my attention fixed on the action, or the nerves of sensation must maintain the muscular action. If these nerves be paralysed, the action must be guided by the attention and by sight. The following case, related by Sir Charles Bell, is a good example:—

"A mother, while nursing her infant, was seized with a paralysis, attended with the loss of muscular power on one side of her body, and the loss of sensibility on the other. The surprising and, indeed, the alarming circumstance here was, that she could hold her child to her bosom with the arm which retained muscular power, only so long as she looked at the infant. If surrounding objects withdrew her attention from the state of her arm, the flexor muscles gradually relaxed, and the child was in danger of falling."—*Bell on the Hand*, p. 244.

These consensual changes have something secondarily automatic or reflex in their character, for although when I fix my attention on the action, I am conscious of a sensation; yet when I am occupied with other subjects, as in talking, I have not the sensations, and yet the nerves of sensation are acting in concert with the nerves of motion, and maintaining the action. The sensational has been converted into a *reflex* action.

Next we may notice the connexion between volitional action and sensation in the process of speaking or singing. We need not go back to babyhood, though the study of it is most interesting; but we will tax our mature consciousness for the supply of information on this subject. I am learning a new language. I wish to acquire the pronunciation of a new sound—say a German guttural. It is a volitional effort. I hear the sound—I fix my consciousness upon it; my vocal muscles, after a few trials, produce it. After a time no effort of volition is needful. Certain letters associated with the first hearing of the sound suggest the idea of the sound, and this the muscular action. The action of the muscles of the voice clearly, then, belongs to the sensational group.

Strictly volitional movements are those which are objects of mental attention, combined with the wish to execute them, and which are not performed but under such circumstances. They are thus distinguished from actions which, though coinciding with, and controllable by our will, are originated and maintained by processes independent of this mental principle.

We have now to inquire how these volitional movements, after a time, pass into the category of instinctive or automatic actions. In fact we are entering the domain of Habit—a field which, though appropriated especially to this discourse, we have had to approach by a circuitous and, I fear, tedious avenue.

Habitual motions are those which have been transmuted from volitional to instinctive,—which have become secondarily automatic,—which from having been compounded of will, idea, and sensation, have become merely sensational, and perhaps, even in some cases, purely reflex. The ego—the consciousness, which was the first mover, has been able to leave the transaction to its subordinate agents, while it is occupied with other actions, or with sensations and thoughts requiring its undivided attention. Of these many have been established in early life. In standing and walking we have examples of complicated series of muscular actions guided by the sensation of equilibrium, and becoming ultimately all but reflex, though originally prompted by the will. That the will is originally concerned we see, not only by our observation of children learning to stand or walk, but also in adults in

whom the apparatus has been weakened by illness or old age, and in whom the mechanism is no longer so self-acting as not to require that mental attention to the several stages of the process, in which volitional action consists.

Speech is another of the habitual or secondarily automatic actions. In this process there is the perception of sound as connected with some object of sight (as in the naming of a thing) and the wish to imitate the sound. The action of the vocal muscles is preceded by sensation, idea, and volition. But after the habit of speaking has been acquired, it becomes purely sensational or ideageneous without intervening volition, and is allied to the instincts.

I here take the liberty of quoting from a paper which I published many years ago:—

"The articulation of every word was once, perhaps, the result of effort, a voluntary exertion of the vocal organ to imitate a sound produced by another. But now it is enough for the word to occur to the mind, and the pronunciation follows, without any intermediate volition, merely because the idea and the action have been accustomed to the relation of antecedence and consequence.

"Again: I may use some word which I not only did not intend, but which I would much rather have avoided, as it may be personally offensive to the individual with whom I am conversing. This word, in all probability, will be found to be similar in sound to that which was present in my mind, but which was not expressed by my voice. The word was the product of a certain aggregation or series of vocal movements, which followed some initial movement common to it, and to that other series which properly belonged to the idea in the mind. This we conceive to be the meaning of what is commonly called a *lapsus linguæ*, and is very different from a malapropism: *the latter is a mistake of the mind, the former is a mistake of the muscles.* A similar error not unfrequently occurs in writing. A perfect master of orthography may commit a mistake of this kind; he may write, for instance, the adverb *there*, though the pronoun was in his mind, merely from an irregularity of muscular succession. The tracing of a word on paper is the result of a particular set of muscular movements; but words of very different meanings may have very similar sets, and even initially identical, as in the instance just mentioned; and hence the mistake arises. We have heard persons say that a bad pen would make them mis-spell; in such a case, the impediment offered by the pen causes an irregularity in the succession of the movements. But it may be asked, how is it that we sometimes utter or write a word no less dissimilar in sound and in symbolical characters, than foreign to the subject discoursed of? The causation in this case is different; the error exists in the mind, and arises from our being occupied with more than one series of ideas; in which case an accidental exchange takes place between the series communicated and that which is retained. To a person engaged in writing when others are talking around him, the accident is very likely to happen. Some word makes a particular impression on his mind and diverts him a moment from his previous train

of thought; but his muscles continue to act, and follow the impulse of the word in question, as of any other that passes through his mind, and germane to the matter in hand.

"From what has been said, then, it is deducible that there are motions immediately consequent on ideas, in the same manner as others consequent on sensations and emotions; but we have not arranged the former in a separate class, because we are not aware of any evidence that *ideas* assume the relation of proximate causes to *motions*, except under the operation of the general law or principle which we have been engaged in illustrating, while sensations and emotions, on the contrary, manifestly produce their appropriate actions, without any reference whatever either to previous association or succession."—*Relations of Mind and Muscle. West of England Journal*, 1835.

Such actions as standing, walking, speaking and handling, are the most primitive arts of life, belonging to man as a species, and contemplated as essential parts of his active existence, without which, indeed, he would be wanting in the outward characteristics of humanity. They correspond to actions which in the lower animals are all but coeval with birth, or which only require the complete development of the organism rather than any process of education. The young kid walks on the day of its birth. The bird does not fly when just out of its shell, because its wings are imperfect.

In these primitive arts, then, belonging to the whole race, there is a more ready and rapid conversion of volition into habit or instinct than in others, which I now proceed to notice. These are the arts acquired by long education and practice, and which belong either to individual man originating them, or to individual man affected by his fellows. They are the manual, the domestic, the social, and the fine arts. In all these arts the limbs have to respond to and work out an idea; and the connexion must be frequently established and repeated by volitional efforts before the art becomes a habit. And yet we shall see here, again, the power of habit, and how, when it is more early acquired, it becomes allied to an instinct or an inspiration.

It is in these arts, indeed, that we may discover some of the most marked examples of habit,—recurring to that character of the principle which we gave at the onset of this discourse, as the conversion of the casual or temporary into the fixed or permanent. For as the muscular actions have been engendered to meet circumstances which are not necessary to the life and endowments of the species; in other words, to subserve wants and answer to ideas which have arisen under particular circumstances or in particular minds, so the conversion of them into actions that may occur without the maintenance of volitional supervision, illustrates very strongly the dominion of habit.

Such combinations of muscular actions as are effected in the sleight-of-hand tricks of a juggler, or in the scarcely less surprising dexterities of an accomplished player on the violin, may never before have transpired, and may never again transpire in any son of Adam, and yet in that individual they may have been so frequently produced, (*i. e.*, the will may have so often effected a junction between the idea, the sensations, and the motions) that the succession is maintained without any mental

attention. The player may talk to you while his hand is bringing out the music; one note suggests the next which has so often proximally succeeded to it, and with it comes the nervo-muscular action according to the principle of association which we have before adverted to. But in cases of this kind we may sometimes in our analysis fail to discover any link formed of an idea or a remembered sensation. Thus, one may begin to whistle without the faintest notion of what tune the muscles of the mouth and cheek will modulate. The muscular actions aggregate themselves into combinations and series unprompted by an idea, unguided by the will, nay, unsuggested by a sensation (for the motion precedes the note evolved), and arranged solely by the law of previous coexistence and proximal succession, repeated an adequate number of times. They are mechanical, automatic, reflex. Originally, perhaps, the order of events had been remembered;—musical sounds associated with certain words. The recurrence of the words brought the sounds, to which the vocal movement responded. But now the first movement sends an impression to the reflex centres, and these maintain the series independently of the sensational, ideagenous, and volitional centres. It is like the running off of a musical machine, except that the series may be deranged at any moment by an idea or emotion or sensation. Therefore it is most likely to be safe from interruption when the mind is in that blank condition to which the most sentient, sentimental, and intellectual are sometimes subject. Therefore there is a physiological meaning in the old line:—

"He whistled as he went for want of thought."

The habitual or *secondarily* automatic actions bear resemblance to the *primarily* automatic in the circumstance that when once established they are liable to derangement rather than assisted by mental attention. Fix your mind on the process of breathing, and it becomes laborious or irregular,—think of the act of swallowing while you are performing it, as in taking a pill, and the action becomes spasmodic, convulsive, abortive. In like manner if you attend to your walking as you pass from one side of a drawing-room to another, it is ten to one but that your gait and carriage will be a series of jerking, swaying, rolling motions, in short, awkward, constrained, ungraceful. Something in such cases must be set down to emotional causes, such as the disturbing influence of anxiety. But from what we remarked before, you may be prepared to admit that the act of volitional attention is closely allied to emotional processes. And it will be found that in certain of the secondarily automatic *mental* operations, this disturbing influence is still more easily traceable.

The readiness with which these habitual movements are established I have already mentioned incidentally, is closely allied to natural aptitude or talent. It is far beyond dispute, that however great may be the power of education and practice, yet that men differ immensely from each other in original capability for particular arts. Men are *born* artisans and artists, as well as poets; for the former, whatever may be the mental requirements, there must, at all events, be an aptness of hand, a quickness of response in the muscles to the ideas in the mind—a

readiness for the establishment of those series, and aggregations of complex co-ordinate movements, which, for their first institution require the repetition of volitional efforts, but which in one man require almost an indefinite number of repetitions, while in another a very few will suffice to convert them into habits. This, however, (natural dexterity) is but the bare mechanical substratum of skill in art. Yet without it a man cannot excel, though his mental capability for the higher processes in the art be of transcendent excellence. He may have the most nimble apprehension, a memory the most retentive and accessible, the most refined and delicate sense of the beautiful, a power of rapidly combining and abstracting those qualities in natural objects, which, when reproduced, will faithfully and vividly represent the originals,—an instinctive knowledge of that subtle symbolism whereby objects are made to evoke in other minds those sentiments which were inspired in the artist as *he* looked on nature, and with all this, a noble desire to make the æsthetical gratification minister to its natural handmaids, Virtue and Religion; he may have all these, and many more qualities, necessary for the accomplished artist, and yet they may only serve him for the appreciation of the achievements of others,—not because he wants "the vision and the faculty divine," but because the hand is not a deftly servant of his thought. But when, indeed, the hand and the eye and the mind do come together, each with the highest qualities, compass, quickness, and co-ordination, then we have a Phidias, a Michael Angelo, a Raffaelle. Many like instances might be adduced; but I hasten to remark, in connexion with this department of our subject, what indeed is well known to every one, that for the formation of many of those habitual co-ordinate actions, nothing is needed but frequency of repetition. They argue no special fitness of organization (though, indeed, even in them a natural cleverness may be discernible), and merely instance the power of repetition. Such are the mechanical processes of every-day life,—the act of writing, the operations of the toilet, the dinner table, &c.

But here the question naturally occurs, why should mere repetition dispense with volitional exertion? To answer this fully, would require a long and abstruse discussion. I must content myself with a summary statement of what seems to me to be the explanation of the fact. The more the action of an organ is augmented the stronger it becomes, and its nutrition increases. This is clearly seen in the development of muscles called frequently into play. I would apply this fact by analogy to the nervous centres. The different nervous centres in the encephalon communicate with each other by commissural fibres. In volitional movements, the action of the commissures between the sensational or ideagenous centres and their related motor centres is excited in the first instance by the will. But the repetition of more or fewer of these incitements appears to suffice for the growth and development of the commissure to a degree of strength and activity adequate to the performance of its functions independently of the will. Thus the volitional action is converted into a habit.

The anormal habits are curious, and perhaps less easy of analysis. They are those actions which subserve no apparent purpose, either as

to the functions of the whole system, or to the will, or the emotions, or the ideas of the individual. What earthly end is answered by biting the nails, twitching the hair, twirling the thumbs, rubbing the hands, kicking the heels, and various other singular and all but unaccountable human actions, which are designated in the vernacular as tricks or fidgets? Why should a person under strong emotion, exhibit such peculiarities of deportment as are attributed to Sir Jacob Kilmansegg on the morning of his daughter's christening?

> "And Sir Jacob, the father, strutted and bowed,
> And smiled to himself and laughed aloud,
> To think of his heiress and daughter—
> And then in his pockets he made a grope,
> And then in the fulness of joy and hope,
> Seem'd washing his hands with invisible soap
> In imperceptible water."

Why was it that when a certain great statesman in the House of Commons was about to make a signal rhetorical effort, his friends were advertized of it by his mechanically rattling his watch-chain and seals for full half-an-hour before he rose? Why do little boys, in saying their lessons, go through sundry mysterious operations of buttoning and unbuttoning their jackets; and why do gentlemen, earnest in argumentation, wildly lay hands on the little implements of a neighbouring lady's work-table, and turn them to strange uses on the adjoining furniture? There must be a law for facts so common, and all but universal.

I believe that they are referable to two principles—one of them is the provision made in the nervous system for the concurrence of muscular action with deep thought and strong emotion. Consider how nearly all the emotions have their natural alliance (in the form of expression) with certain muscular actions in the features and the limbs, and how very important these are in the mutual communication of men in early states of society. Consider also, how often some form of muscular action goes with thought; and again, how common it is to accompany speech with gesticulation; and how in the more important acts of life, thought and motion are going on together. In literary composition, the action of writing is a natural accompaniment. If a man dictates, it is ten to one but he will walk about the room while doing so, because it is natural for some muscular action to accompany profound thought.

But you may say that these considerations do not help us out of the difficulty. For speech and gesticulation being the accompaniments of thought and emotion, why should we add such unmeaning muscular actions as those which have been adverted to? We come then to the other principle, which is, that the motor centres are apt to elaborate more force than is required, and which must be disposed of in some way. Now that there is such a thing as nerve-force cannot be doubted. We talk of force of attraction, of chemical force, of motive force, of electric force; and though none of these forces can be produced as entities, and may be only summary expressions of certain chains of phenomena considered in their causative order, there is as much reason for using the term nerve-force as any of the others. It

is that principle or power which, generated in nervous tissue by virtue of the changes which this substance undergoes in its ultimate muscular nutrition, (in the higher animals, under the operation of blood-changes,) stands in the relation of proximate cause to motion, sensation, and the material ministrations of thought. The common sense or consciousness of mankind, attains to a very near recognition of this power, when it assigns the buoyant movements of childhood to an excess of vitality, and when a man says that he feels such a surplus of irritability or irritation in his system, that he takes active exercise to get rid of it. We have seen how the nervous centres are brought into action simultaneously and co-ordinately. Now if there be great excitement in one, (excitement means exalted vital action,) there will, to a certain extent, be the same in others, more especially in those related. If the emotional centres are strongly excited, there will be a large elaboration of force in the motor centres, some of which force may be discharged in the appropriate featural expressions and gestures; but to expend the whole of it, strange movements and antics will very probably be performed. To let these have free play is a great relief to many persons, while to restrain them requires great self-command and discipline.

In sleep, although the centres of sensation are quiescent, there is often too much activity in the motional centres, and then the nerve-force goes off in a variety of irregular muscular actions, such as kicking, stretching, change of position, tossing off the bed-clothes, &c.

In sensation, in motion, and in thought, nerve-force is generated and consumed. The fatigue, nay the absolute bodily exhaustion, induced by excess in any of these functions is a proof of it. In persons of the nervous temperament, there is at all times an amount of nervous energy produced beyond what is absolutely required for the performance of the normal functions of the brain. Hence it is in these persons that we see the most striking instances of the tricks which we are now considering. Very remarkable is the tendency in some children. They wink their eyes, make strange contortions in their limbs, change their posture, shift the weight of the body from one foot to the other incessantly when answering the slightest question, and all from the excess of motor nerve-force generated concurrently with the little mental excitement. These movements sometimes occur in particular sets of muscles in preference to others, and then they are called habits.

But I cannot leave this part of the subject without remarking that there is good reason for the caution which has often been given, not to interfere too much with these nervous habits; for they are sometimes salutary outlets for morbid irritation. I know a young lady who, when her mind was unusually interested, had a habit of rubbing her forehead, and if this were prevented, either by moral or mechanical means, it seldom failed to induce threatenings of serious disorder in the brain. There are certain kinds of convulsive seizures which may be considered as discharges of nerve-force, like those of electric jars, highly charged. Pardon me for giving you one more practical instance: There are some persons whose brains, when highly excited, say, by a public entertainment, or an interesting conversation or argument,

or even by having fallen into some engrossing train of thought, cannot pass readily into the normal condition. The elaboration of nerve-force continues. If they retire to rest they cannot sleep; they go on thinking, and feeling, and tossing about, or vividly dreaming, when they ought to be "mere clods of the valley." Now the best thing to prevent this, is to take active muscular exercise before retiring, whereby the superabundant force is used up.

Habit, as to Sensation.

On first considering the effects of habit in connexion with sensation, it would seem to be the reverse of what occurs in connexion with motion. Habit, instead of increasing, seems to lessen sensation. We speak of becoming too habituated to things to feel them. The contact of clothes with our skin is habitually disregarded, though, had the investiture been a new ceremony, we should not be able to forget them for an instant during the day. We sit in a room reading or talking, while a time-piece, to the sound of which we are accustomed, strikes the hours and the quarters, without hearing it. The inhabitants of miserable tenements in the worst parts of our towns, (where sanitary matters are neglected to a degree that is a disgrace to civilization, and a dishonour to Christianity,) live unconscious of odours, which strike those who have not been inured to this wretchedness with disgust and terror. The "common light of day" gives us no subjective sensation, no feeling in our eyes, unless we have been unusually excluded from it for a long time, and then it may be blinding.

After considering such facts as these, we remember on the other hand, that education is known to quicken the senses, that the North American Indian is said by habit to see and hear sights and sounds which escape his more civilized brethren; and that the purchaser of tea can try forty or fifty sample-cups in succession, and make his selection of the best flavours with unerring accuracy; and that wine-tasters can, by practice, attain to the delicacy of discrimination which characterized those notable rivals vouched for by Sancho Panza, of whom, as you remember, one declared that the wine tasted of iron, and the other of leather, and each of whom was found to be correct when the cask had been drained to the bottom: for there lay an old cellar-key, with a thong attached to it.

These two sets of facts seem at first sight to contradict each other, but they do not really do so. We have seen that habit in reference to motion strengthens and gives independent power to a connexion established in the first instance by volition. It will be found to be the same in sensation.

What is the process in sensation? An impression is made on a sentient nerve, and conveyed by it to the sensory ganglia, where it becomes connected with the Ego, and is then, and not till then, a sensation.

We have already been speaking of attention, and shown that impressions are presented to the consciousness by virtue of their novelty or vividness, and are detained or dismissed according as they are associated or not with emotions, or compulsorily associated by the will. It

appears to be a law that a repeated impression ceases to become a sensation when its novelty ceases. A different degree of vividness is in fact a new impression, and therefore comes under the law. The final cause of this arrangement is obvious. Were we at the mercy of all the impressions which are perpetually made upon us, were they all converted into sensations, connected thought would be impossible. But the impression may have nothing of novelty, and yet its recurrence will always be followed by sensation, because it has been associated with an emotion. This effect of association is strikingly exemplified in sleep. Impressions of the most violent kind may be made, and yet the sleeper will not feel them if he has been accustomed to them, and not had them connected in his mind with some subject of interest. It is told by Dr. Carpenter, of a great naval officer, that he could sleep through the loudest noises on ship-board, but if any one whispered in his ear the word *signal*, he was awake in an instant. So I know the case of a lady who sleeps profoundly during ordinary noises, but if her relative who is near her, makes in breathing a sound which she has often known to be the precursor of an attack of illness, she wakes immediately. So also with the tick of an alarum.

In the sharpening of the senses by education and training, we again see the influence of the emotions, or of ideas associated with emotions. We look and listen, that is, we attend to sights and sounds, because there is an expectation of good to be received, or a fear of evil to be averted. Again we are said to acquire the habit of attending to certain classes of sensations. Here the emotional interest is more remote. The objects of sensation may be uninteresting, but we exert our volition to compel a coherence between the impressions and the consciousness; and the conviction having been often repeated, and so once established, becomes permanent; the volitional becomes automatic. Habit supplants the will; and thus we have another illustration that the influence of this principle is exerted by virtue of association.

Habit would thus seem the offspring and afterwards the successor and representative of volition. But what is meant by a habit of inattention, a listless habit? Thus a person may fall into the habit of sitting at church and not listening to the service or the sermon. The use of the word in this manner is a metonymy. It is not the habit of not attending, but the habit of attending to your own thoughts in preference to those of the clergyman.

Everywhere in this subject we fall back on the principle of association. Everything in this world is interesting, or important, or influential, according as it is related with other things.

The repetition of volitional impulses gives increased strength to the related centres of motion, because these centres become active. The repetition of impressions which are not taken up by the emotions or the will, and do not pass into sensations, will come to nothing. It is not that habit deadens the sensibility to the repeated impressions, but that the impressions ceasing to be linked with the emotions and the will, having in fact no associations with any thing else in the mental life, die away; or, indeed, never arrive at any thing but an abortive existence.

Habit as to Thought.

In the remarks preliminary to our consideration of volitional motion, I spoke of some of those mental processes which are enforced by the will in efforts of memory. There are others to which I can but very briefly advert, with a view to the prosecution of our immediate subject. Such is the compulsory contemplation of phenomena in a certain order or arrangement, when we reason, whether inductively or deductively, or when we arrange, according to a certain shape or plan those clusters of ideas and emotions which belong to fancy and imagination.

Congeries of ideas, or trains of thought, originally associated or marshalled by volition, may, like the volitional movements, become automatic. The memory of compositions, or of narratives, or of arguments, once attained by efforts of the will, becomes automatic. Recollection passes into remembrance. Nay, like the analogous movements, it is apt to be deranged by mental attention. How often does a person find that the more he tries to remember something, the more he is baffled. He gives it up, talks of something else, and that which had been sought for in vain, comes unbidden. The automatic process of association went on better when the consciousness was not fixed upon it.

Arithmetical calculations illustrate this principle. That 2 and 3 make 5, is at first committed to the memory by means of the will,—after a few repetitions of these three figures, the combination of the two first will bring the third mechanically, that is, 3 will add itself to 2, and bring 5 in its train, and so on with a long column of figures. Mr. Dugald Stewart adduces the case of an expert accountant running his eyes up a column of figures and arriving at the sum total, unconscious of the intermediate mental acts, as an instance of a succession of efforts of attention, so rapid as to have been forgotten. But there seems to me no need of presuming that of which there is no proof. The numbers seen have so often been linked with their equivalents in the memory, that the latter recur passively and mechanically. Thus in consequence of long use and practice 2 and 3 cannot but suggest the idea 5, and 5 meeting 7 brings 12, and 12 meeting 6 brings 18, and with 18 added to 9, 27 links itself, and so on.

Thus the visual and the mental trains of figures interchange without any effort.

These associations have so often been compelled, that they now recur by necessity. It is just as with the player on an instrument; the impulses in the centre of motion so often associated with the notes of music on the paper come without attention, though at one time their co-ordination was only effected by dint of the most laborious attention.

8	39
4	31
9	27
6	18
7	12
3	5
2	
—	
39	
=	

In reasoning, a like automatic process is traceable. The *ergo* fastens itself without effort to the premisses. The conclusion is self-evolved from the major premiss.

And the self-evolution of thought may be traced much further.—Ideas grow out of ideas, without our consciousness of the various stages of their mysterious genesis. The new production suddenly blossoms forth to the astonishment of its possessor, who finds himself all but unawares the maker of a grand discovery. Or to vary the illustration, take the case of a writer of works of imagination. His mind has been stored with a vast assemblage of images, scenes, sentiments, and characters. In some auspicious moment, and sometimes unexpectedly, the scheme of a poem or fable unfolds itself before his mental eye; it is as if his mind up to that time had held all the scenes, incidents, characters and similies in a state of solution, and then by a happy conjunction of outward and inward agencies, the several elements of thought attach themselves to each other by their several elective affinities, and crystallize into their appropriate shapes and colours of beauty and grandeur.*

The secondarily automatic processes of thought, induced by habit, closely resemble those which are instinctive. To the latter class belong the intuitive beliefs. To believe the information of our senses, as that the sun moves, is an instinct. Such also is the expectation of like consequents from like antecedents, or cause and effect. Such, also, is the confident belief in the existence of outward things—the separation of the *ego* and *non-ego*. Such, also, the reference of the existence of things and of ourselves to an unseen Power. These, like other instincts, are variously susceptible of modification from subsequent experience. But in all these cases, the ideas have not been brought together by volitional efforts; they have an innate tendency to cohere. Indeed, they can hardly occur separately. The existence of the one necessarily involves that of the other. They, in fact, grow together. But the ideas which have only been casually clustered, or knit together by the workmanship of volition, may ultimately cohere as strongly. The force of habit will thus equal that of instinct; a faulty train of reasoning may acquire the strength of an original instinctive perception; an oft-told lie becomes a truth, and a superstition may be as hard to break asunder as the simplest and most primitive religion. Education thus vies with intuition—and habit becomes second nature.

These habits of thought, like habits of action, when once acquired are not easily discontinued. They are like grooves in which the mind has been accustomed to slide; if well contrived and fitted, and in a right direction, they are of incalculable value. If not, they are injurious, they prevent the mind from moving in better ordered and more truthward tracks. In the progress of life it becomes more and more difficult to alter our habits. The grooves have worn deeper and deeper, and the volition is less and less willing to trace or carve new ones. It is ridiculous, I should better say, it is mortifying to human nature to watch the awkward and futile efforts of a mind to throw off

* The whole subject of the automatic action of the brain in intellectual processes is treated with great ability and originality in Dr. Carpenter's Chapter on the Cerebrum and its Functions—("Principles of Human Physiology," fourth Edition). The reflex action of the brain with reference to motion had previously been expounded in a highly ingenious paper by Dr. Laycock, read before the British Association, September, 1844.

its long formed habits. Take it out of these prepossessions, and prejudices, and bigotries, and how uneasy and jolting are its movements! for such habits are ruts rather than grooves, which though they allow no rapid motion of the heavy carriage that follows them, yet cannot be escaped. In attempting to quarter the road the movements are made without confidence, and the wheels are perpetually threatening to slip into the old tracks with a crash that endangers the safety of the whole vehicle.

It would be interesting, but time will not allow us, to pass on to an investigation of habit in reference to the emotions and moral sentiments. But the same law will be found to hold good in that department as in those which we have so imperfectly treated.

I must conclude with a few words on the *final cause* of habit.—

As to habits of action, it is obvious that the great use they serve is the economy of time. What would man have accomplished by the end of his life had it been needful for him to attend to his movements in standing, walking, and using his hands and fingers? What progress would thought make, were speakers to be thinking of the sounds they utter, and to be consciously directing and adjusting their vocal apparatus? and where would be the literature of the world, were the mind compelled to pass from its sublime contemplations to the muscular actions which guide the movements of the pen? But the more we consider the subject, whether as to the development of those actions which characterize the species, or as to those acquired accomplishments and dexterities which range from the humblest handicrafts to the loftiest triumphs of the imaginative arts, the more we shall be struck by the gradually increasing subordination and subjugation of the mechanical processes to the more exalted faculties of the mind.

This view would at first, perhaps, make us inquire whether, as these volitional movements which we have been considering ultimately become automatic, it would not have enlarged the capacities of man, had they *begun* as instincts, just as some of them really are found in the lower animals, instead of going through so long a process of evolution and education? A foolish question, as every question must be, which proposes an arrangement of events different from what is obviously a part of the plan of God's universe. Take away the struggling striving will even from these corporeal actions; remove effort, resolution, the conscious initiation of action, perseverance, training, and education, and what is human life reduced to? Gigantic as man's powers become, he was not intended to spring from the earth in their full equipment. Survey him in his infancy, childhood, youth, adolescence, and manhood, and while you become convinced that his gradual acquirements bring him a multitude of enjoyments, as well as difficulties and disasters, you cannot but see that what is evolving in him bears a strict correlation to the powers, emotions, sentiments, and virtuous actions of those, who having arrived at the maturity of *their* powers, are to help *him*, to whom *he* is bound, as they to *him*, by ties which make the affinities of the human family infinitely transcend the transitory parental instincts and gregarious associations of the lower animals; for *they* live and grow up

almost as they were born, devoid of progress, not one whit wiser or more skilful than the first pair that issued from Noah's ark, living for themselves only, or only under a blind impulse providing for another succession. But man having consciously and with pain and labour and peril, acquired his endowments, lives them over again by teaching them to his offspring; and apart from that happier existence to which he knows that he is destined in other worlds, feels that here too he has a kind of immortality; that as he has inherited knowledge, and virtue, and power, he too has to transmit them; that his life and its achievements have a mortal metempsychosis, a translation into the enlarging attributes and brightening destinies of his children, and of unborn generations, and in the production of works which, like Milton, he knows that posterity will not willingly let die, and in the elaboration of systems which, like Bacon, he bequeaths with his fame to the next ages; in this realizing anticipation of a posthumous renown, he survives his own death, passing by his living consciousness far beyond the narrow bounds affixed to his mere corporeal duration.

But while habit, as we have seen, is so useful in abridging labour, in economizing time, in preserving order, and method, and coherence in our thoughts, and in making the practice of virtue and religion easier to us, still it imposes upon us no inevitable compulsion. It is not the blind necessity of an instinct. It is our own fault if we are enslaved instead of being merely assisted by habit. Human agency ought to be able to assert its freedom in this as in every other department of thought and action. The habit should be like a steed, so well broken, that though the will may have thrown the reins on its neck, while otherwise occupied, it can in a moment gather them up, and come to a sudden halt.

Habit, we have seen, is at once the product and the sign of previous volition. And though in certain muscular actions belonging to the species, it closely resembles instinct, yet as to the thoughts and actions of individual men it is widely different. For as the will of every man has its own peculiar form and colour, making an important part of his individuality, so his habits will have their own character and freedom of growth. Those who are attached to him will regard with partiality the very habits which have grown out of his peculiarities. The singularities of his gestures, the eccentricities of his gait, carriage, and demeanour, the oddity of his featural expression, the tone of his voice, his ways and his whims, his fancies and his philosophies, his predilections and prejudices, the whole complexion of his life, and the whole colour of his conduct—his goings out and his comings in, his risings up and his lyings down,—all are valued, because they give us more vividly the express image of him who is endeared to us for his own individual sake.

But while there is the utmost latitude for the formation and the relinquishment of habit according to the will and humour of the individual, the habit which has grown out of a strong character will inevitably impress itself on others. A strong character does not depend on the intellect, but on the original strength of the will, and on the habits which it has originated. It is one whose actions have sprung out of its own

individual will. The very limitation of the intellectual vision may strengthen the volition by the repetition of its objects and by the iteration of its correlative actions. This self-possessed, self-contained, self-originating power, is sure to affect others. It is a new centre of motion to those who are ever ready to derive their impulses from without, rather than from their own life.

Thus we see the two elements in social life antagonizing each other, and yet bringing out the most important results;—the potential freedom of every individual—and the unfelt compulsion of a passive imitation. And were any argument needful beyond what has been so often urged by moralists and divines as to the formation of habits which may become so powerful either for good or evil, in the *individual* character, it is to be found in the consideration that in our influence on others, we are responsible not only for what we directly do or directly teach, but also for that insensible operation of our characters which, in proportion as they are strengthened by habit, are in that same proportion sustained in their capability of impressing and moulding to their likeness, the wills, the affections, the thoughts, and the actions of our fellow men.

APPENDIX.

WHILE these sheets have been passing through the press, the popular mind has been wonderfully excited and occupied by the Table-movement. As this rage is likely to be even more ephemeral than these pages, it may be worth while to record very briefly the phenomenon, and to offer what appears to be the rational explanation of it. Three or four, or more persons, stand or sit round a table with their fingers slightly resting upon it, the little fingers of each individual resting on those of his neighbours. After patiently waiting in expectation for fifteen or twenty minutes, rarely sooner, one of the company observes that the table begins to move, and forthwith it either moves in a rotatory or in a straightforward direction, according to the shape of the table and the disposition of its legs; and the operators move with it. In one case which I have heard of—the table having been rickety, and its top more mobile than its supports, the movement consisted in a swaying backwards and forwards of the former part, while the latter remained steadfast.

It will be inconsistent with the gravity of the future historian of our era to depict the scenes of which this movement was the centre; else one could wish that posterity might not be defrauded of some amusement, together with the satisfaction of a conscious superiority, while contemplating this mania of their ancestors. Yet they would scarcely believe that in many of the club-houses of London, comprising among their members some of the most distinguished personages of the country, might have been seen groups of earnest and excited elderly gentlemen solemnly trotting round tables in the performance of this mysterious rotation, and showing by their countenances that they were convinced that themselves and the table were the subjects of a new development of force which was about to revolutionize the physical philosophy of the age, and to indicate the long-sought-for bridge which opens the dark gulf between living and inanimate matter. Alas, for their philosophical aspirations!—

> Usque adeo res humanas vis abdita quædam
> Obterit.

To a person dispassionately observing the phenomenon for the first time it surely would naturally occur, that as the things in immediate contact were, on the one hand, inert (according to common philosophy,) matter, and, on the other, a number of bodies made up of nerves, muscles, bones, &c.,—in short, an apparatus especially designed for imparting motion to surrounding objects, that the movement of the table was somehow dependent on the action of the said instruments of motion. He is met, however, by an assertion on the part of the operators, that they communicated no motion to the table, that they

only kept their hands in contact with it, and that it moved by virtue of the electricity passing out of their bodies. A magnificent leap to a conclusion! But the observer is not so agile in his inferences. He examines the facts attentively, and ventures to think that it is superfluous to invent any new causes for the explanation of a fact which, though curious, falls within some of those categories of motion which have been the topics of the foregoing lecture. The movements of the hands or fingers laid on the table that give an impulse to it are not volitional, but secondarily automatic; partly ideagenous—that is, following the idea or expectation in the mind; partly consensual, through the muscular sense, or simply reflex. A certain time is required in order that there may be the necessary unity of expectation and unity in the direction of the unconscious operation. In many cases, the table, though very light and mobile, will not stir at all. In these instances the operators are not all impressed by the same idea, or the hands of some are more under the influence of the will than of the automatic agency. Marvellous stories are told of the table's moving on one or two legs only; or, we should rather say, that the fact is told as if it were marvellous, when the simple explanation is, that the motive force is stronger on one side of the table than of the other.

It is perhaps worth while to remark, that in addition to the automatic sources of motion referred to, there are two others; one, that of the insensible resting of the hands on the table when the operators are fatigued; the other, and a not unimportant source, the predominance of the flexors over the extensors. The fingers are kept in contact only with the table, that is, are prevented from pressing upon the table, by the volitional action of the extensors. A very short time suffices for the withdrawal of this volitional exertion, and then the flexors act by their tonic contraction. The multiplication of this through forty or fifty fingers will be enough to cause in the table sundry creakings and irregular movements, very exciting to the imaginations of the experimenters, whose minds unconsciously surrender themselves to the idea of a movement in the table, and then a series of actions in the right direction follows almost immediately as a matter of course.

[Since this note was written, the Author has read a very clear and sensible report on the subject in the *Medical Times and Gazette* for Saturday, June 11, 1853. Those who require further satisfaction as to the cause of the popular phenomenon, may study, with advantage, Dr. Carpenter's section on the "Influence of Expectant Attention on Muscular Movements," and Sir Henry Holland's remarks on the "Effects of Attention on Bodily Organs," in his "Chapters on Mental Physiology."]

CLIFTON, *June* 14, 1853.

[*From the* PROCEEDINGS *of the* ROYAL SOCIETY, May 28, 1857.]

CROONIAN LECTURE.

ON THE CAUSE OF THE RHYTHMIC MOTION OF THE HEART.

By JAMES PAGET, Esq., F.R.S.

I have selected for the subject of my lecture, the cause of the rhythmic motion of the heart; guided to this choice, partly by the belief that the Croonian lecture must have some relation to muscular motion, and partly by the interest which I have acquired in the subject in the course of observations extended, though with many and long interruptions, over some ten or twelve years.

It is not necessary that I should enter on any consideration of the various opinions that have been entertained on the cause of the heart's peculiar motion. Let me first show what it is, and how it differs from the other motions in the same body, which are visible to the naked eye.

In a beheaded tortoise, or any other of the Amphibia, the muscles of the trunk and limbs are usually in perfect rest, unless disturbed: those in the head may act so as to produce a kind of gasping and swallowing movements, at distant and nearly regular intervals (ar. imperfect kind of rhythmic motion): the digestive and other mucous canals are at rest, or move with slow worm-like actions; but the heart maintains, with perfect regularity, the rhythmically alternate contractions and dilatations of its auricles and ventricle; its several movements being ordered, not only in the manner of their succession, but in rhythm, *i. e.* in the proportions of time which they severally occupy.

Why is this difference? To what may we refer as the cause of this apparently peculiar mode of muscular movement? In answer, let me show (for it is a condition of the Croonian foundation that experiments should be shown) that the cause of the rhythmic action is in the heart itself; not in any of the great nervous centres through

which other muscular movements are excited, including those in the separated head, which will cease at once on destruction of the medulla oblongata and brain.

The evidence of this is in the continued action of the heart after it is cut out of the body. It may remain at rest for a few minutes after the excision; but then, as if recovering from shock or fatigue, it again begins to act, and thus will continue for many hours acting as regularly as it did when its connexions were unbroken.

The time during which the action of the cut-out heart will be maintained is different in the several classes of the Vertebrata; but some such continuance may be observed in all, and in all the experiment is enough to prove that the rhythmic action does not depend either upon distant nervous organs, or upon the blood which naturally flows through the cavities of the heart; for as soon as the heart is cut out, its cavities are emptied and no blood flows through them.

Thus, then, for a first conclusion, we may be sure that the cause of the rhythmic action of the heart is something in the heart itself; and this, notwithstanding the variations of the rhythm, which may be produced by morbid or artificial states of organs far distant from the heart.

But the cause, whatever it be, is not equally in all parts of the heart; for when its parts are in certain manners separated, some continue to act rhythmically, and others cease to do so. If, for example, the cut-out heart be divided into two pieces, one comprising the auricles and the base of the ventricle, the other comprising the rest of the ventricle, the former will continue to act rhythmically, the latter will cease to do so, and no rhythmic action can be, by any means, excited in it. The piece of ventricle does not lose its power of motion, for if it be in any way stimulated, it contracts even vigorously; but it never contracts without such an external stimulus, and, when stimulated, it never contracts more than once for each stimulus. Without losing motility, it has lost all rhythmic power, and all appearance of spontaneous action. We can, indeed, make it imitate a rhythm by stimulating it at regular intervals, such as every two or three seconds; but in any experiment of this kind, it will presently appear how much sooner the motor force is exhausted by the artificially excited actions, than by the apparently equal actions of the natural rhythm.

Other sections of the heart, and experiments of other kinds, would show that the cause of the rhythmic action of the ventricle, and probably also of the auricles so long as they are associated with it, and not with the venous trunks, is something in and near the boundary-ring between the auricles and ventricle; for what remains connected with this ring, or even with a part of it in a longitudinally bisected heart, retains its rhythm, and what is disconnected from it loses rhythm.

An experiment related by Heidenhain*, seems to show more precisely where the source of the rhythmic action of the ventricle is seated. If the ventricle of a frog's heart be separated, and every part of the septum of the auricles be removed from its base, so that its cavity may be perfectly single; and if, then, it be set upright on a board with some blood in its cavity and around it, it will be found that as pieces are cut away from its upper border, so its pulsations become less and less frequent, till at length, when a zone of a certain depth has been removed, they cease altogether. The depth of the zone to be cut away may be nearly one-third of the length of the ventricle; and somewhere in this zone we must assume lies that on which the rhythmic action of the ventricle, when alone or with part of the auricles, depends.

But wherever may be precisely the sources or centres of the rhythmic action of the heart, or any of its parts (and most of these details, I think, have yet to be determined, even for the hearts of Amphibia, and much more for those of other orders), these experiments seem enough to prove that the rhythm does not depend on the properties of the muscular tissue alone or independently. If it were so, it would be highly improbable that certain portions of the heart being separated should lose rhythmic action, and others just like them, so far as their muscularity is concerned, should retain it.

This conclusion is confirmed by many experiments invented by Professor Stannius†, and often repeated by myself and others, with results varying little from those which he obtained.

Perhaps the most remarkable of them is, that if a ligature be tied tightly round the place of conflux of the great veins entering the

* Quoted in Canstatt's 'Jahresbericht' for 1855, p. 130, from his "Disquis. de nervis organisque centralibus cordis." Berlin, 8vo.

† Müller's 'Archiv,' 1852, p. 95.

auricles of a frog's or tortoise's heart, the rhythmic action immediately, or after a few beats, ceases, and is suspended for three or many more minutes, and then returns in the ventricle alone (I believe) at a much slower rate than it had before.

This result is not due to the stoppage of the flow of blood into the heart, for if the veins be tied separately at a distance from the auricles, the rhythm continues. Neither is it due to the loss or great diminution of mere motility; for if the heart thus brought to rest be stimulated, it acts once, or rarely more than once, its action travelling from the part stimulated over all its substance. The result seems due to an injury done to something beneath or behind the ligature, from or through which some influence would naturally be conducted from the conflux of the veins to the auricles, and thence to the ventricle; for in the other direction, *i. e.* in the trunks of the veins behind the ligature, even to a great distance from the auricle, rhythmic motion still continues, and is scarcely, if at all, changed. And by variously placing the ligature, it would be found that, wherever tied between the venous sinus and the auriculo-ventricular ring, all the parts behind it would retain, and all those before it would lose (at least, for a time), their rhythm.

This indication of the experiment (that the loss of rhythm, namely, depends on injury of something beneath or behind the ligature) is confirmed by another, whose whole import, however, I am quite unable to explain.

If, when a heart is thus brought to rest by a ligature tied round the conflux of the veins, another ligature be tied round the boundary ring of the auricles and ventricle, and including the bulbus arteriosus of the frog, or the two aortæ of the tortoise, the rhythmic action remains suspended in the auricles, but at once begins again in the ventricle, or, if it have not wholly ceased there, is much accelerated. We thus obtain a condition of the heart in which the great veins and the ventricle act rhythmically, though not with the same rhythm, but the auricles between them are at rest.

Now, in all these facts there appears sufficient evidence that the source of the rhythmic action of the heart is not in the muscular structure alone, or in its relation with the blood in the cavities, or in the vessels of the heart; for these things are not in any of the experiments more disturbed in one part of the heart than in another.

The nutritive relations of the muscular tissue and the blood are sufficient for the maintenance of mere motility, but they are not sufficient for the maintenance of rhythmic motion; they can maintain the power of acting upon external stimulus, but cannot give spontaneity of action, or regulate the time or manner of acting.

It is interesting to observe in these experiments three different modes of action of the heart: (1) the truly rhythmic, in which the contractions of its several parts, or of some of them when separated, ensue spontaneously (*i. e.* without evident stimulus or change of external conditions), and observe a definite order and proportion of time; (2) such as may be excited by stimulus, which, beginning at the cavity stimulated (whichever it may be), are effected simultaneously, or with a scarcely appreciable difference of time, by all parts of the walls of that cavity; then follow, with a certain interval, in the other cavities; and then are not repeated in the same order, but give place to the true rhythmic action, which they may for once have prevented or inverted; (3) those which ensue on stimulus, when the heart or any of its parts is utterly exhausted, and which commence at the point stimulated, and thence slowly travel once to all other points connected with it by continuity of muscle.

Now, of these three modes of action, the last may be ascribed to mere motility, such as the intestines and the stomach have, and such as is maintained by the nutritive relations of the muscular substance and the blood in its vessels. The second may be due to the same, acting with more energy; or it may be ascribed, as by Bidder* and Rosenberger†, to the action of reflecting nervous centres in the ganglia of the heart. But what of the third, the rhythmic, which every experiment shows to be essentially different from both of these?

It is explicable, and all the experiments are consistent, on the belief (which many before me have entertained) that it depends on certain nervous centres in the nerve-ganglia of the heart; which centres, by spontaneous discharges of nerve-force, cause the muscular structures to contract. These centres (rhythmic centres as they have been called) being injured or hindered in their operation, the rhythm ceases, though the motor power is not lost; or these centres, being cut away with certain portions of the heart, the other portions cease to

* Müller's 'Archiv,' 1852, p. 163.

† De centris motuum cordis. Dorpat, 8vo, 1850.

have rhythmic motion, though still capable of repeated single actions when artificially stimulated.

Such nervous centres may exist among the numerous ganglia in the heart, and the indications of their position in the frog's heart given by the experiments, agree very well with the dissections by Bidder and Rosenberger. There is, indeed, nothing in the structure or aspect of the ganglia presumed to be rhythmic, which can make us sure that they can act as nervous centres of peculiar force, much less (if it were possible) that they must act rhythmically. But the assumption of such rhythmic nervous centres is justified, not only by its sufficiency as an hypothesis, and by its accordance with the existence of ganglia in the appropriate places in hearts, but by the analogy of the only other example of naturally rhythmic muscular movements in our own or any other mammalian economy,—I mean the respiratory movements. These are certainly determined in their rhythm by the medulla oblongata. It is true, they are greatly influenced as to their rate and force, though not as to their very existence, by external conditions, and are amenable in a measure to the power of the will; and the rate and force of their rhythm are much more dependent, than are those of the heart's, on the conditions of distant parts. Nevertheless, essentially and habitually, the medulla oblongata is not only the nervous centre, but the rhythmic nervous centre, of the respiratory movements. When a certain portion of it is destroyed, the proper respiratory rhythm ceases, and nothing can renew it, whatever stimulus or variety of external conditions be present. Any of the respiratory muscles may be again stimulated to contraction; some of them will again, and for a few times, contract repeatedly and at regular intervals, imitating the old rhythm*; but there is no longer any combination or timely succession of the movements of the many respiratory muscles, as for their proper purpose.

* Such surviving rhythmic movements of the respiratory muscles may, perhaps, be ascribed to the continuance of the rhythmic nutrition, of which mention is made in a later part of the lecture, and which, though not the primary cause of their rhythmic action, must coexist with it; but other than the respiratory muscles may thus for a time act rhythmically when dying, or after their nerves are divided. See especially the observations of M. Brown Séquard in his 'Experimental Researches.' The facts may be very difficult to explain, but are not inconsistent with what I offer as the explanation of the rhythmic action of the heart.

In like manner, it may be that the rhythmic movements of the lymphatic hearts in Amphibia depend on a part of the spinal cord as their nervous centre; but this is doubtful*; doubtful I mean, not whether there be a nervous centre for the rhythmic movements, but where it is seated. The case of the respiratory movements, however, is clear, and should be reckoned as of great weight in the argument for the nervous origin of the heart's action.

The experiments on the heart's action which I have shown, have been selected as the simplest and most significant. I pass by many more, and say concerning them only this,—that I believe there are none whose results are inconsistent with the belief that I have expressed as to the cause of the rhythmic action of the heart. There are some, of which the results are difficult to explain; but the difficulties relate to questions of nerve-physiology†, and do not affect the simple conclusion that the rhythmic action of the heart in the Vertebrata depends on the operation of nervous centres in the ganglia on or near the substance of the heart: I say, in the Vertebrata; for as yet we know (I believe) nothing of the origin of the corresponding movements in the Invertebrata, though we may well believe that in them, and especially in the lower among them, the rhythmic movements of any pulsating vessel, analogous to a heart, are independent of any nervous system. Such a difference in the two great groups would correspond with similar differences in nearly all other parts of their several economies. The more highly developed the nervous system is, the more are its operations influential in those of every other part and system; and, for the heart, we may suppose that the making-over of its rules of action to a proper nervous system is on purpose that it may the more quickly correspond and sympathize

* See the Experiments of Schiff, Mayer and Budge, in Canstatt for 1850, p. 126; and those of Heidenhain in the same, for 1855. p. 130.

† I refer, especially, to those on the pneumogastric nerves, of which the continued galvanic stimulus stops the heart's action. The phenomena observed in this and similar experiments appear to me very similar to those of *shocks*. As a violent shock of any kind may exhaust the power, or suspend the action, of the brain or spinal cord, so may a shock by violence or galvanic force similarly affect the power of the rhythmic nervous centres for the heart. And the general explanation of all may be, that the nutrition of a nervous centre, and thereby the maintenance of its power, requires rest, and that this rest cannot exist while nerves in relation with it are under irritation.

with other organs, with which, but for their connexion through the nervous system, it could only sympathize more slowly through the medium of the blood.

But, now, it will not have escaped you to question,—why are these nervous centres rhythmic in their action? granting all that has been said, why is it that these nervous centres accumulate and discharge nerve-force, as it would seem, not only spontaneously, but at time-regulated intervals? To put on them, rather than on the heart's muscles, the work of rhythm, is only to put the real difficulty of the matter a step further back.

This is very true; and I will add, that they who hold that the rhythmic, like the ordinary, action of muscles, cannot ensue without stimulus, are in the like predicament, at a step still further back; for how, it may be asked, is the stimulus (of the heart suppose) applied or generated at regular intervals? And the difficulty is not in the case of the heart alone, but in that of all the rhythmic muscular movements. For why, it may be asked, does the medulla oblongata rhythmically excite the respiratory muscles into action? and if it be answered that it is itself stimulated by venous blood, or by impressions on the pneumogastric and other centripetal nerves, then how does the venous blood, or any other substance, thus rhythmically and not constantly stimulate? Constant stimulus, or constant production of an excess of nervous or muscular force, might produce constant muscular action, whether continuous or disorderly, but could not alone maintain a regularly interrupted action, in which the lengths of time of action and of inaction are in definite proportion.

This shifting of the question as to the cause of rhythmic motion suggests that we should enter on a larger inquiry, and take notice of whatever organic processes are performed with rhythm; and this seems the more necessary, while we consider that rhythmic action is not tied to any particular structure, whether muscular or nervous, nor employed in only one or two purposes, as blood-movement or breathing; nor even limited to the animal kingdom: so that, really, the peculiarity we have to study is not one of force, or not one of force alone, but one of *time* as an element in the organic processes. No explanation of the rhythmic action of the heart, therefore, would be sufficient, which did not involve or appear consistent with some general law to which we may refer all other rhythmic organic pro-

cesses, that is, all such as are accomplished with time-regulated alternations, whether of motion or any other change.

Probably, the simplest example of rhythmic motions yet known is that detected by the acute researches of Professor Busk* in the *Volvox globator*. At a certain period of the development of this simplest vegetable organism, there appear, in each zoospore, or in the bands of protoplasm with which the zoospores are connected, vacuoles, spaces, or cavities, of about $\frac{1}{5000}$th of an inch in diameter, which contract with regular rhythm at intervals of from 38 to 41 seconds, quickly contracting and then more slowly dilating again.

The observations of Cohn†, published about a year later than those of Mr. Busk, but independent of them, discovered similar phenomena in *Gonium pectorale* and in *Chlamydomonas*, the vacuoles, like water-vesicles, contracting regularly at intervals of 40 to 45 seconds. The contractions and the dilatations occupy equal periods, as do those of our own heart-ventricles; and in *Gonium* he has found this singular fact, that when, as commonly happens, two vacuoles exist in one cell, their rhythms are alike and exactly alternate, each contracting once in about 40 seconds, and the contraction of each occurring at exactly mid-distance between two successive contractions of the other.

Here, then, we have examples of perfect, and even of compound, rhythmic contractions in vegetable organisms, in which we can have no suspicion of muscular structure, or nervous, or of stimulus (in any reasonable sense of the term), or, in short, of any one of those things which we are prone to regard as the mainsprings of rhythmic action in the heart.

The case of ciliary movements is as simple; the cilia having a perfect rhythm, so that their alternate and opposite movements are of definitely proportioned length; and that, where many move on the same surface, they all keep time precisely, by moving, not all simultaneously, but in time-regulated succession. Here, too, we have no trace of muscular or nervous structure, or of stimulus; yet there is a perfect rhythm in which even myriads of cilia keep time,

* Transactions of the Microscopical Society of London, May 21, 1852.

† Untersuchungen über die Entwickelungsgeschichte der mikroskopischen Algen und Pilze. Breslau, 4to, 1854.

although there is no evident connecting medium between them, nor, generally, any continuity of structure.

I cite these two instances—the ciliary motion and the contraction of the vacuoles or vesicles in the minute Algæ,—as the simplest examples of rhythmic movements—simplest in regard of method, of structure, of apparent uniformity of circumstances, and of spontaneity. And if I were to enumerate all the instances of rhythmic motion that I can find, other than those of hearts, their variety would suffice to show, that for the explanation of rhythm, we must find something much wider than any peculiarity in the structure or nerve-supply of hearts. The time-regulated movements of Oscillatoriæ and their congeners; those of the lateral leaflets in *Desmodium gyrans*, and of the labellum in *Megaclinium falcatum**; the movements of spermatozoa and of their analogues in the vegetable kingdom; the constant rhythm of the nutritive yelk in the ovum of the Pike†; the movements of the pulmograde Acalephæ; the rhythmic actions of the larger veins in the Bat's wing:—all these make a heterogeneous list if we look to structure or to office; their only apparent mark of resemblance is that they are all rhythmical in action, *i. e.* they all observe a rule of time in the manner of their action, a rule in which there is a regulation not only of the times at which successive and alternate actions follow one another, but of those during which each action is continuous.

But there is another thing common to all rhythmically acting organs: they are all the seats of nutritive processes; and I believe that their movements are rhythmical, because their nutrition is so; and rhythmic nutrition is, I believe, only a peculiar instance, or method of manifestation, of a general law of Time as concerned in all organic processes. In other words, I believe that rhythmic motion is an issue of rhythmic nutrition, *i. e.* of a method of nutrition, in which the acting parts are at certain periods raised, with time-regulated progress, to a state of instability of composition, from which they then decline, and in their decline may change their shape, and move with a definite velocity, or (as nervous centres) may discharge nerve-force.

Regarded as phenomena of nutrition, the chief things in which

* Lindley; and Morren in the 'Annales des Sciences Naturelles.'

† Reichert in Müller's 'Archiv,' 1857, p. 46.

rhythmic changes, whether attended with motion or not, may seem strange, are (1) that they are not continuous, but interrupted, and, as it were, alternating between action and inaction, or between progress and regress; and (2) that they are very minutely observant of time. But, in both these regards, they are examples of very general laws of organic processes; alternations of action and of rest, or of opposite actions, being common phenomena of organic life, and all organic processes being regulated with exact observance of time.

In all organic processes, laws of time are observed as exactly as are those concerning weight, and size, and composition.

In the largest view, in that cycle of mutations, according to a parental type, in which the life of every individual (at least among the higher organisms) consists, the successive changes are accomplished with the same exact regard to time as to every other particular. The offspring at each period of its course attains the same condition as the parent at the corresponding period had attained; the rules of weight, of shape, of composition, and of time, are all alike observed in the reproduction of the parent in the offspring.

And, within this view, we may observe how all the parts, or even all the elemental structures, of any organism, keep time in its development. Prematurity is, probably, even more rare than is preponderance, among them.

And so to natural death; for rare as it may be, there is a death even among men, in which, with uniform and synchronous decay, all parts arrive at the same time at the stage of incapacity for work.

It is the same everywhere. How evident is the observance of a law of time in the organic phenomena of the seasons! or, with more minute regulation, in those of sleep and waking, not only among animals, but, much more exactly, in the leaves and flowers of plants; or in their unfoldings; or in the movements of stamina, the dehiscence of fruits and the like, whether in succession or coincidently! In all of these, occurring as they do precisely when they are necessary for the welfare of the individual or the race, we may observe a time-regulation of the organic processes, which, for its precision, passes calculation. For though many of these processes may be very dependent, as to the variations of their rate, upon external conditions, and especially upon variations of light and heat, yet their mean or proper rate is not explained by these conditions, nor is wholly

due to them; a distinct law of periodicity, various according to the species, is observed in all; and even what the external conditions do effect, they effect by their influence on processes of cell-life, which they can alter in respect of time in only the same measure, and with the same limitations, as they can alter them in respect of quantity, or any other character.

Instances of the time-regulation of processes in the animal economy are as evident: witness the returns of thirst and hunger, the regulated times for digestion, the rates of excretions, the daily risings and fallings of temperature, the times for the development of ova, for the growth and development of the uterus, and of all the parts appertaining to gestation and to lactation.

Equally is time observed in processes of disease. It is most evident, in one sense, in all intermittent and other periodic affections; but, in another way, as evident in eruptive fevers. In small-pox, for example, not only is the time set in which the complicated process of eruption will be achieved, but, though every pustule is, in its appearance, independent of the rest, yet all are together in regard of time, because there is the same time-regulation for all. And so, when we watch the progress and the natural remedy of some spreading disease, such as erysipelas or gangrene, the observance of time is, in the whole process, as exact as that of quantity, or any other characteristic of the disease.

I dwell the more on these facts, because, familiar as they are to all, their importance as indicating a general law of organic life seems to be overlooked, and especially their bearing on the explanation of the rhythmic action of the heart has not been duly considered. For not only are many of the processes to which I have referred instances of regular and time-ordered alternations of action, and therefore, in the strict sense, rhythmical; but they are all instances of that exact observance of minute periods of time, which, on a superficial view, appears as the most singular and inexplicable character of the rhythm of a heart. For, in all these cases, the final correctness of the result of the organic process, the punctuality of its event, is proof that it was exactly timed in every stage; that it was, throughout, regulated to the second; just as the action of the heart is, or as the accuracy of a chronometer, at a year's end, is proof, or very nearly proof, that it kept right time in every hour and every second

of the year. In the maturation or the development of an ovum, for example, or in the ripening of seeds, a law of time, various in different species, is observed by all, and their punctuality in arriving at the climax of maturity, after many hours or days or months of progressive change, is evidence that they were chronometric in all their course thitherward. And where many such processes are concurrent, though severally independent, as in the contemporary maturation of all the ovules of an ovary, we may say that their concurrence is like that of many exact chronometers; and their final punctuality, like that of the chronometers, would prove that, in each unit of time, each did a certain and proportionate amount of work.

In all organic processes, then, there is as minute a regulation of time as there is of quantity, or shape, or quality of matter. Time-work is not a singular characteristic of quickly rhythmic organs; it is a rule of life; and its rate in each organism is neither determined, nor beyond certain limits alterable, by external conditions, or by any appreciable qualities of weight or composition (as are the time-relations of inorganic masses); but is determined by properties inherited, and inherent in the very nature of the organism, and is least alterable by external conditions in the highest organisms.

But though the general law of chronometric nutrition (if I may so call it) may be evident, yet it may be objected by some, that it is proved only for such nutritive processes as are long-continuous and cumulative; and that it is an unwarranted assumption to think of a rhythmical or frequently interrupted nutrition. To which objection the answer may be, that whether we regard a rhythmic nutrition as the cause of rhythmic motion or not, we are obliged to hold such a method of nutrition as a fact. For we can be nearly certain that in the heart, as in other muscular, or any other parts, the successive impairments and renovations of composition, which constitute the process of nutritive maintenance, are severally accomplished during the successive periods of action and of repose, all exercise being attended with impairment of composition, such as can be repaired only during repose. Now the only repose of the heart's muscles, and I suppose of its nervous system also, is in the brief intervals between their successive actions; and in these intervals, and, therefore, with a rhythmic nutrition coordinate with its rhythmical action, the heart-structures must recover from the changes suffered

in their actions. Whether, then, as a cause, or as a consequence, where there is rhythmical action there must be corresponding rhythmical waste and repair; for we cannot reasonably suppose that the heart, or any other similarly acting organ, has, as a special prerogative, an exemption from the law of impairment in or by exercise: such an exemption is, indeed, inconceivable.

Now if rhythmic nutrition be thus proved as a necessary attendant of rhythmic action, it must be regarded as the cause, not the consequence, of the action; for in all cases nutrition has precedence of other actions in organized bodies; and the time-regulation of nutrition is a general and principal fact, and is a cause, not a consequence, of many phenomena which we trace in other organs than the heart, and many of which are attended with time-ordered movements.

I suspect that, to many, that which will seem most difficult of acceptance, is the belief that in so quick rhythmic actions as that of the mammalian heart (for example), or that of cilia, there can be a corresponding quickness of alternation of the progressive and retrogressive changes which essentially constitute nutrition. It must be admitted that, when we watch these movements, they appear, at first sight, very unlike anything that can result from nutritive changes, in which we are apt to think of a certain deliberation and quietness. But all rhythmic movements are not thus rapid; and when we watch the actions of a heart reduced to move only once in two or three minutes (as a frog's may be by ligature around the venous sinus), the appearance is like nothing more than it is like that of a process of nutritive changes, in which the structures gradually reach a climax of instability, and then quickly change. Whatever value then there may be in the appearance of a rhythmic action as an indication of its cause, it might be adduced on either side of the inquiry.

But let me add, that the nutritive changes to which I here refer, do not involve the supposition of any rapidly successive making and unmaking of the structures of the rhythmic organ, whether the heart or any other. We have probably held too much of the making and unmaking of elemental parts as essential to their maintenance by nutrition. In the modelling of parts during development and growth, such complete changes probably occur; but in mere maintenance of parts there is no evidence of their frequent or ordinary

occurrence, and to assume it is contrary to the fact, that we rarely find any rudimental structures among the perfect ones. In the most active muscles of the adult, for example, I doubt whether a rudimental or developing fibre could be found; we have sufficient chemical evidence of a constant change of material in them, but no evidence of an equal or parallel change of structure. And so in the blood: the change of material is very rapid, but the change of structures, which we may in some measure estimate by the proportion of white or rudimental blood-cells, is probably slow*. And again, in the secreting glands, excepting those of the skin and the breasts, we have no evidence, and, I think, no sufficient reason to believe, that in all cases the gland-cell-walls dissolve or burst in the act of secretion, so as to need the entire new formation of fresh cells. For we find in most of the active glands no considerable number of either rudimental or degenerate cells; and the observations of Ludwig and Rahn on the secretion of saliva indicate, as many other facts do, that in ordinary secretion (which is the ordinary nutrition of glands) the cell-contents, gradually transformed, flow out through the persistent cell-walls.

Nutritive maintenance, then, probably requires nothing more than molecular substitution. Atoms even of the refuse substance may be passing out, and atoms of the renewing substance passing into places among the structures of a comparatively persistent framework. Cell-walls or their analogues may be long-lived, while their contents are undergoing continual mutation. Such a process of molecular interchange and passage is, indeed, visible in the absorption of oil through the epithelial cells and villi of the intestines; and this is probably only a coarse example of the ordinary manner in which cells change their contents in the nutritive processes. Changes like these may well consist with the quickest rhythmic action.

I would thus, then, conclude as to the most probable explanation of the rhythmic action of the heart:—

1. In the Vertebrata it is due to the time-regulated discharges of

* The very small quantity of iron, in proportion to the quantities of the other constituents of the blood-cells, found in the excretions, is another indication of the comparatively slow waste of the red blood-cells, and may suggest, besides, that in nutritive maintenance there is not an equal mutation of all the component substances of a structure.

nerve-force in certain of the ganglia in and near the substance of the heart, by which discharges the muscular walls are excited to contraction.

2. In Invertebrata, the corresponding pulsatile movements of hearts or vessels are probably independent of nerve-force.

3. The time-regulated rhythmic action, whether of the nervous centres or of the independent contractile walls, is due to their nutrition being rhythmic, *i. e.* to their being, in certain periods, by nutritive changes of composition, raised, with regulated progress, to a state of instability of composition, in their decline from which they discharge nerve-force, or change their shape, contracting.

4. The muscular substance of the heart in the Vertebrata, governed in its rhythmic action by appropriate nervous centres, has a rhythmic nutrition of its own, corresponding and coordinate with theirs; the impairments of its structure during action being repaired in repose.

5. Rhythmic nutrition is a process in accordance with the general laws of organic life, very many organic processes being composed of timely-regulated alternate action and inaction, or alternate opposite actions, *i. e.* being rhythmical, with larger or shorter units of time; and all organic processes being chronometric, *i. e.* ordered according to laws of time as exact, and only as much influenced by external conditions, as are those relating to weight, size, shape, and composition.

ON

SIGNOR CARLO MATTEUCCI'S

LETTER

TO

H. BENCE JONES, M.D., F.R.S. &c.

EDITOR OF

AN ABSTRACT OF DR. DU BOIS-REYMOND'S RESEARCHES

IN ANIMAL ELECTRICITY.

BY

EMIL DU BOIS-REYMOND,

MEMBER OF THE ACADEMIES OF SCIENCES

OF BERLIN AND VIENNA, ETC.

LONDON:

JOHN CHURCHILL, PRINCES STREET, SOHO.

MDCCCLIII.

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS-STREET,
COVENT GARDEN.

CONTENTS.

PAGE

INTRODUCTION 5

§ I. SIGNOR MATTEUCCI'S ATTACKS UPON THE AUTHOR 6

II. REMARKS ON THE PERSONAL ATTACKS AND SCIENTIFIC OBJECTIONS CONTAINED IN SIGNOR MATTEUCCI'S PAMPHLET . 7

III. GENERAL REMARKS ON SIGNOR MATTEUCCI'S CLAIMS OF PRIORITY 9

IV. HISTORY OF THE DISCOVERY OF THE MUSCULAR CURRENT UP TO SIGNOR MATTEUCCI'S FIRST PAPERS ON THAT SUBJECT 11

V. SIGNOR MATTEUCCI'S RESEARCHES ON THE SO-CALLED FROG CURRENT, AND ON THE MUSCULAR CURRENT UP TO THE TIME OF THE AUTHOR'S FIRST PAPER ON THE SAME SUBJECT 12

VI. STATEMENTS ON THE MUSCULAR CURRENT CONTAINED IN THE AUTHOR'S FIRST PAPER 16

VII. SIGNOR MATTEUCCI'S AND THE AUTHOR'S RESPECTIVE CLAIMS TO THE DISCOVERY OF THE LAW OF THE MUSCULAR CURRENT EXAMINED 19

VIII. FURTHER REMARKS ON SIGNOR MATTEUCCI'S CLAIMS TO THE LAW OF THE MUSCULAR CURRENT 23

IX. ON THE NEGATIVE VARIATION OF THE MUSCULAR CURRENT DURING CONTRACTION 27

X. SIGNOR MATTEUCCI'S SUCCESSIVE STATEMENTS REGARDING THE ELECTRIC DEPORTMENT OF MUSCLES DURING CONTRACTION 30

XI. SIGNOR MATTEUCCI'S CLAIMS TO THE DISCOVERY OF THE NEGATIVE VARIATION OF THE MUSCULAR CURRENT DURING CONTRACTION EXAMINED 35

CONCLUSION 38

ON

SIGNOR CARLO MATTEUCCI'S

LETTER

TO

H. BENCE JONES, M.D., F.R.S.

ETC. ETC.

INTRODUCTION.

Signor Carlo Matteucci, of Pisa, lately addressed a pamphlet* to my friend Dr. Bence Jones, reproaching him for having edited an English translation† of Dr. John Müller's (of Freiburg) Abstract of my Researches in Animal Electricity.‡ Copies of this pamphlet were sent to many scientific men of England and Germany, and, probably, of France.

As the statements contained in the Abstract, of which Signor Matteucci complains, are all taken from my German work,§ I naturally feel bound to answer for them. It would be of no use to do so in German, for in Germany my original work can be procured by every one; and in it such proofs are given in support of these statements (WITHOUT ANY EXCEPTION), as do not admit of the slightest doubt regarding their accuracy. With respect to France, I may also, at least for the present, consider any further discussion with Signor Matteucci as superfluous, since a Committee of the Academy of Paris examined into the

* "Lettre de Charles Matteucci à Mr. H. Bence Jones, F.R.S. &c. &c. Editeur d'une Brochure intitulée 'On Animal Electricity,' ou Extrait de Découvertes de Mr. du Bois-Reymond." Florence: Imprimerie Le Monnier. 1853. 8vo.

† "On Animal Electricity: being an Abstract of the Discoveries of Emil du Bois-Reymond," &c. Edited by H. Bence Jones, &c. London: John Churchill. 1852. 8vo.

‡ "Bericht über die neuesten Fortschritte der Physik." Braunschweig: 1849—1852. S. 768.

§ "Untersuchungen über thierische Elektricität." Berlin, Verlag von G. Reimer. Bd. I., 1848. Bd. II., Abth. 1, 1849. 8vo.

question three years ago, and gave a verdict by which I am most willing to abide.

In England, on the contrary, the matter is as yet almost new, and requires, perhaps, some elucidation on my part in order to be judged correctly. This information I have endeavoured to give in the following pages. In going over them, the reader, I trust, will remember the fatality naturally attached to the condition of every defendant, namely, that often many pages may be necessary for confuting an accusation expressed in a single line.

As I had no means of knowing who among the scientific men of England were favoured by Signor Matteucci with copies of his pamphlet, I have been obliged to give my reply as wide a circulation as possible by the ordinary way of publication.

§ I. Signor Matteucci's former Attacks upon the Author.

It may be inferred from the preceding that it is not the first time that I have been the subject of Signor Matteucci's attacks. He has written against me thrice previously, and each time he has been the assailant.

The first time was in 1845.* I had mentioned his name once previously in my first paper of 1843, and I did so very respectfully. I did not answer this first attack explicitly, although it was an exceedingly violent one. Some years later, it is true, I reviewed Signor Matteucci's papers, in the Report of the Physical Society of Berlin,† and in my German work, and of course made the best of my defence. But as these strictures were written in German, they seem to have escaped his attention at the time. It was not until the spring of 1850, when I communicated two papers to the Academy of Paris,‡ in which he was faithfully and respectfully quoted, that he attacked me again in the

* "Annales de Chimie et de Physique." 3e Série, t. xv., p. 64. "Archives de l'Electricité," par Mr. de la Rive, t. v., p. 382.

† "Die Fortschritte der Physik im Jahre 1845." Dargestellt von der physikalischen Gesellschaft zu Berlin. I. Jahrgang. Redigirt von Dr. G. Karsten. Berlin. 1847. S. 492. See also the same work, II. Jahrgang (1846). S. 436. III. Jahrgang (1847). S. 392.

‡ "Comptes-rendus," &c. 25 Mars et 8 Avril 1850. t. xxx., p. 349 et 406. "Annales de Chimie et de Physique." 3e Série, t. xxx, p. 119 et 179.

"Comptes-rendus."* I was allowed to answer him in the same journal, and I believe I did so in the most irrefragable manner.† Nevertheless, some time afterwards, Signor Matteucci published a pamphlet in French against me at Florence, very similar to that which is now under consideration, containing, to a great extent, the same personal charges, the same objections to my methods of investigation and to my experimental results, and making the same claims to priority.‡ Copies of it were likewise distributed to many scientific men of Europe. By favour of the Academy of Paris, it was printed in the "Comptes-rendus," notwithstanding its length exceeded the limits prescribed to communications of persons not belonging to the Academy.§ I opposed him again in the same journal with the most forcible arguments,‖ and, moreover, the above-mentioned Committee of the Academy decidedly espoused my cause.¶ And yet Signor Matteucci now enters the lists once more, bringing forward the very same charges which I have already so often, and, as I thought, so satisfactorily confuted.

I am desirous of mentioning these facts, because Signor Matteucci does not allude to them in his last pamphlet, so that any one unacquainted with the circumstances might easily be led to suppose that I have provoked this controversy.

§ II. Remarks on the Personal Attacks and Scientific Objections contained in Signor Matteucci's Pamphlet.

I do not mean to reply by personal abuse to the language which Signor Matteucci has used.** I shall not at present even

* "Comptes-rendus," &c. 22 April 1850. t. xxx., p. 479.

† "Comptes-rendus," &c. 29 Avril et 6 Mai 1850. t. xxx., p. 512 et 563.

‡ "Réponse aux deux dernières Lettres de M. du Bois-Reymond;" insérées dans les Nos. 17 et 18 des "Comptes-rendus" de l'Académie, et en général à toutes les observations faites par le même auteur sur quelques-unes de mes recherches d'électro-physiologie: présentée à l'Académie des Sciences, par M. Matteucci. Florence: Imprimerie Le Monnier. 8vo.

§ "Comptes-rendus," &c. 3 Juin 1850. t. xxx., p. 699.

‖ "Comptes-rendus," &c. 22 Juillet 1850. t. xxxi., p. 91.

¶ "Comptes-rendus," &c. 15 Juillet 1850. t. xxxi., p. 28. The Commissioners were MM. Magendie, Becquerel, Despretz, Rayer, and Pouillet. The latter was the Reporter of the Committee.

** Signor Matteucci, *e.g.*, charges Dr. John Müller, Dr. Bence Jones, and myself, with a lie (mensonge, p. 8), for having said that "*he always preserved the nervous trunks, and a piece of the spinal column when forming his frog batteries*" (Abstract,

reply again to his scientific objections, but I shall content myself with saying that Signor Matteucci states that these objections are only preliminary ones, as he is going to review the whole of my "Researches" in the new edition of his "Traité" (pp. 3, 14), which he is about to publish. Moreover, as far as he explains himself, he still maintains, as he always did, that my methods of investigation are bad and that my results are wrong. He persists, in short, in contesting nearly all the facts concerning animal electricity, which he has not been fortunate enough to discover himself, as, *e.g.*, the nervous current and the whole world of phenomena which depend on it, (p. 14.) Signor Matteucci thus contests facts which have now been witnessed by the most illustrious physicists and physiologists of England, France, and Germany; facts which were carefully examined and emphatically authenticated by a Committee of the Academy of Paris; facts, indeed, which of late years are demonstrated in public lectures in most German universities, by men like Helmholtz, Ludwig, Bischoff, Vierordt, &c.

I therefore cannot consider it necessary to contend for their accuracy here, or to defend the methods by which these facts were obtained. To give a specimen of the manner in which Signor Matteucci judges of these methods, he ventures to suggest in plain terms, that the only motive why I experiment on a single nerve or muscle of a frog, instead of on batteries formed of twenty legs of frogs, as he did, in imitation of Bunzen* and Nobili,† is that I want to proceed differently from him.

p. 76). He says:—"*On peut lire dans mes Mémoires, que je n'ai laissé les filets nerveux dans mes piles musculaires que quand j'ai voulu en étudier l'influence.*" The passage of the "Abstract" in which Signor Matteucci ventures to point out a LIE, forms part of the chapter which explicitly treats of the so-called frog-current, and most evidently does not relate to what he calls "*piles musculaires*," but to what he calls "*piles de grenouilles*." That passage is so perfectly true in reference to the piles de grenouilles, that the nerves and pieces of the spinal column may even be seen in the diagram which Signor Matteucci gives of such a pile in his "Traité des Phénomènes électro-physiologiques des Animaux." Paris, 1844. Planche II., fig. 16. He also says in the same work, p. 99:—"*Afin d'étudier l'influence des différents parties de la grenouille dans le courant propre, j'ai commencé par enlever tout-à-fait les deux nerfs spinaux et le morceau de moelle épinière,*" &c. Hence it follows that the usual mode of preparing the frogs was, to preserve the nervous trunks and the pièce of the spinal column, for otherwise he would not have been able to remove them in this particular case.

* Gilbert's "Annalen der Physik." 1807. Bd. xxv., S. 155 (1805).

† "Annales de Chimie et de Physique." 1828. t. xxxviii., p. 238.

"*La seule raison, qui est bien loin d'être scientifique, par laquelle on fait tant d'efforts pour employer dans les expériences d'électricité musculaire une autre méthode, laquelle consiste principalement à supprimer la pile musculaire et à y substituer un seul morceau de muscle et un galvanomètre excessivement délicat, n'a pu être que celle d'opérer différemment de ce que j'avais fait,*" (p. 6.) I think I need not answer such an assertion. New and valuable results, obtained by means of an experimental method, certainly are the best proof of its excellency; and a great many judges, both competent and impartial, have told me that my methods fairly stood that test.

§ III. General Remarks on Signor Matteucci's Claims to Priority.

I now turn to the main subject of Signor Matteucci's letter—viz., to his claims to priority.

Signor Matteucci first denounces the author of the Abstract for purposely omitting to refer to some of his papers anterior to mine, which papers, in his opinion, entitle him to lay claim to some of my experimental results. I venture to assert that such a proceeding would not for a moment have been entertained by Dr. John Müller, Dr. Bence Jones, or me. For my part, I may say that there is scarcely a single line, not only of Signor Matteucci's almost innumerable memoirs on animal electricity, published in the English, French, and Italian periodicals, but even of the whole literature of electro-physiology, which I have not carefully perused, scrupulously weighed, and at last faithfully noted at its exact worth in my German work. Signor Matteucci, who says he has "*écouté la lecture des deux gros volumes de M. du Bois-Reymond*," (pp. 9, 14, 15,) has had the means of knowing that I have done so.

Signor Matteucci's accusation against the German author and English editor of the Abstract is rather inconsiderate. He says:—"*Vous prétendez dans ce chapitre composer l'histoire de mes travaux d'électro-physiologie, et vous vous arrêtez à l'année* 1838, *qui est justement l'époque à laquelle mes travaux sur le courant propre et le courant musculaire de la grenouille et sur la contraction induite ont commencé. Malheureusement il n'est que*

trop vrai qu'avant cette époque mes publications étaient trop pressées et imparfaitement étudiées," &c., and after having quoted several papers which Dr. Bence Jones, according to him, ought to have mentioned, he continues:—"*De tout cela vous n'avez pas dit un mot dans le chapitre qui paraît destiné à donner l'histoire de mes recherches,*" (p. 2); and pp. 6, 7:—"*Tout cela a été oublié dans l'histoire que vous faites de mes travaux d'électrophysiologie qui précèdent ceux de M. du Bois-Reymond.*" Now, everybody who will take the trouble of opening the Abstract at p. 95, will find there a whole chapter (Chapter XII., entitled "On the discovery of the law of the muscular current,") solely devoted to a minute analysis of those papers. Signor Matteucci afterwards notes it himself. He says, p. 8: "*Dans le chapitre XII je suis cité plusieurs fois,*" &c. Yet he did not retract his accusation.

Again, Signor Matteucci says, p. 11:—"*Au chapitre XVI il est dit que je n'ai pas prouvé l'existence du courant musculaire dans les animaux vivants: cette assertion est évidemment contraire à la vérité, car j'ai démontré l'existence du courant musculaire sur les grenouilles et sur les pigeons vivants.*" Now, opening the Abstract at the page 125, the reader will find: "Chapter XVI. On the current of the live UNDISSECTED frog. Matteucci long since obtained currents from the DISSECTED muscles of frogs, whilst they were still in connexion with the living body of the animal. But these experiments do not prove, EVEN IN MATTEUCCI'S OWN OPINION, that the current existed in the animal previous TO ANY DISSECTION or experimental arrangement. Du Bois-Reymond has succeeded in demonstrating the current in the live frog WITHOUT ANY DISSECTION." The phrase, "but these experiments do not prove, EVEN IN MATTEUCCI'S OWN OPINION," &c., alludes to the following statement, contained in Signor Matteucci's "Traité," p. 123:—*Chap. IX. Vues théoriques sur la cause du courant électrique musculaire. Les résultats auxquels nous sommes parvenus sont bien loin de prouver l'existence de l'électricité libre dans les animaux vivants.*"

Such are the foundations on which Signor Matteucci grounds his defamatory assertions against two honourable men, one of whom, it is true, is my countryman, but, beyond that, utterly a stranger to me; the other, it is true, is now my intimate

friend, but not my countryman, and connected with me only by the interest he took in my researches!*

§ IV. HISTORY OF THE DISCOVERY OF THE MUSCULAR CURRENT UP TO SIGNOR MATTEUCCI'S FIRST PAPERS ON THAT SUBJECT.

In order to make the nature of Signor Matteucci's claims better understood, it will be necessary once more to trace here the history of the discovery of the muscular current and of its negative variation during the contraction of the muscle.

The first phenomenon ever observed in reference to the muscular current were the contractions of the limbs of frogs, which Galvani sometimes obtained on completing the circuit between the nerves and muscles, without using metals.† Humboldt noticed similar facts on several animals, both cold and warm-blooded.‡ Volta soon succeeded in more closely determining the conditions requisite for obtaining those contractions. He showed that the nerves should be made to touch the aponeurosis of the tendo Achillis on the back of the gastrocnemius muscle, instead of the muscle itself. The efficacy of this arrangement he explained in accordance with his general theory of electro-motive action by the circumstance of a third heterogeneous body—viz., the tendinous fibre—being interposed between the nerve and the muscle. On making the nerve touch the muscle itself, the limb, according to Volta, will be convulsed only when some contamination, either accidental or intentional, at the place of contact plays the part of the third heterogeneous body, which is requisite for the production

* Here is another instance of the same recklessness. Signor Matteucci reproaches me for not having, in my first paper, described the galvanometer employed (p. 11). Now, on the first page of that paper, I have said—"All my experiments, of course excepting those in which the rheoscopic limb was used, were made with a galvanometer of the usual construction, around the frame of which a copper wire, a kilometer long and ·0065 inch of Paris in diameter, forms 4560 coils." And from what follows it may be seen that the sensitiveness of the astatic system was regulated according to Signor Melloni's method of compensation. ("Poggendorff's Annalen," u. s. w. 1843. Bd. lviii., S. 1. Anm.)

† "Dell' Uso e dell' Attività dell' Arco conduttore nelle Contrazioni dei Muscoli." In Bologna, 1794, 4°, p. 82.

‡ "Versuche über die gereizte Muskel-und Nervenfaser," u. s. w. Posen und Berlin, 1797. Bd. i., S. 36, 37.

of the current.* Such was the authority which the invention of the pile soon after bestowed even upon the less correct views of the author, that his opinion prevailed among physicists and physiologists up to our time,† and, in fact, prevented them from investigating the matter further, by shutting their eyes to its real importance.

It was not until 1827, indeed, that Nobili, being then engaged in improving the galvanometer, tried, also by means of this instrument, to detect traces of the current which convulses the frog's limbs in the above-mentioned experiments. He made the feet of a frog, prepared after Galvani's method, dip into one of the conducting vessels of the galvanometer, the sciatic plexuses touched the other, and he observed that the needle was deflected so as to indicate an UPWARD current—that is, a current, directed in the frog's legs, from the feet to the sciatic plexuses (Abstract, p. 68). He moreover found that the intensity of this current, which he called FROG-CURRENT, may be increased by increasing the number of frogs brought into the circuit, thus forming, as it were, a voltaic pile of frogs. As to the origin of the current, Nobili, doubtless ignorant of the peculiar efficacy which Volta discovered in the expansion of the tendo Achillis in Galvani's experiment without metals, strangely conjectured that the frog-current was a thermo-electric current, and arose from the more rapid cooling which he supposed the smaller mass of nerves underwent in comparison with the slower cooling of the larger mass of muscles.‡

§ V. Signor Matteucci's Researches on the so-called Frog-current, and on the Muscular Current up to the Time of the Author's First Paper on the same Subject.

Nobili's discovery passed almost entirely unnoticed; and the question remained in that state for about ten years longer, when

* "Collezione dell' Opere del Cav. Conte Alessandro Volta." Firenze, 1816. t. ii., p. I., p. 256.

† See Moser in "Dove's Repertorium der Physik." Bd. i., 1837. S. 251.—Prevost, in the "Archives de l'Electricité," &c. 1842. t. ii., p. 633.—Longet, "Traité de Physiologie." t. ii. Paris, 1850. t. ii., p. 130, Note 2.

‡ "Annales de Chimie et de Physique." 1828. t. xxxviii., p. 225; 1830, t. xliv., p. 60.

Signor Matteucci began to work at it. He most undoubtedly has the merit, and I have always given him the credit of having first discerned the importance of the phenomena in question, and the insufficiency of the theories previously proposed, and of having prosecuted their further examination. In 1840, Signor Matteucci published at Paris a compilation of his papers on Animal Electricity, under the title: "Essai sur les Phénomènes électriques des Animaux." The principal step which in this book is made beyond Nobili's range of knowledge consists in the remark, that in order to obtain the frog-current it is not at all necessary to prepare the frog according to Galvani's method, and to connect its nerves and muscles by the galvanometer wire, but that it is sufficient to deprive the frog of its skin and to connect certain points of its body by that wire, as, *e. g.*, its head or back with its legs. The current appears in the same direction as in Nobili's original experiment (Essai, p. 76): "This discovery, as it is said in the Abstract, p. 26, entirely overthrew the old Voltaic doctrine and Nobili's theory of thermo-electricity, as well as the idea of any electro-chemical action."

Signor Matteucci sent a copy of his work to Baron Humboldt. Baron Humboldt gave it Professor John Müller, the celebrated physiologist of Berlin, who gave it to me in the spring of 1841, inviting me to repeat Signor Matteucci's experiments, and, if possible, to extend his researches. Such was the origin of my labours on Animal Electricity. In November, 1842, I communicated a paper to Professor Poggendorff, containing the results I had obtained up to that time. This paper was printed in the "Annalen der Physik und Chemie," Bd. lviii., S. 1, January, 1843, under the title: "A Preliminary Account of an Investigation of the so-called Frog-Current, and of the Electric Fishes."

In the meanwhile, however, Signor Matteucci also had published new observations. In September, 1841, he made the following statement:—*La* PARTIE INTERNE D'UN MUSCLE, *mise en communication avec une autre partie quelconque du même animal,* NERF, SURFACE DU MUSCLE, PEAU, *&c., produit un courant qui va dans l'animal de la partie musculaire à la partie qui ne l'est pas.*"* This statement Signor Matteucci usually quotes as the

* "Comptes-rendus," &c. 6 Septembre 1841. t. xiii., p. 540.

first in which he gave an account of the law of the muscular current. But the interior and the surface of the muscle, both forming part of the muscle, evidently the direction of the current in the muscle is not unequivocally determined even in that passage.

In February, 1842, Signor Matteucci repeated the same statement, adding, however, some details:—"*De tous les résultats obtenus sur les grenouilles et sur des animaux à sang chaud, on peut tirer cette conclusion:* 1° *Que la grenouille et les animaux à sang chaud donnent un courant électrique lorsque la partie interne d'une masse musculaire et sa surface sont mises en communication avec un arc conducteur;* 2° *Que le nerf qui appartient à une masse musculaire, et tout le système nerveux en général, peuvent faire l'office de la partie interne d'un muscle dans la production de ce courant;* 3° *Que le courant est dirigé, dans l'animal,* DE L'INTÉRIEUR DU MUSCLE OU DE SON NERF A SA SURFACE OU A SON TENDON."* Thus far, Signor Matteucci's new statements were known to me when I published the paper above-mentioned, in which, of course, they are duly quoted and even discussed at length.

These statements were literally taken from an extensive paper, which was afterwards printed at full length in the second volume of the "Archives de l'Electricité," edited by M. de la Rive, but was not published before the 3rd of November, 1842. Signor Matteucci in this paper first described that experimental arrangement which he has since called "*pile musculaire*," and which he still considers as most appropriate for the demonstration of the electric properties of muscles. The pile musculaire consists of a certain number up to twenty thighs of frogs, roughly cut across, and disposed in the manner represented in the annexed diagram. By connecting the two ends of the

FIG. 1.



[*Simplest case of the Muscular Current observed by Signor Matteucci, up to January, 1843. Published November, 1842.*]

* "Comptes-rendus," &c. 21 Février 1842. t. xiv., p. 315.

battery—viz., the stump of the knee in *a*, and the cross section of the thigh in *b*, by the galvanometer wire, a current is obtained in the direction of the arrow.*

This current was considered by Signor Matteucci as an entirely new phenomenon, quite distinct from the frog current;—nay, so wholly unconnected with it, that he admitted that the muscular current was found in all animals, including the frog, whereas the frog current is said by him to belong to the frog exclusively. He seems to have ascribed to this animal, although unprovided with any peculiar organ for that purpose, an electromotive power similar to that of the electric fishes.† Even as late as 1844, Signor Matteucci, after having developed some hypothetical views concerning the muscular current, says: "*Ces vues hypothétiques ne peuvent pas malheureusement s'étendre aussi bien aux phénomènes du courant propre de la grenouille.* J'AI CHERCHÉ INUTILEMENT L'EXISTENCE D'UN COURANT ANALOGUE AU COURANT PROPRE DE LA GRENOUILLE, DANS UN TRÈS GRAND NOMBRE D'ANIMAUX, *je n'ai jamais trouvé que le courant musculaire. Les animaux les plus rapprochés de la grenouille, des salamandres, des anguilles, des tortues, ne m'ont jamais donné que le courant musculaire; dans tous les cas, il a fallu invariablement, pour obtenir les signes d'un courant électrique, avoir dans le circuit l'intérieur du muscle et sa surface.* DANS LA SEULE GRENOUILLE ON TROUVE EN MÊME TEMPS LE COURANT MUSCULAIRE ET LE COURANT PROPRE *qui existe, sans altérer le muscle, sans mettre à nu sa partie interne, et qui est dirigé de la surface du muscle à son nerf dans l'animal.*"‡ Signor Matteucci, in these papers, therefore also admits the possibility of the two currents—viz., the frog and the muscular current—counterbalancing each other in the frog's leg, so that at one time the former, at another the latter, may prevail in the circuit.§

* "Archives de l'Electricité," t. ii., pp. 434—436.

† "Essai sur les Phénomènes électriques des Animaux." Paris, 1840. p. 84.

‡ "Traité des Phénomènes électro-physiologiques des Animaux." Paris, 1844. pp. 126, 127.

§ Signor Matteucci, in his last pamphlet, p. 8, appeals to Galvani, Humboldt, and Nobili, as having likewise considered that the frog current was a phenomenon exclusively belonging to the frog, in order to exonerate himself for having once assented to that opinion. But in doing so he is altogether mistaken. Nobili, who ascribed a thermo-electric origin to the frog current, of course did not even consider it as a phenomenon confined to the animal kingdom, much less to a single species. As regards Galvani and Humboldt, almost every line of their writings is sufficient to prove that throughout their experiments on frogs they tried to detect general facts,

§ VI. Statements on the Muscular Current contained in the Author's First Paper.

Such were Signor Matteucci's ideas concerning the muscular and the frog current, at the time when my first paper appeared in print, and even much later. The statements on the same subject contained in that paper are the following:—

1. Currents, in all respects similar to the so-called frog current, may be observed in any limb of any animal, whether cold or warm-blooded. These currents, in some limbs, are directed upwards, as in the frog's legs—in others, downwards. They are of different intensity in different limbs. But their intensity and direction are always the same in the same limb of different individuals of the same species.

2. The electro-motive action on which these currents depend does not arise from the contact of heterogeneous tissues, as Volta supposed, for the different tissues, the nerves, muscles, and tendons, in an electric point of view, are quite homogeneous.

3. These currents are produced by the muscles. If any undissected muscle of any animal be brought into the circuit longitudinally, it generally exhibits an electro-motive action, the direction of which depends on the position of the muscle on the ends of the galvanometer circuit, according to the law which will immediately be stated. Thus, the current might be a downward one, or it might be an upward one.* The current of

applicable to the muscles and nerves of all animals. Humboldt accordingly extended Galvani's experiment without metals to the Rana temporaria, Hyla arborea, Lacerta agilis, Mus musculus (see above, p. 11). Galvani himself, it is true, in his experiments without metals, seems never to have forsaken that animal to which he owes the immortality of his name. But Aldini, Valli, and many others made experiments with other animals without ever mentioning that by doing so they passed beyond the limits of Galvani's original ideas. The strange opinion, therefore, that the Rana esculenta is an electric animal comparable to the Gymnotus and the Torpedo, solely owes its origin to Signor Matteucci, and I, in opposing it, only restored the investigation to its former true direction.

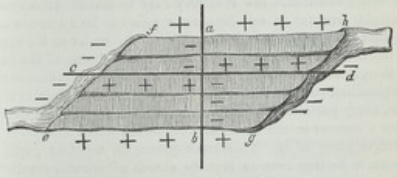
* Signor Matteucci, when enumerating in his last pamphlet the mistakes with which, according to him, my first paper abounds, also says:—"*Il est dit que certains muscles de lapin et de pigeon et de la grenouille même, donnent des courants de direction contraire à celui de la jambe de la grenouille*" (p. 11); and:—"*M. du Bois-Reymond, dans son Mémoire de* 1843 *et dans ses deux volumes publiés longtemps après, a trouvé bien des anomalies dans la direction des courants obtenus des différents muscles entiers de la grenouille*" (p. 10). It is difficult to understand how Signor Matteucci, who asserts that he observed the law of the muscular current, can contest a fact so easily demonstrated by experiment, and so simple to explain by theory. See further on, section 5, p. 18.

the whole limb is nothing but the resultant of the partial currents which are engendered by each muscle of the limb; and the frog current as well as the similar currents observed in other animals, are thus simply reduced to a general MUSCULAR CURRENT. I THEREFORE INVENTED AND FIRST USED THIS TERM, AND IT DOES NOT OCCUR IN SIGNOR MATTEUCCI'S PAPERS ANTERIOR TO MINE.*

4. To make the law, according to which this current may be regularly obtained, more easily understood, it will be useful to premise some definitions.

By LONGITUDINAL SECTION of the muscle, I understand a surface formed only by the SIDES of the fibres of the muscles considered as prisms. By TRANSVERSE SECTION of the muscle I likewise understand a surface formed by the BASE of the fibres of the muscles again considered as prisms. Both the transverse and the longitudinal section may be either ARTIFICIAL or NATURAL ones. In fig. 2, which of course is an ideal one,

FIG. 2.



a section of the muscle through $a\ b$ would be an artificial transverse one; a section through $c\ d$ an artificial longitudinal one. As to the natural transverse section, it is at each end of the muscle formed by the ends of all the fibres, and hidden beneath a coating of tendinous tissue, which is in connexion with the tendon itself, and, in an electric point of view, plays the part of an indifferent conducting body, ($e\ f$, $g\ h$ in the diagram.) Thus, the above-mentioned aponeurosis of the tendo-Achillis on the back of the gastrocnemius muscle

* It is first used by him in the "Comptes-rendus," &c. 23 Janvier 1843, t. xvi., p. 197.

covers the under natural transverse section of that muscle. Finally, the natural longitudinal section of the muscle is that part of its external surface which extends from one natural transverse section to the other, being free from the tendinous coating, and exhibiting the red colour peculiar to muscles, (*f h*, *e g* in the diagram.)

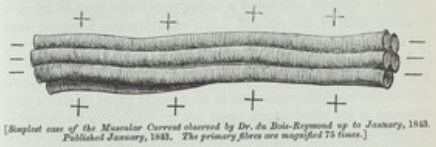
This being thus stated, the law of the muscular current* may be shortly expressed as follows:—ANY POINT OF THE NATURAL OR ARTIFICIAL LONGITUDINAL SECTION OF THE MUSCLE IS POSITIVE IN RELATION TO ANY POINT OF THE NATURAL OR ARTIFICIAL TRANSVERSE SECTION. (See the algebraic signs in fig. 2.)

5. This very important law implied and explained at once all the cases of electro-motive action previously observed in muscles, including Signor Matteucci's latest statements (which, as I have already said, are quoted in my paper as far as I knew them), as well as Volta's long-forgotten remark concerning the peculiar efficacy of the tendon in Galvani's experiment. By means of the above-mentioned law it is very easy to understand why the muscular current in one instance appears to be an upward, in another a downward one; according as the under or the upper of the two transverse sections is made to touch one of the ends of the galvanometer wire, whilst the other end is applied to the longitudinal section of the muscle. Again, according to that law, every particle of a muscle, however minute, ought to produce a current in the same manner as the whole muscle, or as a larger piece of it. This consequence in my first paper is shown to be true even as regards shreds of muscle, consisting

* Signor Matteucci also is accustomed to speak of "the laws of the muscular current." But hitherto he did not mean by these laws, as I do, the general and condensed expression of the conditions, according to which a given muscle may be made to display its electro-motive action, but the detailed statement of the influence which several circumstances, generally of a deleterious or injurious character, exert upon the electro-motive power of muscles ("Comptes-rendus," &c. 3 Juin 1850. t. xxx., pp. 700, 701. "Traité," &c., 1844, pp. 58, 67). My manner of using the term in question is, however, plainly the more correct of the two; and accordingly it not only prevails among German writers, but it is also sanctioned by the Committee of the Academy of Paris ("Comptes-rendus," &c. 15 Juillet 1850. t. xxxi., p. 46). Signor Matteucci himself, in the pamphlet under review, seems to have yielded to some consideration of this kind; for he now, very incautiously indeed, indifferently uses the term in both acceptations (p. 13).

only of a few primary fibres, and such as, accordingly, admit of microscopical investigation, as shown in fig. 3.*

FIG. 3.



[*Simplest case of the Muscular Current observed by Dr. du Bois-Reymond up to January, 1843. Published January, 1843. The primary fibres are magnified 75 times.*]

6. As to the nerves, I have stated in my paper that they are possessed of an electro-motive power, which acts according to the same law as the muscles. Whilst still in organic connexion with the muscles, and forming part of a circuit in which the muscles give rise to a current, the nerves simply play the part of an inactive conducting body, provided their own current be prevented from entering the circuit.

§ VII. SIGNOR MATTEUCCI'S AND THE AUTHOR'S RESPECTIVE CLAIMS TO THE DISCOVERY OF THE LAW OF THE MUSCULAR CURRENT EXAMINED.

In order to facilitate the comparison of Signor Matteucci's statements and those contained in my first paper, I have put both into the following table:—

SIGNOR MATTEUCCI'S STATEMENTS IN NOVEMBER, 1842.	DR. DU BOIS-REYMOND'S STATEMENTS IN JANUARY, 1843.
There are two distinct currents, namely— 1. *The Frog Current.* The leg of the frog is endowed with an upward electro-motive power, peculiar to this animal alone. 2. *A New Current, not yet named.* In the frog and in warm-blooded animals, the surface of the muscle, including the expansion of the tendon, is positive in relation to the interior of the muscle, the nerve which ramifies in the muscle, and the whole nervous system.	There is but one current, namely— *The Muscular Current.* Any part of the natural or artificial longitudinal section of any muscle of any animal is positive in relation to any point of the natural or artificial transverse section. The nervous system, setting aside its own electro-motive power, which obeys the same law as the muscles, plays the part of an inactive conductor with respect to the muscular current.

* Signor Matteucci, who still considers that a pile of twenty thighs of frogs, as shown in Fig. 1, is the best contrivance for demonstrating the muscular current, has ridiculed the statement contained in my first paper, concerning the possibility of

Such, then, was the state of knowledge on both sides at that time. Signor Matteucci, it is true, first pointed out several experimental arrangements in which the artificial transverse section of muscles exhibited a negative electric state in relation to other parts of the animal. His experiments, however, were not even made on simple muscles, but on whole limbs of frogs, including bones, ligaments, tendons, nerves, large bloodvessels, &c.; whereas he never attempted to prove that these different tissues are not heterogeneous in an electric point of view; nay, whereas in his third attack upon me he explicitly owns:—"*Je ne connais pas une expérience qui me prouve que le tendon n'est et ne peut être autre chose qu'un simple conducteur de l'état électrique de la partie intérieure du muscle.*"*

Signor Matteucci, most undoubtedly, was on the threshold of the discovery of the law of the muscular current. So was Signor Grimelli, who nearly at the same time proved that Galvani's experiment without metals succeeds much better on making the sciatic nerve touch the artificial transverse section (*sezione trasversale*), instead of the external surface of the gastrocnemius muscle.† So was, in truth, old Volta, too, when he pointed out the peculiar efficacy of the aponeurosis of the tendo-Achillis, that is, of the under natural transverse section of the gastrocnemius muscle in Galvani's experiment. They all had met with some particular instance of electro-motive action displayed by the muscle, but none of them—and Signor Matteucci no more than any one else—succeeded previously in reducing those particular instances to one general principle, which not only explained all the cases ever observed before, but even led the way to cases yet unknown.

observing the muscular current even on such microscopical shreds of muscle ("Comptes-rendus," &c., 3 Juin 1850, t. xxx., p. 704). After having shown this experiment to most physicists and physiologists of England, France, and Germany, I need not insist on its possibility here. In his last pamphlet, however, Signor Matteucci seems to have erred another way with respect to the same point. He says:—"*D'abord*, ET POUR ETRE VRAI, *on lieu d'un morceau microscopique il faut désormais entendre qu'on parle d'un morceau de muscle qui est employé à former le circuit du galvanomètre*" (p. 10). He, therefore, now seems to think that I have boasted of having performed experiments on microscopical shreds of muscle, whilst, in truth, these experiments were made with whole muscles. I cannot understand how Signor Matteucci was led into this new mistake. A short account of my experiments on microscopical shreds of muscle is given in the "Abstract," p. 100.

* "Réponse aux deux dernières Lettres de M. du Bois-Reymond," &c. Florence. 1850. p. 4. See above, p. 12, note 2, several quotations to the effect of showing that the different tissues are generally considered to be heterogeneous in an electric point of view.

† "Giornale Litterario-Scientifico Modenense." Fascicolo di Aprile e Maggio, 1842.

Signor Matteucci, at the time when my paper was published, had no idea of the negative condition of the natural transverse section of muscles. He supposes that its electric condition is the opposite of that of the artificial transverse section, and that it is the same as that of the natural longitudinal section, whilst, in fact, it is just the contrary.

He, further, had no idea whatever of the electric condition of the artificial longitudinal section. Signor Matteucci, indeed, persists in contending that it is quite indifferent whether the artificial transverse section be so called, as I have proposed, or be called interior of the muscle, as he proposes. But by doing so he clearly shows that even now he does not thoroughly understand that law of the muscular current, which he professes that he discovered. The interior of the muscle, evidently, comprehends the longitudinal section as well as the transverse one. According to Signor Matteucci's statement, therefore, the artificial longitudinal section ought to be found in a negative condition when compared to the natural longitudinal section. I need hardly repeat that such is not the case. The two kinds of longitudinal sections are perfectly homogeneous. Signor Matteucci thus wrongly defined the electric condition of the muscles as if they were comparable to a mass of copper thoroughly coated with zinc; whilst I, in my first paper, in fact, compared the muscles to a bundle of very thin copper wires, the cylindrical surfaces of which were coated with zinc, whilst the bases were left uncovered. And yet Signor Matteucci unhesitatingly writes in his last pamphlet:—"*Toutes mes expériences sur le courant musculaire que j'ai publiées dans mes Mémoires de* 1838 *à* 1842, ET AUXQUELLES ON N'A RIEN CHANGÉ DEPUIS," &c.—(p. 10.)

Again, Signor Matteucci asserts that the nerve which ramifies in a muscle (nay, the whole nervous system) partakes of the negative electric state wrongly ascribed by him to the interior of the muscle. On connecting the nerve and the tendon by the galvanometer wire, a current is said by him to be obtained which proceeds from the former to the latter in the animal. This

pretended current he strangely identifies with the muscular current. I need not say these statements are altogether wrong.*

The reader is now, at length, fully acquainted with the state of the question, and enabled to judge of the justness of Signor Matteucci's complaints. Signor Matteucci is not contented with the fact that I have, on every occasion, proclaimed that he first pointed out the negative electric condition of the artificial transverse section. He further claims the discovery of the law of the muscular current; and yet

1. His statements concerning that law, anterior, nay, even those posterior to mine by some years, have been shown by me to be altogether incomplete and wrong.

2. He does not, even now, rightly understand that law, for he persists in the statement, that the interior of the muscle is negative in relation to its surface.

3. He does not, even now, implicitly grant the identity, in an electric point of view, of the artificial transverse section, and of the expansion of the tendon: "*Il m'est impossible d'admettre sans réserve l'identité du rôle du tendon avec celui de l'intérieur du muscle.*"—(pp. 10, 11.)

4. But, at all events, he explicitly and repeatedly grants that I first pointed out that identity: "*C'est M. du Bois-Reymond qui a publié le premier que le courant soi-disant propre de la grenouille existait dans les muscles entiers d'autres animaux, à la condition de faire arc entre la surface du muscle et son tendon.*"—(p. 5.) And: "*Il a trouvé dans les muscles entiers d'autres animaux, outre ceux de la grenouille, des phénomènes plus ou moins analogues à celui du courant propre, à la suite*

* Signor Matteucci, it is true, says, in his pamphlet, p. 9:—"*J'ai eu pourtant soin de décrire dans mon Mémoire de 1842, les expériences qui prouvent que les fils nerveux se comportent comme des simples conducteurs, semblables à un fil de chanvre ou à un morceau de papier imbibé d'eau;*" and further: "*En citant mon Mémoire de 1842, je viens de rappeler que je ne m'étais pas trompé sur le rôle des nerfs dans les expériences d'électricité musculaire.*" I am sorry to state that Signor Matteucci's memory failed him when he wrote those lines. His first correct statements on the inactive part which the nerves play in the experiments on the muscular current, were published in April, 1843, three months after my first paper. An error of *date* of such importance, I should think, ought not to occur in writings like Signor Matteucci's pamphlet. But this is, moreover, the second time that he has committed this same error. It is also to be found in his third attack upon me ("Comptes-rendus," &c., 3 Juin 1850, t. xxx., p. 701). I cannot refrain from asking what abuse would have been poured upon my friend, Dr. Bence Jones, if he had stated that my first paper appeared in print in 1842 instead of in 1843.

desquels il a été conduit à admettre que les extrémités tendineuses des muscles jouent le rôle dans les electro-moteurs musculaires de l'intérieur de ces tissus. Quoiqu'à cette époque, comme mes Mémoires le prouvent, j'eusse déjà, comme M. Cima de son côté l'avait fait, trouvé cette généralisation du courant propre de la grenouille, je n'ai pas manqué de déclarer que M. du Bois-Reymond avait publié cette découverte le premier." (p. 15; see passages to the same effect at pp. 8 and 11.)

That identity, however, which Signor Matteucci still contests, and, accordingly, so readily yields up its discovery, most evidently was the very thing to be discovered and explained here, and it was, as it were, the very key to the law of the muscular current. So I confidently may leave it with my reader to draw the right conclusion from these premises. His inference, I trust, will be the same as has already been drawn by the scientific men of Germany, and by the Academy of Paris.*

§ VIII. Further Remarks on Signor Matteucci's Claims to the Law of the Muscular Current.

Before leaving this point of the controversy, I beg to add a few more remarks.

1. I cannot forbear once more calling to mind that, setting aside my German publications altogether, I three years ago brought forward very nearly the same arguments as those above in the "Comptes-rendus," in reply to Signor Matteucci's second attack. (See above, p. 7, note 2.) He, of course, was at liberty not to feel satisfied with my defence. But unless he had preferred dropping the controversy altogether, which no doubt would have been the best plan, he ought to have discussed my arguments one by one, and to have shown where they were

* If the law of the muscular current had not been published by me in January, 1843, Signor Antonio Cima, of Cagliari, would be most entitled to its discovery. That philosopher delivered a paper to the Academy of Bologna in December, 1844, in which the undissected muscles of all animals are shown to be possessed of currents like the so-called frog current, and the identity, in an electric point of view, of the artificial transverse section and of the expansion of the tendon, is unequivocally proclaimed. (Antonio Cima, "Saggio storico-critico e sperimentale sulle contrazioni galvaniche e sulle correnti elettro-fisiologiche" in Signor Zantedeschi's "Raccolta fisico-chimica Italiana," &c. 1848. Vol. iii., p. 421.) This paper, as will soon be seen, is three months anterior to the paper of Signor Matteucci in which the same statements are contained. Signor Matteucci himself, in his last pamphlet, grants to Signor Cima the prior claim to these statements. See above, p. 23.)

wrong. Instead of doing so, Signor Matteucci, after having kept silence for some years, now brings forward just the same assertion of priority, without mentioning a single word regarding the reply which I formerly made to him on the same subject.

2. Signor Matteucci, as late as 1845, continued to repeat the erroneous statements contained in his paper of November, 1842. They were reprinted at length in his "Traité des Phénomènes électro-physiologiques des Animaux," Paris, 1844; only in this book the nerves are said to play the part of inactive conductors. In the report of the fourteenth meeting of the British Association, held at York in September, 1844, it is said:—"*M. Matteucci explained the specific voltaic current (courant propre) of the frog as being a current which is detected only in the frog, and which is directed from the feet to the head of the animal.*"*

In April, 1845, at last, more than two years after my first paper had been published, Signor Matteucci wrote in the "Comptes-rendus" as follows:—"*Les résultats les plus curieux, auxquels je suis parvenu dans ces derniers travaux, sont relatifs au courant propre de la grenouille. Je puis maintenant affirmer que ce courant n'appartient pas exclusivement à la grenouille, mais qu'il se manifeste dans tous les muscles de tous les animaux, pourvu que ces muscles présentent à leurs extrémités une terminaison tendineuse inégale. . . . Si j'ai bien compris les derniers travaux anatomiques faits sur la structure des muscles, sur ses rapports avec les tendons et le sarcolème, je ne puis pas hésiter à regarder le courant propre ou du tendon à la surface du muscle, comme le cas le plus simple du courant musculaire.*"† About the same time, Signor Matteucci wrote in the "Philosophical Transactions of the Royal Society" as follows:—"*But why should the proper current belong exclusively to the frog? This is the problem the solution of which I have long been anxious to arrive at, and hope finally to have given a satisfactory answer. . . . . The following are the experiments which led me to generalize the fact of the proper current.*"‡ . . . . And then follow some experiments

* "Transactions of the Sections," p. 38.
† "Comptes-rendus," &c. 14 Avril 1845. t. xx., p. 1098.
‡ "Philosophical Transactions for the year 1845," P. ii., pp. 298, 299.

and considerations, to the effect of showing that currents, similar to the so-called frog current, may be obtained from the muscles of other animals, and that the expansion of the tendon, in an electric point of view, may be considered as representing the interior of the muscle.

In these papers my name is not mentioned. In his last pamphlet, however, Signor Matteucci explicitly declares:—"*Dans mes Mémoires publiés dans les Philosophical Transactions* (1845), *j'ai décrit les expériences faites avec des piles de muscles entiers pris sur différens animaux, et ces expériences ont démontré que ce phénomène appelé jusqu'alors du courant propre, existait dans tous les animaux, et que l'extrémité tendineuse fonctionnait comme l'intérieur du muscle. Il est vrai qu'à cette époque je connaissais le Mémoire que M. du Bois-Reymond avait publié en* 1843. . . *C'est par la lecture de ce Mémoire que j'ai été amené à étudier avec ma méthode l'existence et les lois du courant électrique appartenant aux muscles entiers de tous les animaux.*"—(p. 11.)

These facts need no comment. Signor Matteucci confessedly knew my paper, in which the existence of currents similar to the frog current in the undissected muscles of all animals, and the identity, in an electric point of view, of the expansion of the tendon and of the artificial transverse section are most fully stated; and yet, WITHOUT MENTIONING MY NAME, he wrote these lines:—"*This is the problem the solution of which I have long been anxious to arrive at, and hope finally to have given a satisfactory answer.*" He now confesses:—"*C'est justement par la lecture de ce Mémoire que j'ai été amené à étudier l'existence du courant électrique appartenant aux muscles entiers de tous les animaux;*" and yet, in May, 1845, WITHOUT MENTIONING THAT PAPER, he wrote—"*The following are the experiments which led me to generalize the fact of the proper current.*"

In September, 1845, a letter of Signor Matteucci, directed to M. Dumas, was printed in the "Annales de Chimie et de Physique." In this letter he for the first time attacked me, and also Professor John Müller (of Berlin)—the latter, for having, in his "Elements of Physiology," after a faithful statement of Signor Matteucci's researches, ascribed to me the discovery of

the law of the muscular current.* Signor Matteucci in that letter casually says, and in a rather depreciating tone:—"*Il est juste de dire que, quoique d'une manière incomplète, il a le mérite d'avoir indiqué le premier le rôle de la substance tendineuse dans le courant propre tel que je viens de l'indiquer dans mon dernier Mémoire.*"†

According to his recent avowal, he at least ought to have said, that he was indebted to me for the fundamental idea of that paper. Yet, on the ground of that passage, Signor Matteucci says in his last pamphlet:—"*Je tiens aussi à vous faire remarquer que je n'ai pas manqué de* FAIRNESS *en déclarant publiquement que c'était M. du Bois-Reymond qui avait publié le premier que le courant soi-disant propre de la grenouille existait dans les muscles entiers d'autres animaux, à la condition de faire arc entre la surface du muscle et son tendon.*"—(p. 5.)

Signor Matteucci forgets that he has forfeited the merit of that apparent concession—1. By publishing in the "Annales de Chimie et de Physique" for August, 1846,‡ a translation of his paper in the "Philosophical Transactions," WITHOUT MENTIONING MY NAME; 2. By writing to M. Dumas, in August, 1846, WITHOUT MENTIONING MY NAME:—"*J'ai été satisfait d'être parvenu à démontrer que le courant propre existe dans tous les animaux, qu'il est dû à une structure particulière que j'ai déterminée des faisceaux musculaires* (!), *et qu'il n'est, suivant toutes les probabilités, qu'un cas de courant musculaire;*"§ 3. By communicating the same statement to the British Association at Southampton, in September, 1846,‖ WITHOUT MENTIONING MY NAME; and 4. By stating in his "Leçons sur les Phénomènes physiques des Corps vivants," Paris, 1847, pp. 211, 212, WITHOUT MENTIONING MY NAME:—"*Récemment, en étudiant plus attentivement le courant propre, j'ai pu m'assurer que c'était un phénomène qui appartenait à tous les animaux..... Ayant fixé ainsi les conditions desquelles dépend le courant propre, je crois pouvoir en généraliser l'origine et le réunir au courant musculaire,*" &c.—in short,

* "Handbuch der Physiologie des Menschen." Bd. i., 4. Auflage. Coblenz, 1844, S. 557.
† "Annales de Chimie et de Physique." 3e Série. t. xv., p. 67.
‡ 3e Série. t. xviii., p. 114.
§ "Comptes-rendus," &c. 17 Août 1846. t. xxiii., pp. 356, 357.
‖ "Transactions of the Sections," p. 8. "The Athenæum," No. 987, p. 998.

by repeatedly proclaiming as his own discovery facts which he now confesses that he derived from my paper.

3. Lastly, although Signor Matteucci, in September, 1844, still maintained at York the specific nature of the so-called frog current (see above, p. 24), and although he, at page 11 of his last pamphlet, owns that it was by reading my paper of January, 1843, that he first, in 1845, was led to generalize the fact of that current, he nevertheless, at page 5 of that same pamphlet, says:—"*Il suffit de lire mes Mémoires d'électro-physiologie de cette époque* (*de* 1838 *à* 1842), *pour acquérir la certitude, qu'avant de connaître le Mémoire de M. du Bois-Reymond j'étais parvenu à trouver sur d'autres animaux le courant qu'on* (that is to say, nobody excepting Signor Matteucci) *avait cru d'abord propre de la grenouille seule.*" And at page 15: "*Quoiqu'à cette époque* (that is, at the time when he first read my paper), *comme mes Mémoires le prouvent, j'eusse déjà, comme M. Cima de son côté l'avait fait, trouvé cette généralisation du courant propre de la grenouille,*" &c.

I again abstain from any comment on these facts.

§ IX. On the Negative Variation of the Muscular Current during Contraction.

I now turn to the second subject of Signor Matteucci's claims.

Since the middle of the last century, innumerable attempts have been made to detect any electric phenomenon exhibited by the muscles during contraction. Signor Matteucci himself had made some random experiments on that subject.* They were all made in vain.

In the summer, 1842, as soon as I had mastered the electric phenomena of muscles when at rest, by the discovery of the law of the muscular current, I also turned my attention to that most important problem, and, I am told, was fortunate enough to solve it satisfactorily.

I simply put a muscle into the circuit of the galvanometer, and waited until the needle took up its new position of equilibrium under the influence of the muscular current, whatever

* "Essai sur les Phénomènes électriques des Animaux." Paris, 1840. p. 36.

its direction in the muscle might be, in consequence of the position of the muscle on the ends of the galvanometer circuit, and in accordance with the law of the muscular current. I then proceeded to tetanise the muscle—that is, to make it contract powerfully and uninterruptedly as long as possible, in order to protract the action, and therefore to increase the effect exerted upon the needle by any change which the electric condition of the muscle might undergo during the contraction. In order to effect this the muscle was still attached to its motor nerve, which, however, did not form part of the circuit of the galvanometer, and to which, therefore, any stimulus could be applied without endangering the equilibrium of the needle otherwise than by the very effect to be observed—namely, by any electro-motive action taking place during the contraction of the muscle.

The result was, that at the moment when the muscle was convulsed a sudden and considerable decrease of its action on the needle occurred. This decrease lasts as long as the tetanus itself, and cannot be accounted for by any change of the resistance of the circuit, for it is also obtained when there are two muscles in the circuit, the currents of which counterbalance each other; in this case no perturbation of the electric equilibrium can ever arise from any change of the resistance of the circuit.

Thus far my results on that subject were published in my paper of January 1843 (pp. 12, § 32). It is well known that I have since extended those experiments successfully to the living body of the frog, of the rabbit,* and even of man (Abstract, chap. xix.), and that I have detected a negative variation† of a similar description which the nervous current undergoes while the nerve is conveying to the brain, or to the muscle, those material changes which give rise to sensation and motion. (Abstract, chaps. xxv.-xxx.)

It is well known, too, that by the negative variation I have

* "Monatsberichte der Berliner Akademie." Januar 1853. S. 118.

† Instead of "decrease," I usually say "negative variation" of the muscular current during the contraction, because as yet I have not been able to make out whether during contraction there is only a decrease in the intensity of the current, or whether the direction of the current is reversed. (Abstract, p. 147.) In the following remarks the phenomenon in question, for the sake of brevity, will be called simply—"The negative variation."

succeeded in explaining that interesting phenomenon, first pointed out by Signor Matteucci in October, 1842,* namely, the contraction sometimes shown by the limb of a frog B, called the RHEOSCOPIC LIMB, the nerve of which is placed in any position on another limb A, when the latter is made to contract.

Signor Matteucci considered that contraction as "*a first fact of induction of that force which circulates in the nerves, and which arouses muscular contraction*," and he therefore adopted for it the name, casually bestowed upon it in England, of "*the Induced Contraction*."†

I, on the contrary, proved by the most striking experiments, that the so-called "Induced Contraction" is nothing but the common physiological effect which the negative variation of the muscular current of the primary contracting muscle A exerts on the nerve of the rheoscopic limb B, in accordance with my general law of the excitation of nerves by an electric current. (Abstract, chap. vii.) The nerve of the rheoscopic limb, therefore, ought to be placed on the limb A, so as to complete the circuit between the longitudinal and the transverse section of the muscle, that is to say, so as to be pervaded by the muscular current, otherwise no "Induced Contractions" will occur. As, then, there is no nervous induction acting at a distance here, as Signor Matteucci strangely conjectured, and as, after having given the theory of the phenomenon, I might well feel entitled to alter its name according to the more correct views I had introduced respecting its nature, I ventured to call it "Secondary Contraction" instead of "Induced Contraction," as it was miscalled by its discoverer.‡

Signor Matteucci, in his last pamphlet, says I did so, "*pour lui donner une dénomination différente de celle que j'avais adoptée*" (p. 12). He contests the general law of the excitation of nerves by an electric current (p. 7). He still denies that it is necessary, in order to obtain the secondary contraction, that the nerve of the rheoscopic limb should be traversed by the muscular current of the primary contracting muscle (p. 13).

* "Comptes-rendus," &c. 17 Octobre 1842. t. xv., p. 797.

† "Philosophical Transactions for the year 1845," P. ii., p. 303.

‡ "Abstract," Chapter XVIII. This explanation of the secondary contraction was first printed in Professor John Müller's (of Berlin) "Handbuch der Physiologie des Menschen." Bd. i., 4. Auflage. Coblenz, 1844, S. 557.

He, moreover, still refuses to admit even the electric origin of the secondary contraction (p. 13), and he persists in asserting that the rough and complicated form under which the secondary contraction first occurred by accident, whereby its true nature could not be perceived, is the most simple and most appropriate possible (pp. 12, 13).

With respect to these assertions I can but repeat here what I have already said at the commencement (see above, p. 8), namely, that I cannot deem it necessary again to contend for the accuracy of facts, and for the correctness of views which in France are sanctioned by the Academy of Paris,* and form part of the public instruction in Germany, unless new experiments be adduced against them. As long as Signor Matteucci will content himself with repeating again and again the same statements from time to time, without in the least regarding the arguments against them published by me years ago, I on my part shall not, I hope, be blamed for not taking further notice of his assertions.

§ X. Signor Matteucci's successive Statements regarding the Electric Deportment of Muscles during Contraction.

In his paper of 1838, literally reprinted in the "Essai" (1840), Signor Matteucci said:—"*Une autre cause qui modifie grandement le courant propre de la grenouille, c'est son état tétanique. . . . L'influence du tétanos est telle que le courant propre manque toujours lorsque la grenouille en est attaquée. Nous n'avons plus de contractions, ni de signes au galvanomètre.*"†

I therefore thought he had been fortunate enough to observe the negative variation before me, and accordingly I said, in my first paper, before relating the above-mentioned experiments: "Matteucci, in his 'Essai,' has stated that the frog current disappears DURING the tetanus."

By saying this, however, I was far from doing justice to myself.

* In the Report of the Committee of that Academy, concerning my Researches, it is said: "*Ce fait fondamental* (the negative variation) *donne une explication directe de la contraction induite de M. Matteucci.*"—"Comptes-rendus," &c. 15 Juillet 1850. t. xxxi., p. 36.

† "Bibliothèque universelle," &c. Mars 1838. Nouvelle Série. T. xviii., p. 164. "Essai," &c., pp. 81, 82.

The following quotations will show most clearly that Signor Matteucci, when writing the passage of the "Essai" just alluded to, and even a long time after the publication of my first paper, had not the slightest idea of the negative variation. He himself, indeed, has since given quite another interpretation of that passage.

In his paper of September, 1845, in which he first attacked me, he says, with regard to my explanation of the secondary contraction, with which he had become acquainted through Jourdan's French translation of John Müller's "Elements of Physiology":—"*M. du Bois-Reymond pense que le courant musculaire est interrompu par la contraction, et que cette interruption doit déterminer une rupture d'équilibre dans le nerf de la seconde préparation, et par conséquent, amener la contraction du second muscle. Ce sont les mêmes expressions avec lesquelles M. Müller rapporte l'hypothèse de M. du Bois-Reymond, faite pour expliquer la contraction induite. Il m'a été impossible de me faire une idée de la valeur physique de ces expressions.* IL PARAÎT QUE M. DU BOIS-REYMOND ADMET QUE LE COURANT MUSCULAIRE OU PROPRE S'AFFAIBLIT OU S'INTERROMPT PENDANT LA CONTRACTION MUSCULAIRE; MAIS IL N'Y A AUCUN FAIT QUI VIENNE A L'APPUI DE CETTE IDÉE."

Signor Matteucci, therefore, in September, 1845, pronounced that the fact of the negative variation, most clearly and explicitly stated in my first paper, was an hypothesis gratuitously invented by me in order to account for his so-called "Induced Contractions!"

Signor Matteucci continues:—"*Galvani avait bien vu que les signes des contractions propres s'affaiblissent ou disparaissent dans la grenouille prise de tétanos: moi-même j'ai bien confirmé ce fait; mais il faut remarquer que cet affaiblissement se montre, parcequ'on prend pour indication du courant propre la contraction de la grenouille même, en repliant sa jambe sur les nerfs lombaires.* MAIS ON NE TROUVE PAS CELA EN MESURANT LE COURANT PROPRE OU LE MUSCULAIRE AVEC LE GALVANOMÈTRE. *La différence est donc due à l'état d'excitabilité du nerf dans l'animal tétanisé. Ce n'est que dans un cas, que j'ai noté dans mes premiers travaux et que j'ai vérifié après, que l'on pourrait trouver la preuve du principe admis par M. du Bois-Reymond. J'ai trouvé que les grenouilles prises dans l'état de surexcitation*

développée par l'emploi de la noix vomique, preparées à la manière ordinaire et disposées en pile, donnent un courant propre plus faible que celui que l'on obtient en agissant sur des grenouilles qui n'ont pas subi l'action de la noix vomique. Mais, si l'on réfléchit QUE LES CONTRACTIONS NE PERSISTENT PAS DANS LES GRENOUILLES PRÉPARÉES ET DISPOSÉES EN PILE, *on ne pourra pas voir, dans ce fait unique, la démonstration du principe invoqué par M. du Bois-Reymond."**

Hence it may be seen that Signor Matteucci's statement, in the "Essai," concerning the influence of the tetanus on the muscular current, referred to quite another subject than at first glance it seemed to do. That statement, in truth, is explicitly acknowledged by its author solely to refer to the state of the current AFTER the tetanus has ceased. Signor Matteucci only wanted to say that the current of a frog, the muscles of which are exhausted by previous tetanic contractions, proves weaker than the current of a fresh frog, as might have been easily foreseen.

If after this any doubt could be entertained as to the true import of the passage of the "Essai," it would be removed by the statement, that during the years 1843—50, subsequent to the publication of my first paper, Signor Matteucci has made repeated attempts to detect some electric phenomenon in the muscles during contraction, without ever alluding by a single word to the passage of the "Essai" as bearing upon the same subject.

In January, 1843, Signor Matteucci wrote in the "Comptes-rendus"—"*Les signes du courant propre de la grenouille, démontrés par le galvanomètre,* AUGMENTENT *au même instrument dans l'acte de la contraction.*"†

In his "Traité," &c., published in 1844, Signor Matteucci repeated the same statement, and more fully detailed the experiments which led him to it. His object in trying these experiments was chiefly to account for the secondary contraction, to which, at that time, he was still inclined to ascribe an electric origin. He prepared ten frogs after Galvani's method—that is,

* "Annales de Chimie et de Physique." Septembre 1845. 3e Série, t. xv., pp. 69, 70.

† 23 Janvier 1843. t. xvi., p. 197.

leaving the sciatic plexuses and a piece of the spinal column. He put them into the circuit so as to form a pile; the piece of the spine of each of the frogs rested on the feet of the following one. When the needle had taken its new position of equilibrium, under the influence of the frog current, Signor Matteucci excited contractions by wetting the nerves with a solution of caustic potassa. He says—"*Aussitôt l'alcali appliqué, les contractions commencent et l'aiguille, dans le même temps, s'avance de 5°, 6°, et quelque fois de 10° au delà de l'angle où elle était fixée. Quelques secondes après les contractions cessent, et l'aiguille revient à sa position pour descendre ensuite lentement vers le zéro comme elle l'aurait fait sans la déviation extraordinaire. Il faut avouer que l'apparition de deux phénomènes, c'est-à-dire, la production des contractions et la plus grande déviation de l'aiguille, se correspondent exactement. . . . . Est-il maintenant bien exact de conclure que le courant propre augmente par la contraction du muscle? Peut-on expliquer différemment le résultat de nos expériences?*"*

Signor Matteucci, indeed, and with good reason, felt uneasy about the question whether the increase of the current at the moment of the contraction was not simply due to the decrease of the resistance of the circuit caused by the alkali; and as he did not succeed in devising a proper method for avoiding that cause of error, he at last despondingly concludes:—"*Je n'ose pas affirmer que la question soit complétement résolue, et je me suis arrêté, ne sachant pas par quelle voie avancer pour la résoudre.*"†

In the same work, however, the statement of the "Essai" is also reprinted, in which the tetanic convulsions are said to impair the intensity of the frog current,‡ without any connexion whatever being understood to exist between the two statements; and in fact no such connexion existed in their author's mind, for the prior and seemingly correct statement of the "Essai" referred only to the state of the current after the tetanus had relaxed; the more recent and erroneous statement, on the contrary, referred to the state of the current during the act of contraction itself.

In his paper "On Induced Contractions," printed in the "Philosophical Transactions" for 1845, Signor Matteucci resumed these experiments, and finally succeeded in satisfying himself that the increase of the current formerly observed by him to

* "Traité," &c. 1844, p. 138. † Ibid. p. 141. ‡ Ibid. pp. 109 110.

take place during the contractions was only due to the decrease of resistance caused by wetting the nerves with alkali. He therefore then says—"*Let us then conclude that direct experiment answers negatively to the question we proposed to solve, whether there were evolution of electricity in muscular contraction.*"* And this statement he has since repeated several times—first, in his letter to M. Dumas, of September, 1845:—"*J'ai varié de mille manières les expériences pour découvrir s'il y a un dégagement d'électricité dans la contraction musculaire. J'ai employé pour cela les instruments les plus délicats et tous les soins possibles, et je dois conclure que ce dégagement d'électricité ne peut pas se démontrer par l'expérience;*"† then, in 1846, in the French translation of the paper of the "Philosophical Transactions," published in the "Annales de Chimie et de Physique;"‡ again, in 1847, in a letter to M. Dumas, printed in the "Comptes-rendus":—"*On ne trouve aucune augmentation dans le courant musculaire pendant la contraction du muscle;*"§ and, once more in the same year, in his "Leçons sur les Phénomènes physiques des Corps vivants," p. 277:—"*Concluons donc que l'expérience directe répond négativement à la question que nous nous sommes addressée s'il y avait développement d'électricité pendant la contraction musculaire.*" Lastly, in 1849, after I had given a short account of my experiments on the negative variation as observed on the living body of man, Signor Matteucci, who in vain attempted to repeat these experiments, wrote in the "Comptes-rendus":—"*C'est avec le plus vif intérêt que j'ai répété cette expérience. J'avais espéré qu'elle me tirerait une fois pour toutes de l'incertitude dans laquelle je suis depuis la découverte de la contraction induite. Malgré un très-grand nombre d'expériences, dans lesquelles j'ai tâché de découvrir la vraie nature de la contraction induite, j'ai dû à la fin conclure qu'il m'était impossible de décider si la cause étoit un dégagement d'électricité par la contraction, ou un véritable cas d'induction nerveuse . . . J'ai donc bien démontré que, de l'expérience de M. du*

* "Philosophical Transactions for the year 1845." P. ii., p. 307.

† "Annales de Chimie et de Physique." Septembre 1845. 3e Série, t. xv., pp. 67, 68. The reader will remember that in that same letter Signor Matteucci gave the true interpretation of the passage of the "Essai," and ridiculed what he called my "hypothesis" of the negative variation.

‡ "Annales de Chimie et de Physique." Octobre 1846. 3e Série, t. xviii., p. 122.

§ 15 Mars 1847. t. xxiv., p. 415.

Bois-Reymond, il ne résulte pas la preuve du développement de l'électricité par la contraction musculaire."*

The failure, indeed, of all his endeavours to discover some electric phenomenon connected with muscular contraction, greatly contributed to make him reject the electric origin of the secondary contraction, and to mislead him into that strange hypothesis of a nervous induction, by which he has accounted for that phenomenon, and to which he still seems to adhere.

§ XI. Signor Matteucci's claims to the discovery of the negative variation of the muscular current during contraction examined.

Such, then, were Signor Matteucci's successive statements on the electric action of muscles during contraction. Setting aside the interpretation which he afterwards gave of the passage of the "Essai," he successively asserted—

1. That the muscular current disappears during the contraction.

2. That the muscular current increases during the contraction.

3. That the muscular current remains unchanged during the contraction.

By the two latter statements, Signor Matteucci most evidently would have destroyed his claim to the discovery of the negative variation to which the first statement might have entitled him, if he himself, in September, 1845, had not given quite another interpretation of that first statement.

As late as 1849, Signor Matteucci maintained that no electric phenomenon is exhibited by the muscles during contraction. In 1850, however, the Report of the Committee of the Académie des Sciences no longer allowed him to doubt the accuracy of my experiments, and the insufficiency of his own. He therefore since admitted the correctness of my statement concerning the negative variation, although he still denied the possibility of accounting by it for the secondary contraction.† And ulti-

* "Comptes-rendus," &c. 25 Juin 1849. t. xxviii., p. 783.

† "Comptes-rendus," &c. 3 Février 1851. t. xxxii., pp. 131, 132.

mately, Signor Matteucci, in his third attack upon me, ventured to claim the discovery of the negative variation on the ground of the above-mentioned passage of the "Essai."*

In my reply, I opposed him almost literally with the same arguments which I have used above. I convicted him, by literal quotation of his own words, of having since given quite another interpretation of the passage in question, and of having, during seven years after my publication of the negative variation, repeatedly stated, first, that the contraction is attended with an increase of the current, and afterwards, that no change whatever becomes perceptible in the electric condition of muscles during contraction.

Signor Matteucci, however, without mentioning one word regarding my reply, in his last pamphlet, once more brings forward the same assertion of priority. He says, at p. 4: "*Parmi mes expériences de cette époque* (*de* 1838 *à* 1842) *sur le courant propre, il y a celles qui prouvent évidemment les modifications souffertes par le courant de la grenouille sous les contractions tétaniques ;*" and, at p. 15: "*Lorsque, longtemps après la publication de ce Mémoire, j'appris par l'édition française du 'Manuel de Physiologie' de M. Müller, que ce Mémoire existait et que j'ai pu en lire une traduction, j'y ai trouvé, comme il m'arrive toujours, et à part les vues hypothétiques, qu'il avait vérifié mes expériences sur le courant musculaire ;* QU'AVEC LE GALVANOMÈTRE IL AVAIT OBSERVÉ UNE DIMINUTION DANS CE COURANT PENDANT LA CONTRACTION, CE QUI CONFIRMAIT UNE DE MES ANCIENNES EXPÉRIENCES," &c.

Thus, in September, 1845, Signor Matteucci pronounced my discovery of the negative variation a mere hypothesis, and declared that the passage of the "Essai" solely related to the state of the current AFTER the tetanus had relaxed. But, as soon as he could no longer question the truth of that discovery, he stated that he never took it for anything but a mere confirmation of his own statement in the "Essai," which he then asserts to refer to the state of the current DURING the tetanus. I shall abstain from drawing any conclusion from these facts.

But the following passage of the pamphlet yet calls for some

* "Comptes rendus," &c. 3 Juin 1850. t. xxx., p. 705.

remarks: "*Vous auriez été plus juste et plus exact en disant, que n'ayant pas un galvanomètre aussi sensible que M. du Bois-Reymond, je n'avais pu réussir, par des expériences tentées sur des piles musculaires en contraction, à confirmer le fait que j'avais vu précédemment en opérant sur la grenouille entière tétanisée.*" (p. 12.) Signor Matteucci, therefore, ascribes his inability to observe the negative variation to the want of a sufficiently sensitive galvanometer. In expressing that opinion, he no doubt forgot what he had written a few pages before: "*M. du Bois-Reymond a certainement rendu un service à la science, puisqu'il a réussi à donner au galvanomètre une si grande sensibilité : mais je le répète, un tel instrument n'était pas nécessaire pour étudier le courant musculaire et ses lois, car toutes mes expériences* ET TOUTES CELLES QU'ON PEUT FAIRE SUR LES MUSCLES, *réussissent très bien avec des galvanomètres ordinaires et avec des piles musculaires.*" (p. 6.)

But Signor Matteucci is altogether mistaken with respect to this point. With his galvanometer of 2500 coils of wire, I will undertake to show the negative variation most manifestly, provided, of course, I be allowed to use those methods of experiment which are so despised by Signor Matteucci. The galvanometer with which I first observed that phenomenon is that described in my first paper, being of 4560 coils of wire only. (See above, p. 11, Note 1.) On the other hand, Signor Matteucci might have at his command the most sensitive of those magnificent apparatus, recently made at Berlin, by M. Sauerwald, under my direction, and having from 25,000 to 30,000 coils of wire, and, unless he should consent to exchange his methods for mine, he will remain quite as unable as ever to repeat my experiments.

Be this as it may, Signor Matteucci, in the passage under consideration, desires to be excused, on account of the insensibility of his galvanometer, for not having repeated, with muscular piles, the observation which he says he made upon a single frog. But it is hard to conceive why a single frog should give a stronger current than a pile of twenty half thighs of frogs; and, if it did, Signor Matteucci was at perfect liberty to repeat the observation, related in the "Essai," upon a single frog, instead of using a muscular pile.

At all events, Signor Matteucci, in that passage, clearly ad-

mits what was sufficiently evident without that admission, from the whole of the above statements,—namely, that, since that observation to which, in September, 1845, he most unequivocally ascribed quite another import, he never saw anything like a negative variation of the muscular current on contraction. Yet he claims the discovery of that negative variation. So we have here an instance, unprecedented, I believe, in the history of science, of a claim being laid to the discovery of a phenomenon, which the claimant never saw, and, perhaps, never will see.

CONCLUSION.

There are many other points in Signor Matteucci's pamphlet which suggest similar remarks to me. But I am afraid the patience of my readers will hardly have been equal to the prolixity of the preceding detail. So I only beg to declare, that if I forbear from remonstrating against any other passage in that pamphlet, it is by no means because I submit to its meaning, or because I am unable to confute it with arguments quite as unanswerable as those adduced above.

Signor Matteucci, at the end of his pamphlet, promises that this attack upon me shall be the last one. Nobody, indeed, will be more glad of this determination than I; but to nobody, perhaps, will it be more profitable than to Signor Matteucci himself.

The new edition of his "Traité," which he is going to publish, will afford him an opportunity of at last turning to account the observations on his claims contained in my successive replies. As regards that new edition, however, I cannot refrain from expressing to Signor Matteucci some apprehension about his design to review the whole of my researches in it. (See above, p. 8.)

Signor Matteucci's own researches in animal electricity, as far as they have been successful, and setting aside his valuable papers on the torpedo, have hitherto only extended to some particular cases of the muscular current, the general law of which he still seems not to understand aright; to the influence which several circumstances, generally of a deleterious or injurious character, exert upon that current; and, lastly, to the secondary

contraction, which he discovered, but did not succeed in tracing to its true origin. Now a mere inspection of the table of contents of the "Abstract," or of the short conclusions enumerated in its last chapter, will suffice to show that I have enlarged our knowledge of this class of phenomena, far beyond the point at which Signor Matteucci arrived.

From the preceding pages it might appear, indeed, as if I had only worked at the same subjects as Signor Matteucci. But this is only because in his pamphlet he confined himself to an examination of those passages of the "Abstract" in which his name happened to occur (pp. 3, 14). Thus, of course, all those parts of that book escaped his censure, and consequently were passed over in silence, in which subjects utterly unknown to him are treated of, as in such parts there was no occasion to mention him.

These parts, however, form rather the greater part of the "Abstract." The statements thus far adduced in the present reply were mostly taken from my paper of January, 1843. Since that time, up to the present, I have incessantly and exclusively been employed in prosecuting these researches. Of the results which I have obtained I will only mention the following here.

To the law of the muscular current, as it was stated by me in my first paper, I soon added the law according to which different points also of the longitudinal or transverse section alone produce currents on being connected by the galvanometer wire (Abstract, p. 92); then the law, according to which the intensity of the muscular current varies with the position of the muscle on the ends of the galvanometer circuit, whether the distance of these ends remain unchanged or be allowed to vary also; again, the law according to which that intensity varies with the length and thickness of muscles (p. 115). The same laws I proved to apply to the nerves (pp. 164, 172).

I examined more closely into the electric action of the natural transverse section of muscles, and succeeded in explaining all its apparent anomalies by pointing out what I have called the parelectronomic layer of the muscle, that is, a layer of muscular substance situated beneath the expansion of the tendon, the electromotive power of which more or less counteracts that of the rest of the muscle (p. 116). I showed that the electromotive power of contractile tissues

varies in the same ratio as their mechanical energy (p. 161). I determined the electric action of the skin of the frog and of man,* and by the results of this investigation I succeeded in demonstrating the muscular current during the state of rest of the muscles, even on the live undissected body, at least in frogs (p. 125). As to the electric condition of muscles during contraction, my principal results are already mentioned above, at page 28. I traced the nervous current through the whole nervous system, and through the whole series of animals (Abstract, p. 169). The electromotive power of ganglia I demonstrated on the abdominal ganglia of the crab. I pointed out those highly remarkable modifications of the electric deportment of nerves when under the influence of an electric current, which I designated by the name of the electrotonic state of nerves (p. 174). The negative variation, which I showed that the nervous current underwent on tetanising the nerves, has been also alluded to already, at page 28. Indeed, I succeeded in 1847, after four years' toil, in transmuting into a slight motion of the magnetic needle, those internal motions of the nerves which, on reaching the muscles, would have caused convulsions, on reaching the brain, would have aroused sensation (p. 187—193). Since the middle of the last century, some result of this kind had been longed for by physiologists, and consequently aimed at in a great many fruitless experiments.

I omit several other points of no less importance here, because their examination does not (or at least not so indispensably as that of the points just alluded to) require those peculiar means of investigation devised by me twelve years ago, and since fully detailed in my German work. The above stated results, for the most part, can only be obtained by means of a galvanometer of high sensitiveness, of the conducting vessels with their cushions or some similar contrivance, and of that host of little auxiliary apparatus well known to those who ever watched my experiments. They cannot be obtained, in short, by means of an ordinary galvanometer, and of piles of muscles or nerves, for the very simple reason that the conditions necessary in order to obtain them generally are so delicate, that they often are hardly

* "Monatsberichte der Berliner Akademie, März 1852," S. 111.

to be fulfilled, even on a single muscle or nerve, and that, accordingly, it would be impossible to do so on twenty muscles or nerves at once. Thus, it is perfectly obvious to any one acquainted with this subject, that Signor Matteucci's repeated attempts to discover a variation of the muscular current during contraction (without mentioning several other circumstances) could not but fail, because of the impossibility under which he laboured of making the ten frogs which composed his pile contract at the same time.

Signor Matteucci, however, in his last pamphlet, asserts, that his method of experimenting on twenty half thighs of frogs is the only commendable one:—"*Je soutiens, ce qui d'ailleurs est bien évident pour tous ceux qui n'ont pas d'intérêt contraire, que la méthode des piles musculaires est la seule vraie. . . . . Personne au monde qui pourrait examiner de ses propres yeux la forme et les qualités d'un objet de dimensions assez grandes, se donnerait la peine de le réduire très petit pour l'observer avec une loupe*" (p. 6). Signor Matteucci's comparison is very good indeed, but it is wrongly applied, for there are many things here, and very important too, which, to continue his simile, cannot be seen with the naked eye, but require the use of a most powerful magnifying apparatus in order to be perceived. Yet Signor Matteucci condemns and rejects my methods altogether, by which alone for the present such means of magnifying are afforded, and he does not in the least seem inclined to employ them for repeating my experiments.

Signor Matteucci, therefore, is an utter stranger to all those results, and he will necessarily remain so. In consequence, I am sorry to say, I am compelled TO QUESTION HIS COMPETENCE AS A JUDGE OF THE ACCURACY OF MY STATEMENTS; and I protest beforehand against the results of the discussion to which he says he will submit my experiments and theory in the new edition of his "Traité," unless, in the course of the trial, he furnishes sufficient evidence that he has followed minutely the directions given by me in my German work for repeating my observations.

THE END.

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS STREET,
COVENT GARDEN.

ON THE PATHOLOGY OF RHEUMATISM.

By Francis T. Bond, B.A., M.B. (Lond.), Medical Tutor, Queen's College, Birmingham.

The statement with which Dr. Copland prefaces his very able and comprehensive article on the subject in the *Dictionary of Medicine*, that "although rheumatism is, owing to geographical and social circumstances, one of the most prevalent diseases in the British Isles, yet it is one respecting the nature and treatment of which exists the greatest diversity of opinion and the least amount of undisputed knowledge," may be regarded as no exaggerated description of the state of medical science with regard to this frequent and important affection. Whether this state of obscurity be owing to the natural intricacy of the subject, and of the many collateral questions which must be answered before much hope can be entertained of solving the original problem, or to the various theories which have at different times, and in accordance with the dominant doctrines of the day, been invoked with the intention of throwing light upon it, but have served rather as *ignes fatui* to lead astray from the true path of inductive generalization, it is difficult to say: certain it is that to any one who peruses even the standard monographs upon the subject, putting out of question the articles upon it in systematic works, and the numberless brochures which are to be found upon the shelves of any large library, its pathology and the principles of its treatment must appear to be in a chaotic and most unsatisfactory condition. Perhaps there is no single disease with which the practitioner is brought so frequently in contact, and yet in whose alleviation he acts more empirically or with greater doubt than in acute rheumatism; nor is there any better indication of the distance that must be traversed before we can hope to arrive at the goal of its true and rational pathology, than an enumeration of the various medicaments which have successively been suggested for its cure, or which are still employed, though often the

inspiration of different and conflicting theories. Such a condition of the therapeia of any disease will probably depend upon one of two causes: either the disease itself must be of a Protean character and must require much careful investigation to establish the relationship of its varieties to one another, and to adapt to them a fitting curative treatment; or the remedies which have been used with alleged, though often with most questionable success, have not been directed so much against the disease itself as a whole, but rather to the subdual of its symptomatic and subsidiary phænomena, which upon this supposition must be of a numerous and complicated nature. In the case of rheumatism, which has been, as it were, the battle-field of a host of specifics and nostrums, the conviction forces itself upon the observant mind that the amount of relief which does in some cases unquestionably attend their employment, affects for the most part only the minor concomitants of the affection, and has little or no influence upon the progress or duration of the affection itself. The greatest triumph of science is not to find out that a certain cause produces a certain effect, but to discover why it does so: and although for philanthropic reasons the medical practitioner may be only too well satisfied that his practice, though empirical, is attended with a certain proportion of success, yet it will be ever his highest aim to investigate the path by which that success has been attained; and thus to eliminate, by a cultivation of the conditions under which alone it is attainable, all the elements of doubt and obscurity which perplex the course of the man who is influenced by the blind dictates of experience only. But to establish the connexion between the means thus employed and the results to be obtained, it is necessary that he have an exact and accurate knowledge of all the phænomena upon which he is to base his treatment, that he do not overlook any important ones, and above all that he know how to subordinate the less to the more essential, uninfluenced by the sometimes more apparent severity of the former, and the less obtrusive significance of the latter. An analysis of the nature of an attack of acute rheumatism, of its accompaniments and consequences, will show that it is upon this latter rock that the founders of the modern pathology of that disease have struck; that they have given a primary place to what is only a secondary phænomenon, and that they have ignored the most constant and essential feature of the disease. So long as investigations are pushed in this direction, and this most imperfect, and to a certain degree false, view of the affection is employed as a basis for working out its pathology and for bringing it into correlation with other affections, so long must all hope of explaining the import of the many phases which it exhibits, and above all of establishing a rational prophylaxis against its attacks, be abandoned. It is with the view of pointing out what the author considers to be the fundamental feature of the disease, and calling attention to some of its collateral peculiarities

which seem to be generally overlooked, that the present paper has been written. And though he cannot but feel that there is no little temerity in thus stepping forward where so many better men have stood before him but to fail, yet his consolation is, that although this individual attempt may not realize that success which in the cause of science he would desire, it may lead others to proceed in the path thus indicated until their investigations are crowned with happier results.

It will be well, before proceeding on an independent line of research, to examine briefly one or two of the more plausible theories which have been propounded to explain the phænomena of rheumatism, and which have seemed to meet with most support from clinical experience or scientific research. The commonest, and at the same time one of the oldest, refers the disease to the combined influence of cold and moisture upon the body, without indicating any constitutional condition as a prerequisite to its development.* That cold alone is not sufficient to produce the disease is shown by the fact, that the results which that agent produces on a previously healthy body are of a totally different kind to those of rheumatism. We hear hardly anything of the disease—at any rate, in its acute form—amongst sailors, who have been exposed in whaling voyages and exploring expeditions to the severest cold of the Arctic regions. And even in the marine, of the royal and merchant services, as a class, we find much less of it than might have been expected, considering the exposed nature of its occupation to all the vicissitudes of temperature and moisture. In fact, it might almost be affirmed that *habitual* exposure to a low temperature rather counter-indicates than predisposes to acute rheumatism—the influence of cold, whenever the attack can be traced to its effects *alone*, being most powerful on those who, by their habits and occupation, are generally most protected from it. Nor does the additional influence of moisture much facilitate the explanation; for, of the thousands who are daily exposed to both cold and wet in these northern regions, but a very small proportion incurs the disease; whilst a very large number of those who do, have not been inordinately exposed to either.† Moreover, if cold were even its most frequent exciting cause, we ought to find the disease predominating in the coldest months of the year, and the coldest regions of the earth: whereas, M. Chomel states, that the months of April and May are those in which the largest number of rheumatic patients are admitted into the Parisian hospitals—a statement which will probably be borne out by English experience. The statistics of the army also, as quoted by Dr. Fuller, prove that for every 1000 men, fifty-seven cases of rheumatism

* Watson: vol. ii, p. 676; third edition. Bouillaud: *Recherches nouvelles sur le Rhumatisme*; pp. 88, 91.

† For some good remarks on the questionable nature of the connexion between cold and many inflammatory diseases, vide *Jones and Sieveking's Path. Anat.*; p. 114: also *Henle's Handbuch der rationellen Pathologie*; b. ii, s. 234.

occur in the mild and genial climate of the Cape of Good Hope, fifty in England, and only thirty in the cold and variable climate of Nova Scotia. And, for my own part, I have seen more and severer cases of acute rheumatism in six weeks in St. Mary's Hospital, situated as it is in one of the warmest and most sheltered of the London districts, than I have witnessed in as many months in the Royal Infirmary of Edinburgh, with nearly three times the number of beds. It cannot be doubted, however, that many cases of rheumatism do appear to recognize a chill as their principal exciting cause; but it is probable that, if many of those in which this element, when discovered, is considered sufficient to account for the origin of the attack were more carefully examined, it would be found that a more potent influence still has been at work—that of fatigue; and any one who will carefully analyse a large number of cases of rheumatism will, I think, unfailingly come to the conclusion, that corporeal exhaustion is by far the most frequent exciting cause of the disease.

Another theory, which carries the etiology of rheumatism a step further back, by indicating the existence of a previous morbid condition of the blood, and has met with a ready reception on account of its support by men of deservedly great authority in the profession, refers the disease to a suppression of the elements of the cutaneous excretion—more especially of lactic acid, which, thus accumulating in the system, gives rise to the various phænomena of the affection. Unfortunately, there are two or three weak points in this otherwise very promising theory, which owes its existence, I believe, primarily to Dr. Prout, but has been subsequently adopted by Drs. Todd, Williams, Fuller, and most recent writers: 1stly. It has not been shown that any excess of lactic acid has ever been detected in the blood of rheumatic patients: indeed, the very question of the possibility of its detection at all in that fluid is so debatable, that it must be considered at present in too problematical a condition to render it a trustworthy basis upon which to erect any hypothesis.* On this point Dr. Garrod—a certainly very competent authority—says: "With respect to the presence of lactic acid in rheumatic blood, although we by no means wish to deny its occurrence, yet, we should hesitate to receive it as a fact until further evidence is produced."† 2ndly. Even if it were present in any excess, it is somewhat difficult to see any connexion between its presence and the phænomena of the disease. Why lactic acid in the blood should give rise to exudations in and about the different fibrous structures of the body does not seem at

* "The same may be said of the idea propounded in recent times by Williams and Fuller, which refers the cause of rheumatism to an extreme production of lactic acid in the organism. No one has either demonstrated this excess (the few cases in which the blood has been found to be acid prove nothing on this point), nor has it been experimentally shown, that the accumulation of lactic acid in men or animals produces such phænomena as we observe in acute rheumatism."—Vogel in *Virchow's Handbuch der speciellen Pathologie und Therapie*; b. i, s. 482.

† *British and Foreign Medico-Chirurgical Review*; 1854; p. 384.

all evident, nor has any one attempted, experimentally, to show that it can do so.* Were these tissues the natural emunctories of the acid, its excess might be supposed, under certain circumstances, to produce a hyperæmic and inflammatory condition of them; but the fact that its elimination takes place by the kidneys, alimentary canal, and skin, and that its principal seat of formation in the economy is amongst the muscular tissues, renders this hypothesis most gratuitous and groundless. 3rdly. There is an excess of other acids in the excretions, and, therefore, possibly in the blood, *e. g.*, uric, acetic, oxalic, &c., of any one of which the same effects might have been as reasonably predicated as have been assumed of lactic acid. 4thly. It admits of much doubt whether the disease is ever preceded by a greater suppression of the cutaneous excretion than the prodromata of all inflammatory diseases bring with them, as is asserted by the advocates of this hypothesis, or whether "rheumatic patients usually labour under a harsh and inactive condition of the skin during the intervals between their attacks," as they have been said to do. It is certain that in the chronic form of the disease this is far from being the case; and Dr. Graves particularly points out, in his *Clinical Lectures*, the constant and copious diaphoresis which often attends the subjects of chronic rheumatism. 5thly. The extreme tendency to sweating which occurs during an acute attack of the disease, and is cited as the effort of nature to get rid of the excess of lactic acid, may be explained in a much more satisfactory manner, as will be hereafter shown. Such are some of the principal objections which may be urged against the present popular theory of rheumatism, and which, however imperfect and unsatisfactory it may be, has at least this merit, that it indicates a tendency to the investigation of the chemical and physiological features of the disease, as something which must be studied in addition to the strictly clinical ones—a process which, if pursued but a step further than it has yet been carried, will lead to a correct appreciation of its pathology. And this theory is also a good illustration of the tendency of modern medicine in general towards a rational humoralism; to refer to a disordered state of the blood either in the quantity or quality of one or more of its constituents, diseases which had been previously regarded and treated only as so many local irregularities.

Now, before entering into a discussion upon the nature of rheumatism, the first question to determine is, In what does a typical acute attack of the disease consist, and what are its most essential features? They may be summed up as follows: an individual, previously perhaps in *apparent* good health, is seized with symptoms of pyrexia, generally after severe

* Dr. Richardson's experiments, though ingenious, cannot be held to have established this point; and any conclusions from them are worthless until it can be shown that other organic acids do not produce the same result.

fatigue, exposure to cold, mental emotions,* or some other depressing agency. The general disturbance increases, and at last culminates in a local affection of the larger joints, of the heart, or more rarely of some other internal organ; the local affection taking on the form of heat, pain, and swelling of the joints, or of effusion into the pericardium with or without valvular affection of the heart. The joint swelling and the pericardiac effusion consist of an exudation of a more or less fibrinous nature, deposited in the former case amongst the tissues surrounding the articulation, or in the synovial capsule itself.† This goes through the usual phases of exudations in general, its elements either breaking down after a time and becoming resorbed into the circulating current, or taking on a low kind of vitality and assimilating themselves to the tissues with which they are contiguous. As the process of resorption proceeds, the excretions are greatly augmented, evidently with the partial object, at least, of carrying out from the system the results of this local disintegrating process, probably also of a more general disintegrating process which is going on throughout the whole vascular system, concerning which more will be said hereafter. The perspiration is acid and copious, the urine is loaded with a dense sediment, and a critical diarrhœa is by no means uncommon. With the subsidence of the local effusion, and the general pyrexia, the disease disappears, leaving behind it nothing but a state of great prostration of the vital powers and anæmia, together with a certain amount of stiffness in the joints, dependent in some cases upon mere disuse, in others upon the presence of organized new growths —leaving behind it also, not infrequently, disease of the heart resulting from the same condition. The patient now enters upon the convalescent stage, but is generally long before he recovers his natural health and strength; and he is extremely liable to relapses, especially from bodily exhaustion, or too early a return to an animalized form of diet. The most noteworthy feature in his subsequent history is, that his future liability to the disease is for the most part vastly increased by the first attack; in fact, the liability increases in a direct ratio with the frequency, and to a certain extent with the protracted duration of the attacks—a sure mark of the diathetic character of the disease. These are the chief points which reveal themselves to the clinical observer; the researches of the chemist, however, add another and most important item to our knowledge. It is a fact, most thoroughly established by the researches of Andral, Becquerel, Simon, and other physiological chemists, that the fibrine in the blood of patients suffering from an

* M. Gubler gives three cases of rheumatic cerebral affection, in two of which it had been preceded by powerful depressing moral agencies.—*Archives Gén. de Méd.*; Mars, 1857.
M. Vigla also refers to the predisposing influence of moral causes in developing cerebral rheumatism.

† For descriptions of the state of the joints exhibited in several autopsies of acute rheumatism, see Cossy: *Archiv. Gén. de Méd.*; vol. xxxiii, p. 286.

attack of acute rheumatism, exhibits a marked elevation above the normal ratio, higher as a rule than in any other phlegmasial disease—even pneumonia rarely attaining so high a figure. This hyperinotic condition of the blood exists from the very first onset of the affection, and, according to Dr. Parkes, before any local symptom makes its appearance;* and it bears a definite relation to the general inflammatory disorder present in the first stages, as well as to the amount of excreta in the later ones. It is also accompanied by a most marked diminution in the number of blood globules and in the proportion of albumen. Taking the normal ratio of fibrine in healthy blood as about four parts in 1000, Andral has found that of acute rheumatism to range as high as ten, whilst other observers even give thirteen as the maximum: that of the worst cases of pneumonia rarely exceeding nine parts in 1000. So of the blood globules: taking 135 as their normal standard, they fall in phlegmasia generally to a mean of 123, in acute rheumatism to 118, and in pneumonia occasionally as low as 113 parts in 1000.† This altered relation of the fibrine and blood globules is the most important blood phænomenon in acute rheumatism; others have been recorded, but they are neither of sufficient importance, nor sufficiently well authenticated, to render a notice of them necessary here.

Now, on taking a broad view of these concomitants of the attack, what are those which would appear to be the most characteristic of the disease—to be the most typical, either by their constancy or their physiological importance—to be those from a study of which, in comparison with similar features in other diseases, fruitful results may be most rationally expected?

For any one who carefully considers this question, I think that there can be but one answer, viz., that the most important features of the disease are the exudation and the hyperinosis, most important because most constant, and because they are of weighty pathological import, as indicating considerable local and constitutional disturbance. The pain, heat, and swelling of the affected joints are merely local symptoms of the exudation, and depend for their intensity entirely upon its amount. The increased excretions, especially the perspiration upon which so much stress has been laid, are entirely of secondary importance, and are physiologically referable either to the breaking down of the exudation and the elimination of its elements in a soluble state from the system, or to a metamorphosis of the abnormal amount of fibrinous matter in the blood, lactic and uric acids and urea being only different stages in the transformation of the fibrine group of compounds from their place of highly vital tissue-elements to the inassimilable and inorganic condition which is necessary for their excretion when no longer subservient to the

* Gulstonian Lectures on Pyrexia.—*Med. Times and Gazette*; 1855: lect. ii.
† Becquerel and Rodier: *Pathological Chemistry*; Speer's translation, p. 99.

uses of the organism. It may, however, be alleged that the febrile symptoms are more important in a pathological point of view than the exudation or the hyperinosis, as they indicate a general state of constitutional disturbance, and are always proportionate to the severity of the disease. This idea derives some small support from the term rheumatic fever, which is so often used as a synonym for acute rheumatism, and from a corresponding notion which has grown out of it, and has led some nosologists to place the disease in the list of specific fevers, and has entirely lost sight of the local affection. Now to class rheumatism with the special exanthematic fevers, and to indicate thereby that they have anything more in common with one another than increased bodily temperature, rapid pulse, and altered relation of the nutritive and destructive powers of the system, is simply a confusion of ideas founded upon a misconception of terms. The facts, that the latter are for the most part epidemic and contagious, the former never; that the latter by one attack give a greater or less immunity from succeeding ones, whereas the case is just reversed in rheumatism; that this latter affection is invariably accompanied by a hyperinotic condition of the blood, whilst in the special fevers the fibrine is below its normal standard; and that the local affection in rheumatism is nearly always present, and comes on in the very commencement of the disease, while it rarely appears till late in the course of the exanthemata, and is often enough absent altogether, are, independently of other weighty considerations, distinctions of sufficient importance to sever completely the one from the other. The importance, however, of the febrile symptoms of rheumatism cannot be over-estimated, either in a pathological or a therapeutic point of view; but then it must be remembered that they are only *symptoms* after all—the outward manifestations of an internal derangement of the economy, and not the cause of the derangement itself. To address ourselves to them, therefore, in the hope of finding a solution of our pathological difficulties, would be about as reasonable as to seek for the cause of tubercular phthisis in the racking cough, the profuse night sweats, or the intense diarrhœa which characterize that affection.

Having thus endeavoured to establish the prior importance of the hyperinosis and the exudation over all the other phænomena of rheumatism, the question which now presents itself for consideration is, What is the relation which these two features bear to one another? Is the hyperinosis merely an effect of the reaction of the local disease upon the system at large, as is generally believed, or is it the primary source of the exudation, the causative agent of the latter, without which it could never exist? I believe this last to be unquestionably the true statement of the case, and shall endeavour to bring satisfactory evidence to show that it is so. Considered simply in a teleological point of view, the former supposition throws no light whatever upon either the economical

object of the hyperinosis, or upon the mode of its connexion with the local affection; whereas, on the latter hypothesis, it is highly probable, *à priori*, that an excess of fibrinous plasma rapidly accumulating in the blood to an amount which is incompatible with perfect health, should be attracted, as it were, out of the vascular system, in the shape of an exudation to those tissues for which it has a physiological affinity—such a process, in fact, being no more than a mode of excretion. In urging this consideration it must not be forgotten, as is well shown by Andral in his researches on the blood of patients who have suffered from burns, that *traumatic* lesions do most undoubtedly *give rise* to a hyperinosis, apparently as a result of their reflex upon the constitution at large, or upon the vitality of the blood in particular; but we are now dealing with the hyperinosis of *idiopathic* lesions which are clearly not amenable to the explanation that may be given in the former case: for though it is only reasonable that the economy should, on the reception of injury to any portion of it, prepare immediately an unusually large stock of that material which is specially devoted to its repair, to apply such an explanation to idiopathic lesions, and say that nature first inflicts injuries on herself and then makes preparations for repairing them, is surely most gratuitous and unnecessary. The most powerful argument, however, in favour of the sequential relationship of the exudation to the hyperinosis in acute rheumatism is derived from a study of the phænomena of the disease, and from a tentative application of the hypothesis to them with a view of ascertaining how far the one is a correct explanation of the other. Thus: a man is seized with the usual prodromata of what turns out to be afterwards acute rheumatism. At this period of the disease, on the hypothesis above suggested, the fibrine of the blood becomes rapidly augmented, from some cause or other to be hereafter discussed, and from being thus elevated above what should be its normal proportion in the blood, its vitality as a blood constituent is lowered, and it exhibits a greater tendency than is natural to it to leave the circulating current and pass into the sphere of the local attraction of the tissues with which it is homologous. At this juncture let us pause to inquire if we have any data for determining in what part of the body this exudation is most likely to occur. In this inquiry we shall be much assisted by bearing in mind that great law of nutrition, in accordance with which each tissue attracts from the blood, by a species of elective affinity, the elements of that fluid out of which it is built up; the muscles attracting the elements of muscular tissue, the different fibrous structures their constituents, and so on throughout the whole economy. But if each tissue attracts its elements from the blood, it is but reasonable to infer that there is a correlative affinity on the part of the blood elements each for its own tissue, so that the attraction is mutual, and may be represented as a constant tendency to union between the two, the mersion of

the mere tendency into actual union being brought about by local or particular causes—on the part of the tissues by their constant wear and decay, and consequent necessity for repair; on the part of the blood by the, fluctuating preponderance at different times of one or more of its constituents: hence we have in the body, as in the great world of physics, a constant attraction of molecular particles for given centres, and at the same a certain bond of union between the particles themselves which can only be destroyed by a preponderance over it of the centrical attraction. Now if we have an excess of the fibrinous element in the blood at the onset of an attack of acute rheumatism, as is supposed, the *direction* of its localization when passing out of the vascular system will be evidently determined in favour of those tissues with which it has the closest homological affinities—affinities which, in most cases, as is shown by the results, lie with certain forms of fibrous tissue. Moreover, its local deposit will be influenced by the physical or physiological relations of each fibrous tissue; by the comparative proximity which it may exhibit to the special type of the fibrine exuding (adopting the supposition of Lehmann, that there are several allotropic varieties of fibrine, and consequently of fibrous tissue); by any special peculiarity which might confer upon one or more of them a claim, as it were, to the attraction of the fibrine when on the point of exuding; and by the local stasis of the circulating fluid, from any cause whatever, in or about any one of them.

Firstly: *with regard to the mechanical relations of the part.* We should expect a large exudation to occur most readily into those structures which exhibit the greatest facilities for its accommodation, *e. g.*, synovial and serous sacs, intermuscular and intercellular spaces, &c. Secondly: *the especial type of the fibrinous plasma.* In the acute form of the disease it would seem to assimilate itself to the fibrous tissues of the joints and heart; the fibrous tissue of the lung, as in the rheumatic form of pneumonia which M. Trousseau asserts that he has occasionally witnessed; or the subcutaneous areolar tissue, as in the rheumatic erythema nodosum described by Dr. Begbie, and MM. Hardi and Behier. In the chronic phase of the dyscrasia it directs its attacks upon the fibrous investments of the muscular and neural tissues, causing muscular rheumatism, thickened fasciæ, neuralgic pains, &c.; and occasionally upon the fibrous matrix of the liver, kidney, or other organs; of which I shall have more to say bye and bye. Thirdly: an important element amongst the determining causes would be either any natural abnormality of a fibrous tissue, laying it open to the attack of morbid influences—just as morbid growths, or scars, are often the first to feel the effects of prejudicial influences acting on the economy in general—or its temporary weakness from some accidental cause: in illustration of which latter circumstance we have the fact that one of the most frequent, probably by far the most frequent, of

the determining causes of a rheumatic attack of any particular set of joints is over fatigue of those joints.* For example, the occurrence of rheumatism in the knee and ankle joints after a long walk is sufficiently notorious; as it is also the tendency of the affection to the upper extremities of those who use them much, *e. g.*, blacksmiths, &c. The occasional tendency of the rheumatic dyscrasia to vent itself in an attack upon the knee joints of domestic servants after kneeling for some time to wash stairs, &c., which I have more than once noted, is an illustration of the same circumstance; as also is probably the tendency of patients with a nervous irritable heart to take on cardiac rheumatism. The affection of particular sets of muscles, as those of the back after violent exercise of them in rowing, cricketing, &c., is attributable to the same cause; as also may be the more problematical forms of rheumatism of the gravid uterus, of the pharynx, and of some other organs, described by French authors. The influence of mental excitement as a determining cause of rheumatism has been before alluded to, but much remains to be investigated on this branch of the inquiry. So also we may explain the effects of draughts of cold air on a particular joint giving rise to a subsequent attack of rheumatism in that joint, as is noticed by Mr. Paget: "The depressed nutrition of the joint makes it more liable than any other part to be the seat of inflammation excited by the diseased blood."† Thus there will be four sets of causes, at least, which will determine the point at which the exudation may localize itself, each complex in itself, and each modifying and modified by the others.

It is unnecessary to trace the course of the affection in one of its acute attacks any further, as its subsequent stages have been already briefly alluded to, and are perfectly similar to those of other inflammations. There is one phænomenon, however, attending its resolution, which deserves a passing notice, and that is the increase of the urinary and cutaneous excretions. In the former, urea and uric acid exist in large excess, whilst free acetic, phosphoric, lactic, and oxalic acids have been detected by Vanquelin, Henry, Dr. Bence Jones, and others. The most predominant constituent of the perspiration is lactic acid, but uric (Stark) and acetic (Prout) acids have also been observed in it. Most, if not all, of these substances are products of the degradation of fibrinous matter: the relations of urea and uric acid to highly nitrogenized matters, as exhibited by the experiments of Lehmann—by the recent manufacture of urea by artificially oxydizing albuminous substances by M. Béchamp‡—and by the general excess of these excreta in hyperinotic states of the blood, combined with that of lactic acid, to the muscular juice as determined by the researches of Liebig, amply corroborate this statement as

* Williams: *Principles of Medicine*; second edit., p. 151. This influence of fatigue is also noticed by Dr. Fuller, MM. Hardi and Behier, Gubler, and other authors.

† *Surgical Pathology*; vol. i, p. 462.

‡ *Journal de Chimie et Pharmacie*; Janvier, 1857.

far as these three bodies are concerned—the others, from the smallness of their amount, may be put out of consideration. The preponderance of either urea, uric, or lactic acid in the excretions may depend upon peculiarities of the fibrine itself and its tendency to be metamorphosed into one of these substances rather than into another, or to pass off by one excreting organ rather than another; but it is probably also referable in part to the vital power of the constitution and its ability to oxydize the fibrinous matter to one or other of these stages in its metamorphosis: for the atomic composition of these three substances and their chemical history gives ground for conjecturing that they are only successive grades in the resolution of highly complex albuminoid and fibrinoid bodies into much simpler forms; and it may be assumed, that the power of the system to resolve the whole of the hyperinotic matter in the blood into urea, or its inability to proceed farther in respect to a portion of it than uric and lactic acids, depends upon certain vital peculiarities upon which we can do little else than speculate at present. A careful examination of the ratio of the urinary and perspiratory excretions to one another, and of their constituents, in a series of rheumatic cases, would do much to advance our knowledge on this point, but I have been unable to find any record of such an attempt.*

There are two rather singular facts connected with the pathology of rheumatism which the present seems a fitting place for noticing. The first is, I believe, pretty generally recognized as a clinical fact, though it is seldom or never referred to in books: I mean the great rarity of the combination of rheumatism and phthisis in the same individual. My own experience only supplies me with two instances of this concurrence; and in both of these the rheumatic affection was of a badly defined character; and, judging from that of the oral and written authorities whom I have been able to consult upon the subject, the coincidence is far from common. The only allusion which I can find to it is in Professor Bennett's *Treatise on the Oleum Jecoris Aselli*, where he quotes Brefeld, in illustration of the strong analogy which exists between the gouty, scrofulous, and rheumatic diatheses. That author points out, for instance, that gouty patients frequently produce scrofulous children; and that individuals who in their youth were scrofulous—and especially rachitic—exhibit at later periods of life great tendency to rheumatism. "It has been often observed," he says, "in families of the lower orders, that while the children are scrofulous, the parents suffer from obstinate rheumatism."† And this brings me to the consideration of the second fact, viz., the hereditary connexion of rheumatism and phthisis. I have carefully interrogated a considerable number of patients, who have

* The few, but admirably reported, cases of rheumatic fever given by Dr. Parkes in his Guldonian Lectures *(Sup. Cit.)*, should be excepted from this statement.

† On the *Oleum Jecoris Aselli*; p. 49.

suffered from acute rheumatism, with regard to their family history, and have found, in a large per-centage of cases—especially in those in whom the dyscrasia seemed to have most firmly established itself—that strong evidence was obtainable of a marked family phthisical taint, to such an extent in some cases, that both parents, or two or three brothers and sisters, would have been carried off by the disease; whilst the patients themselves were at the time quite free from any signs of local or constitutional tuberculization. It may be thought that, on looking at the frequency of phthisis in families generally, such cases were only instances of coincidence, and do not warrant any positive conclusion: nor do I wish to place undue stress upon them, for that reason; but the history was generally so clear, and the fatality so marked in most of the cases examined, that I feel, for my own part, little doubt of the truth of the fact, even if it were not a probable one on *à priori* grounds, as well as in accordance with the above observations of Brefeld. From these two circumstances, therefore, I think that we are justified in drawing this conclusion—however paradoxical it may at first sight appear—viz., that there is a strong antithesis, and at the same time a strong connexion between rheumatism and phthisis: a strong antithesis, inasmuch as they rarely occur together in the same individual; and a strong connexion, in so far as their dyscrasiæ seem linked together by hereditary ties. How is this to be explained? I think that it may perhaps be explained in the following manner: Looking at the so-called dyscrasiæ as a group, we observe that one of their most distinguishing characteristics is chronicity. When once they establish themselves in the constitution it is a very difficult thing to get rid of them; and they impress their mark upon the whole system, stamping all its processes continually with the proof of their presence, and giving them a typical peculiarity, as distinct probably, in the case of each dyscrasia from all the rest, as it is from that of normal health. Moreover, though all these separate types appear in a classification to graduate into one another, yet each seems for the most part to possess a distinct and independent identity, and exhibits but little tendency to pass into another type, except under powerful and long continued influences. Such influences, however, are rarely brought to bear upon the individual, unless in the course of a long life, or under peculiarly favourable circumstances: thus, we rarely get examples of cancer and tuberculosis occurring together in their typical forms in the same patient in a state of activity, though we may find the one progressing where the other has been for a long while passsive; and, as shown by Rokitansky, may also find a combined dyscrasia not particularly typical of either. Nor do we often get phthisis and rheumatism occurring concomitantly; and there is very good reason for doubting whether we ever get the true sthenic pneumonia in a patient in whom the tubercular taint is well established, although an intermediate form

of affection is far from rare. The infrequency of pericarditis in phthisical patients, except from tubercle of the pericardium, is also an illustration in point. And the reason for all this seems to be that the dyscrasial taint, whatever it may be, has so moulded the economy in one way, and given it such tendencies to diseases of one type, that it is unable, except under rare stimulus, to take on diseases of another type: the momentum of its progress is, so to speak, too great to be overcome by any but influences of the most potent description. But though the transition of one type of dyscrasia to another be rare in the individual, it appears very probable from what we know of hereditary influences, that it may occur in the transmission of acquired peculiarities from parent to child, and that the inherited dyscrasia of the latter may be elevated or degraded in the scale in proportion as that of one or both parents is more or less marked.* I have not yet been able to determine whether the hereditary relationship of rheumatism and phthisis ever exhibits the phænomenon of atavism, though I suspect that it sometimes does. Of course, it must be perfectly clear that, for the present at least, this explanation of the facts in question is, and necessarily must remain, a purely hypothetical one; yet, it rests for its support upon a principle whose existence is as certain as that of any in nature, viz., that as there are different types of bodily organization—all referable to a primitive archetype—and as there are different types of mental character equally derivable from a psychological archetype, so there are different types of bodily constitution, characterized by definite pathological tendencies, exhibiting a marked influence over the discharge of all the animal functions and the development of all diseases, and bearing a definite relation to the physiological archetype of perfect health. This principle is not so generally recognized by medical observers as it ought to be; for it is one of primary importance in estimating any deviation from the strict line of physiological activity, and lies at the root of all rational pathology.

From the foregoing considerations it follows that rheumatism, like all other local idiopathic inflammations, recognizes in its etiology two distinct elements: a constitutional one in the altered relations of the fibrinous matters of the blood, both as to quality and quantity; and a local one in the general affinity which exists between the fibrous tissues and the hyperinotic plasma, just as there does between the adipose tissue and the fatty matters of the blood when the latter are in excess. It cannot be too strongly insisted on, that this tendency of the hyperinotic matter to the tissues with which it is homologous, is strictly a physiological process of temporary excretion, by which it is removed for a time from the circulating system, when existing in such an amount as is incompatible with the healthy performance of the functions of that fluid. The idea

* See the very interesting chapter on "Hereditary diseases," in Sir H. Holland's *Medical Notes and Reflections.*

of Treviranus, so admirably developed by Mr. Paget, that each individual organ and tissue stands to the blood in the relation of an excreting centre, in virtue of the power by which it extracts from that fluid the elements which are necessary for its own nutrition, but which, if retained in the blood, would be fatal to the nutrition of the rest of the body, no where finds a better illustration than in the case of rheumatism. Here we have a sudden increase in the fibrinous matters of the blood by which a state of things is brought about which is in every way incompatible with a healthy condition of that fluid. To obviate this, a portion of the fibrinous plasma is removed out of the circulating current by an exaltation of the affinity of those tissues which exercise a natural attraction over it. By this means the blood is relieved of a portion of the burden under which it laboured, and is enabled to eliminate the excess of fibrinous matter which remains behind by a proportionable increase in the vigour of its excreting processes, whilst the extra vascular exudation at the same time passes through its metamorphosis so slowly, and reverts to the blood so gradually, as not to embarrass greatly any of the excreting functions; whereas, had the whole amount of fibrine exuded disintegrated in the blood, with a rapidity proportionate to that with which it was produced, a state of intense fever would have been excited, and a sudden increase of labour would have been thrown on the eliminating organs, that would have been highly prejudicial to the general safety of the economy. This latter state of things, it is true, we do get occasionally in those cases where the febrile symptoms are strongly marked, but the local affection almost absent; and these are just the cases whose convalescence is most protracted, and which leave the constitution in the most shattered condition from the violence of the shock to which they have subjected it. The amount of fever is by no means necessarily in a direct ratio to the intensity of the local affection; but it is always so to the metamorphosis of the fibrinous matter, and, consequently, to the subsequent increase in the quantity of excreta. And this is very intelligible; for, the resolution of fibrine into its simpler and more excretable elements is a process of oxydation, and involves a consequent elevation of temperature: hence, it is only to be expected, that where we have this oxydizing process going on very vigorously, we should find a corresponding rise in the general temperature—a symptom too evident in rheumatism, as well as in other diseases accompanied by rapid histolysis, to need further notice. In this light, therefore, the local affection in rheumatism will appear as a sort of safety-valve against the general constitutional severity of the disease, and as a symptom that ought to be rather cherished and encouraged than feared and depressed, except where, from its mechanical obstruction of the action of important organs, as the heart, it is fraught with immediate danger to the patient. In this light, too, we may call the exudation a *primary crisis* of the disease, the sudden exaltation of the excretions

at a later stage will then become a *secondary crisis*; the phænomenon thus bearing a considerable resemblance to the form of crisis, to which Dr. Stokes has drawn attention, as occasionally occurring in typhoid fever, when that disease terminates suddenly during a protracted convalescence in acute tuberculization of the lungs.

The preceding observations, though referring primarily to acute rheumatism, will apply equally well to the chronic form of the disease, in which the joint-affection results either from a succession of subacute attacks; or from a constant hypertrophy of the fibrous tissues, dependent upon an excess of their proper pabulum in the blood. But I am anxious to draw attention to another chronic phase of this most protean dyscrasia which, as far as I am aware, has hitherto received but little notice from the profession, but which is of some importance in a pathological point of view: I allude to a connexion which frequently exists between rheumatism in early life, and fibrous degeneration of the liver and kidney in later years. The whole question of fibrous degenerations, complicated as it is by questions on which the greatest obscurity rests, must undoubtedly be considered as still *sub judice;* but there can be little doubt that the old idea of these changes being necessarily or even frequently the result of acute inflammatory processes, is fast disappearing from the mind of all reflecting observers.* With regard to one form of fibroid degeneration, however, that of the small, hard kidney, it appears to be acknowledged by many recent writers of repute as clinical investigators, that it is essentially distinct, both in its origin and pathology, from the enlarged forms of Bright's kidney, of which Frerichs and others have asserted it to be only a subsequent stage; and that it is closely allied to the fibroid degenerations of the liver, lungs, and other organs by which it is often accompanied.† It seems probable, also, that careful clinical observation will establish the fact, that there are at least two forms of cirrhosis of the liver, and that the small contracted form is distinct in its origin and pathology from the enlarged form, as has been suggested by Dr. Todd. However, this is no place to discuss these points: I only refer to them, *en passant*, as connected with the question of granular kidney and its relations to fibroid degenerations in general.

Now, that there is often a connexion between renal disease and rheumatism was indicated long ago by Dr. Christison; who, however, does not seem to have attempted any explanation of it. He says: "One of the most frequent secondary affections connected with granular kidney is chronic rheumatism. On investigating the early history of many cases which have come under my notice in the advanced stage,

* See especially Dr. Handfield Jones' articles on Fibroid Degenerations in *British and Foreign Medico-Chirurgical Review*; vol. xiv.

† See Dr. Wilks' cases of Bright's disease, in *Guy's Hospital Reports*, vol. viii; also *Jones and Sieveking's Pathological Anatomy*, p. 619; and *Clinical Lectures on Urinary Diseases*, by Dr. Todd.

my attention has been drawn to the frequency with which reference was made to rheumatic pains as one of the earlier symptoms: repeated instances of the same complications have occurred after the admission of patients into the Infirmary; and, in short, this connexion has appeared to me so far common that I never meet with cases of obstinate chronic rheumatism without being led to make inquiries into the state of the urinary secretion."* An additional argument for the existence of acute rheumatism in the earlier part of the lives of those who subsequently die with granular kidney, is the frequency with which old diseases of the heart are met with in their cases.† In Dr. Wilks' cases above referred to, out of thirty-three cases of death in which the hard contracted kidney was found, and in a large number of which no history whatever was obtained, there are four cases in which it is distinctly stated that the patients had laboured under acute rheumatism in early years; and it is open to doubt whether even in all those from whom a history was obtained this point was investigated. This, it may be said, is no large per-centage, and these cases may have been merely coincidences; but it is rather singular that out of fourty-four cases of the large white, coarse granular, and lardaceous forms of kidney degeneration in the same report, not a single mention of rheumatism is made! The fact, however, that the concurrence is a real and not an apparent one, is corroborated by many cases which I have met with in the journals, in books, or have myself observed, all of which have tended to convince me that the rheumatic dyscrasia is a very frequent, though not an essential element in the production of fibroid degeneration of the kidney, liver, and other important organs. To those who are acquainted with the literature of renal pathology, it does not require reminding that Drs. Todd, Johnson, and others have been in the habit for some time of using the term "gouty" as a synonym for the "contracted" kidney, and of pointing out how frequently it is connected with the gouty diathesis: the former author, indeed, gives some cases in his Clinical Lectures above quoted, which by many persons would be rather diagnosed as rheumatic than gouty in their affinities; but with this exception he has treated the subject in his usually clear and able manner, although, with all deference to his high authority, I cannot but think that he has failed to appreciate the whole of the pathology of the change which he discusses. The connexion between rheumatism and renal disease was not unnoticed by Rayer, who gives an excellent description of the condition of the kidney, both in the chronic and acute stages.‡ As far as my own observations go, these rheumatic cases of granular kidney are the ones which are most frequently accompanied by an allied degenera-

* *On Granular Degeneration of the Kidneys*; p. 97.

† Christison: *Id. loc.*

‡ *Maladies des Reins*: art., *Néphrite Rhumatismale.*

tion of the liver, and occasionally of the spleen and lungs; but more extended observations are requisite to set the matter in an indubitable light. In these cases it would occur as if the fibrine-crasis—so to designate conveniently a condition which could be only otherwise described in a periphrastic manner—at first directed its activity against the fibrous structures of the joints, pericardium, &c., and afterwards levelled its attacks upon the fibrous matrices of the abdominal viscera; first producing hypertrophy of them, and then leading to the contraction which often results from this (as in the cicatrices of burns)—to the atrophy of the organs into whose structure they enter—and finally to complete abolition of their functions. This change in the affinities of the fibrinous plasma must depend either upon an alteration in its type, or upon the exaltation of the fibrous structures of the abdominal viscera over those of the joints as attractive centres. In the latter case it becomes an interesting question whether the increased work which is thrown upon the liver and kidneys in the rheumatic diathesis may not serve as a kind of *point de départ* in exalting the affinities of their fibrous structures for their normal pabulum. In relation to this hypothesis the view of Henlé may be quoted, who considers the hypertrophy and subsequent contraction of the fibrous matrix of the kidney one of the weightiest elements in the production of Bright's disease (the contracted form), applying to it the name of cirrhosis of the kidney.*

In the foregoing considerations on the nature of rheumatism, I have endeavoured to exhibit the hyperinotic condition of the blood as the most important element of the disease, and to show that the different organs to which the rheumatic dyscrasia may direct its activity are probably determined to a certain extent in accordance with variations in the type of the dyscrasia itself. I have also attempted to show that all the phænomena of this disease are perfectly explicable on this hypothesis, and that any theory of their pathology which omits a consideration of the concomitant hyperinosis, is deficient in a most fundamental particular. I might further extend this observation to pneumonia, and to other idiopathic phlegmasiæ, were this the place to do so; but this would lead to a discussion of matters that could not conveniently be included within the limits of an ordinary paper. Indeed so obvious is the necessity of not overlooking this most essential feature in erecting a pathology of inflammatory affections, that it did not escape the notice of Andral, even writing at the time from what was, and almost still is, the hot-bed of the Broussaian doctrines; from whose philosophical and most suggestive little work on Pathological Hæmatology I shall take the liberty of quoting a few passages. "On the other hand," he says, after referring to that hyperinotic condition of the blood, which is undoubtedly an *effect* of a traumatic lesion, "there are of a truth cases

* See his *Handbuch der Rationellen Pathologie*; b. ii, s. 305.

where the alteration recognizable in the solids is so slight and fluctuating in its nature, and occasionally so transient, that one can hardly conceive that it can be the cause of the profound and persistent change which the blood exhibits in these cases. Is not this the case, for instance, in many articular rheumatisms? and is it not remarkable that this disease is one of those in which the increase of fibrine is most considerable? In all this there is much that is yet unknown to be investigated: we have to discover the mysterious relation which unites in those diseases which are called phlegmasiæ the alterations of the solid to that of the blood; but in all cases we may now conclude from this simultaneity of affection in the two, that what we call an inflammation is a disease which does not reside solely in the solid where anatomical examination reveals its existence—that it is not only by so-called sympathetic irradiations that it exercises any influence upon the rest of the organism, and that the alteration which exists in the blood must play an important part. An inflammation is not, therefore, merely a local affection. Is it upon the excess of fibrine that the fever which accompanies every acute phlegmasia depends? In answering this, it must always be remembered that there is an almost constant correlation existing between these two facts; and that if the fever arises at the same time that the fibrine increases, on the other hand it ceases directly the fibrine returns to its normal amount."* Everything which comes from so acute and sagacious an observer as Andral, must carry with it the greatest weight; and these views are confirmed by Becquerel and Rodier in their recent work on Pathological Chemistry, where they observe, "An increase of fibrine in the blood is characterized by a remarkable phænomenon, viz., a tendency to secondary inflammations. The diffuse nature of the joint affection in acute rheumatism, and the occurrence of endocarditis in the course of this disease, as well as of pneumonia, pleurisy, &c., is in all probability a result of the increase of the fibrine."†

There is a strong collateral proof of the connexion existing between phlegmasial affections and a precedent hyperinosis, in the fact established by Andral and other observers, that in the period immediately preceding and following parturition a hyperinotic condition of the blood is a normal and physiological state. The question, therefore, suggests itself —is this period fertile in inflammation? A glance at the nature of the puerperal class of diseases is sufficient to answer this in the affirmative; but the inflammatory tendency is chiefly confined to the pelvic viscera and the parts in their immediate neighbourhood; for the exciting causes of inflammatory affections of the joints, heart, and lungs, are generally absent in women who have been confined for some time to a recumbent or sitting posture, and have been exposed to no vicissitudes of tempera-

* *Hématologie Pathologie*; art., *Phlegmasies*.
† *Op. Sup. Cit.*; p. 57.

ture; whereas the local irritation in and about the uterine region forms an effective determining element for inflammatory affections in that quarter. This theory of the influence of the hyperinoses of pregnancy in predisposing to inflammatory affections is confirmed by MM. Becquerel and Rodier, who remark that "it is not improbable that this increase of fibrine [in pregnancy] may account in a manner for the facility with which the phlegmasiæ manifest themselves during the puerperal period which follows the act of parturition."* Chomel, however, distinctly states that acute rheumatism is by no means an infrequent sequela of parturition; in which he is borne out by Bouillaud, though it might be suggested that some of the cases which have been described by that author, under the designation of *arthrite rhumatismale puerperale*, look uncommonly like purulent affections of the joints consequent upon pyæmia. It is to be presumed that these instances of puerperal rheumatism occur in patients who prematurely expose themselves to vicissitudes of temperature, or undertake labourious occupation before nature has removed the excess of fibrine by the usual outlets of excretion. To this condition is also probably attributable in great part the rapid progress which pulmonary tuberculosis makes after parturition; which Rokitansky explains by referring it to the increased facility which is given to pulmonary action after the uterus is emptied, in accordance with his theory of the dependence of tuberculosis upon a high development of the arterializing process; but looking to the recognized connexion between tuberculosis and a hyperfibrinated condition of the blood, it seems only reasonable that the spontaneous creation of the latter state during parturition should be followed by a tendency to deposit when the exigencies of the system no longer require the fibrine, especially if a nucleus of attraction exists at the same time in the shape of previously deposited tubercle. And as in the different forms of pathological hyperinosis the fibrine takes on a developmental type and power of independent vitality commensurate with the general constitutional vigour of the system, so in the puerperal hyperinosis the excess of fibrine which remains after parturition may do the same; hence the peculiar proneness of the patient to rheumatic or tubercular forms of exudation will depend partly upon the accidental circumstances under which she may be placed, but much more intimately upon the developmental type which the system may be able to impress upon the exudation itself.

Hitherto the pathology only of rheumatism has been discussed: it now remains to be seen what light can be thrown upon its etiology, and more especially upon the origin of the hyperinosis by which it is characterized. The sources of an excess of fibrine in the system probably fall under three heads: 1st, as a result of imperfect primary

* *Op. Sup. Cit.*; p. 56. See also *Mackenzie on the Connection between Phlegmasia Dolens and the Hyperinosis of Pregnancy—Medico-Chirurgical Transactions*; 1853.

assimilation; 2nd, as a result of metamorphic processes—normal in nature, but extreme in amount; 3rd, as a result of defective elimination of the fibrine by the excretory processes provided for that purpose. An excess of fibrine as a result of imperfect primary assimilation may be supposed to arise in two ways: either from a highly animalized form of diet, and a preponderance of the albuminous elements of the food, or from a comparative deficiency of the fatty matters which combine with them to form healthy chyle. With regard to the former source, I know of few facts which would justify our accepting it as the cause of a constant hyperinotic condition of the blood, at least in rheumatism; and the experiments of Majendie and others show that a pure albuminoid diet does not tend to produce exudations of any kind, but rather to develop a species of dyscrasia, which ends in the speedy breaking up of all the tissues.* The partial absence of fatty matters in the food, however, and the consequent formation of an imperfect chyle, is a very different question, and deserves a careful consideration.

The importance of a due proportion of fatty matters in every form of diet to a continuance of health, is so generally insisted on by all physiologists, that it is unnecessary to offer any proofs of it here. I need only refer to Professor Bennett's work on the Oleum Jecoris Aselli for an able summary of the histogenetic functions of fat in the animal economy, and a reference to its diminished amount in the food as an important cause of tuberculosis. The notion of the distaste of phthisical patients for fatty matters has long been entertained, and is, in fact, so patent in well marked cases, that it could not well escape notice; but I am not aware that it was ever regularly demonstrated, or its value statistically determined, until Mr. Hutchinson in an able paper on the "Dyspepsia of Phthisis," in the *Medical Times and Gazette*, about three years ago, showed, by a careful and detailed examination of a large number of consumptive patients, that its existence was even more common than had been supposed. He also pointed out the importance of this distaste in a prognostic point of view, and showed that the greater the distaste of the patient for fatty matters, the less hope was there of the toleration and consequent beneficial effect of the cod-liver oil; and that those cases, also, were the least amenable to any kind of treatment in which the distaste was most intense.

Whilst residing at the Brompton Consumption Hospital some time ago, I examined a large number of patients with the view of testing Mr. Hutchinson's statements, and I can most amply corroborate them as regards the importance of this distaste for fat in phthisical patients. It is true that there are many cases of phthisis in which no such a distaste is traceable; and, looking at the varied circumstances under

* The fact that a disease so closely allied to rheumatism as gout is generally connected with a highly animalized form of diet, is however worthy of consideration in reference to this point.

which that vitiated state of the constitution which results in tuberculosis may be produced, any theory which attempts to ascribe it to one uniform set of causes is illogical and absurd. But there cannot be a doubt when we consider the great histogenetic importance of fatty matters, and especially of those which enter into the constitution of the chyle, that this often long existing and intense distaste, explain its origin as we will, is a very serious and important symptom. From considering the relationship of this distaste to the hyperinotic state of the blood in phthisis, I was led subsequently to extend the same examination to patients labouring under acute rheumatism, and I found that in a very large number of the worst cases—in those in whom the dyscrasia seemed to have established itself with the firmest hold—the same distaste for fatty matters was present, extending in some cases almost to a disgust for them. I should state that I have found this symptom to exist more commonly in female than in male patients: why so, I am unable to suggest, unless it be that the digestive functions of the former class share in the more delicate sensibility and emotional susceptibility which their bodies generally possess as compared with the stronger sex. Many causes have prevented my tabulating the results of this examination in such a form as would have rendered them available for subsequent reference; but the impression produced on my mind by a considerable number of cases is, that in acute rheumatism the distaste for fatty matters plays nearly as important a *rôle* as it does in phthisis. Now, the result of this imperfect assimilation of fatty matters, continued as it is in some cases for many years, must seriously affect the composition of the chyle, and through it that of the blood; and the tendency of this deficiency of fatty matters in the chyle would probably be to leave its albuminoid elements in a state of imperfect emulsification, so to speak, and to give them an undue preponderance in the constitution of the blood, and more especially in that of the blood corpuscles. Now, it is very possible that this deficiency of fatty matters in the newly formed blood corpuscles may lower the standard of their vitality and give them a tendency to premature and sudden disintegration, under the influence of agents by which they would be normally unaffected—a state of things which we shall presently see to be of highly probable existence in the rheumatic dyscrasia especially, and to be one of the most important predisposing elements to acute attacks of the disease. To these remarks upon the importance of the fatty matters of the food, however, it may be objected that physiology has shown that one of the most important functions of the liver is the formation of fat, and that so long as this organ performs its office, the ingestion of fatty matters from without is not absolutely necessary. To which it may be replied—firstly, that in most of phthisis, as also in many of rheumatism, the functions of the liver are most imperfectly performed from a very early

stage of the affection, and, consequently, the supply of fatty matters to the system by that organ must be very inadequately discharged; secondly, that there are reasons for believing that the fat which is generated in the liver is sent into the blood in a free state, and is probably not destined for any higher end than the formation of adipose tissue, or by its oxydation to serve as a calorifacient material; whereas, the fatty matters which are absorbed by the alimentary canal are always intimately combined with albumen, and are probably incorporated at once into the vital unity of the tissues by being taken into the constitution of the newly forming globules. Certain it is that the globules contain a notable amount of fatty matters; and when we consider the whole amount of them circulating within the body, and the constant necessity for a new creation to supply the place of those that disintegrate, it will hardly appear that the quantity of fat taken up by the chyliferous ducts is too large to be accounted for in this way. At any rate, it is very clear that nature would never have dictated the necessity of the due admixture of fatty matters with the food with such force as she has done were such mixture a non-essential one, or could it be dispensed with for any length of time with impunity; and if nothing else were adducible to show the connexion existing in many cases between tuberculosis and *chronic* rheumatism at least, and a deficiency of fatty matters in the system, the sudden and marked improvement consequent upon the administration of cod-liver oil to patients labouring under those diseases would of itself be a highly probable indication of it. I have suggested that the long continued deficiency of fatty matters in the food would lead to a hyperinotic state of the blood: under ordinary circumstances the excess of fibrine would probably be eliminated by the excretions in the form of urates, lactates, urea, &c., a slight pyrexial condition being set up frequently, and the urine becoming more high coloured or the perspiration more copious than usual—a state of things which is notoriously of frequent occurrence in phthisical and rheumatic subjects. It seems, also, highly probable that the severe night sweats which generally distinguish the advance of phthisis are connected with an elimination of the results of the disintegration of this excess of fibrine, just as those of chronic rheumatism may be. It is certain that they must have some physiological function of an excretory character, and that they cannot be explained simply by a reference to the depressed vitality of the patient, inasmuch as they never occur in many diseases which are accompanied by extreme prostration, and in which the blood is in a much more favourable condition for the elimination of its watery portion than it is in phthisis—*e. g.*, dropsy.

We have now to enter upon the consideration of one of the most interesting, but at the same time one of the most obscure, questions connected with the subject of hyperinosis, viz., the rationale of its

immediate production, involving as it does the much debated question of the relationship of fibrine to the other elements of the blood. In the present undetermined condition of our knowledge on this point, it would obviously be unsafe to make any very dogmatic statements upon the positive origin of this substance; yet, on duly estimating the weight of the respective theories which have been propounded on this subject, it seems to the writer that the weight of evidence is decidedly in favour of the doctrine which regards fibrine as a further stage in the transformation of albuminoid matters, and as normally intended for histogenetic purposes; and, also, that the special theory of the late F. Simon, which regards it as a product of the disintegration of the globules, has many important facts to recommend it, though perhaps it may err in being too exclusive as to the possible production of fibrine from any other source. Much of the confusion that has arisen on this subject would seem to depend upon the failure of many authors who have discussed it, to apprehend the fact that there are in all probability several varieties of fibrine, perhaps, as suggested by Lehmann, of an allotropic character. In the present state of our chemical knowledge, our estimation of the nature of these varieties of fibrine can only be arrived at by a study of the affinities which they exhibit in certain pathological conditions for the various tissues of the body; and by such a study, combined with the prosecution of a more exact analysis of the blood in these conditions, it is to be hoped that we shall in time arrive at a more definite acquaintance with the subject than we at present possess.

According to the hypothesis of Simon above referred to, which he has developed with much ingenuity in his work on *Animal Chemistry*,* and which I shall here adopt, notwithstanding the weighty arguments which have been brought against them in some points by Zimmerman,† because in their general tenour they have been confirmed by the researches of subsequent investigators, the fibrine of the blood is the result of the metamorphosis of its globules: this metamorphosis is a vital phænomenon, and is due on the one hand to the influence of the oxygen with which the globules are brought into contact during their passage through the lungs, and on the other to the numerous reactions which occur to the blood when circulating through the various tissues of the body. Further, in addition to fibrine the disintegration of the globules gives rise to the transformation of their hæmatin into the hæmaphæin or colouring matter of the plasma. For the great ability with which Simon has stated his argument, I can only refer to his work above quoted; but a few of the facts upon which it is founded may be here mentioned, especially those which have a bearing upon the subject in hand. It follows from this hypothesis that if the fibrine

* Vol. i, pp. 147 *et seq.*; Sydenham Society's edition.
† See his *Ueber die Analyse des Blutes*, &c.

be the result of the decomposition of the blood globules, wherever the former is in excess the latter should be deficient: that this is really the case is proved by many facts. In chlorosis, in which the fibrine is increased, according both to Andral and Becquerel, the globules are correspondingly diminished; whilst the peculiar discolouration of the skin in that affection would seem to bear out Simon's explanation of the formation of the greenish-yellow hæmaphæin of the plasma. So important is the hyperinotic element in chlorosis in the estimation of Becquerel, that it suffices, according to him, completely to differentiate that affection from simple anæmia. Again: after violent exercise the amount of fibrine in the blood is increased, probably as a result of the unusual disintegration of the blood globules which is necessary to repair the waste of tissue consequent upon the exertion: hence the susceptibility of men who have undergone great exertion to inflammatory affections, rheumatism especially being more frequently induced by fatigue than by any other cause. In pregnancy, where the fibrine is increased, the globules diminish at a very early period of gestation.* Andral, Becquerel and Rodier, and Simon, all refer to the inverse ratio of the fibrine and the globules to one another in phlegmasial affections; and the latter expressly asserts that in all his analyses he has never found the one increased without the other being diminished. There is often a peculiarly chlorotic hue exhibited by the subjects of severe inflammatory diseases, especially pneumonia, which may also be susceptible of explanation on the theory of Simon in reference to the transformation of the colouring matters above quoted. In all the specific fevers, on the other hand, the fibrine is below the standard of health, unless local inflammations are present, whilst the globules exhibit their average amount.

The efficient agent in the transformation of the blood globules, according to Simon, is the oxygen received by the lungs; and in accordance with this theory he thus accounts for the increased activity of this process in inflammations. In all phlegmasiæ the circulation is more or less accelerated, and from its being thus quickened the globules acquire more frequent contact with the oxygen of the air, and pass oftener through the different tissues and organs. It follows, therefore, that more globules are converted into fibrine in a given space of time; hence an absolute increase of this element in the blood, and a decrease in the proportion of globules, which are no longer renewed by the introduction of food. To this theory it may be objected, as Becquerel observes, that these changes ought to take place whenever the circulation is accelerated; and we, consequently, ought to have a hyperinotic state in the specific fevers. To which it may be answered, that although the normal effect of an increased circulation of the blood may be to cause

* Becquerel and Rodier: *Op. Sup. Cit.*: p. 96.

a disappearance of its globules and an increase of its fibrine, yet it by no means follows that this should necessarily be the case under all circumstances; for it may happen that a change in the density or chemical reaction of the blood serum, or the temporary suspension of the functions of those organs whose duty it is to effectuate the disintegration of the globules, may put the latter in a very different condition to that in which they ordinarily exist.* Another objection has been raised to the exclusiveness of Simon's theory, founded upon the fact that not only do the corpuscles of the blood, but also its albumen, decrease in a direct ratio with the increase of the fibrine; hence it has been supposed that fibrine may be a result of the metamorphosis of the albuminous elements of the plasma. Of the fact itself the researches of Becquerel and Rodier leave no doubt, nor is the inference which is drawn from it by any means unreasonable; but is in accordance with other physiological facts. Hence it is possible that there are two sources from which fibrine is derived—from the disintegration of the corpuscles, and from the metamorphosis of the albuminous elements of the plasma; though whether these two may not be ultimately referable to a single one on the supposition that the albumen of the plasma is itself derived from the disintegration of the corpuscles, and an intermediate stage between them and fibrine, may be a subject for discussion. Upon the fact, however, that all these metamorphoses involve in some way or other an active oxydizing process and a consequent production of heat, many pathological phænomena, as well as the experiments of Drs. Richardson, Gairdner, and others, leave no reasonable doubt; but it may be questioned whether the theory of Simon, which attributes the first step in the transformation to the increased vigour of the oxydizing process, does not err in omitting to take into consideration the vitality and affinities of the blood corpuscles themselves, which must vary under different circumstances, and at all times must be more or less under the influence of the nervous system. The experiments of Bernard show that the sympathetic exercises a primary and most important influence over this metamorphosis; but whether it be in merely regulating the physical conditions of the vessels, or in directing the vital affinities of their contents, must be considered as yet undetermined. It must be unquestionably admitted that on many of the details of the formation of fibrine there still rests the greatest obscurity: no one, however, who considers carefully the physiological and pathological data here laid down, can have much doubt that they substantiate the general view of the process here laid down. That fibrine is not a result of the disintegration of the tissues seems to be clearly indicated by all that we know of the certain

* According to M. Cahen the proportion of fibrine in the blood is in an inverse ratio with the alkalinity of the serum, and in all hyperinotic conditions the proportion of the salts of soda is less than usual.—*Recherches Experimentales sur l'Alcalinité du Sang Humain*—*Bulletin de l'Academie de Médecine*; 1850.

products of the metamorphosis of tissue: nearly all the facts with which we are acquainted tend to show that when a member of the albuminoid group of elements has once entered into the constitution of the tissues, its removal from them involves the assumption of a metamorphic condition much more approximated to inorganic compounds than fibrine is—*e.g.*, fat, creatine, uric acid, urea, lactic acid, &c., all of which are known histolytic results. Finally, the doctrine of the formation of fibrine from the disintegration of the blood globules derives some support from the visible phænomena of inflammation, as they have been described by Professor Bennett, Mr. Paget, Brücke, and others: one of those which precedes the exudation of the fibrinous plasma being the aggregation of the corpuscles to one another, and then their disintegration and fusion into a solid mass, which appears to be the source of the fibrinous exudation. The increase of the pigmentary matter in the urine in phlegmasiæ is also a probable indication of an extreme disintegration of the corpuscles, as is suggested by Dr. Parkes (*Loc. Sup. Cit.*), and consequently of the accompanying increase of fibrine.

Before concluding, I wish to say a word or two by way of practical inference from the views above expressed as to the treatment of acute rheumatism. A consideration of the state of the blood in this disease clearly shows that there are two chief indications to fulfil in its treatment, viz., to check if possible the rapid formation of fibrine which characterizes its early stage, and to promote the elimination of that excess by the outlets which nature has provided for it in the renal and cutaneous excretions. The two most practicable methods of accomplishing the first point would seem to be, by the administration of large doses of caustic alkali, as frequently practised, and as indicated by the statement of M. Cahen above quoted; or by the employment of such neutral salts as possess at the same time a solvent action on fibrine, and diuretic properties. MM. Becquerel and Rodier assert that bleeding produces a decided diminution in the fibrine of the blood. This observation is certainly opposed to those of other authors; and, even if it were correct, it is a serious consideration whether this advantage of bleeding is not counter-balanced by the prolongation of the convalescence, and by the tendency to relapse which it produces: if justifiable at all, it certainly is so only in the earliest stage, in strong constitutions, and in a sthenic type of affection. The statement, also, of Becquerel—that immediately after a large bleeding in rheumatism the urine assumes for a period the characters of that of anæmia—is certainly corroborative of its effect in diminishing the amount of fibrine. The fulfilment of the second indication cannot be better carried out than by the treatment sometimes adopted, viz., the copious administration of slightly acid drinks, the necessity of an abundant supply of aqueous matter to the blood being indicated in this as

in all other febrile affections by the extreme thirst present. A knowledge of the true pathology of rheumatism will enable to estimate at their proper worth the real utility of the various specifics which are constantly being vaunted, even in the present day, as a cure for the disease, and to see that rheumatism, like all the other phlegmasiæ, has a definite course to run, and that when once completely established it cannot be cut short. The danger of relapse which a too early adoption of an animalized form of diet entails is well established by experience, and may be explained by reference to the fact that the fibrinous elements of the blood are already in excess, hence the premature indulgence in a meat diet is only adding fuel to the fire which has hardly yet burnt itself out in the system, and instead of giving strength is but arousing the slumbering energy of the disease. But there is one medicament which both theory and experience indicate as a dietetic remedy as soon as the febrile symptoms have disappeared, and the patient is entering upon the convalescent stage; and that is the cod-liver oil. It is somewhat strange that this remedy should have been employed for years with success in the chronic form of rheumatism, and yet be rarely or never used in the acute variety of the disease. If the views which I have endeavoured to substantiate be correct, it is evident that this is almost the only article in the Materia Medica which promises to be of any avail in the constitutional and prophylactic treatment of rheumatism. Looking at the relationship which I have shown frequently to exist between a chronic distaste for fatty matters and the rheumatic dyscrasia, it can hardly be denied that we have here alone a very strong indication for the administration of the oil. All modes of treatment which we at present employ can only claim to be directed against the transient symptoms of the attack, the constitutional predisposition being untouched, and no immunity given against future invasions of the disease. It is with the view, therefore, of combating the dyscrasia itself that I would recommend strongly the administration of the oil during the convalescent stage, and a persistent continuation of it for some time afterwards, in proportion as the dyscrasia seems firmly rooted in the system, or when oily matters are specially indicated by the previous existence of a distaste for them. Of course, as this distaste itself must have a cause, probably in an atonic state of the digestive system, much of the good to be derived from the oil will depend upon the mode of its administration, and upon what measures are taken to ensure its toleration by the stomach. I would also refer to the necessity of impressing upon patients who may require such advice, the importance of cultivating the habit of eating a due proportion of fat with their meat—a habit whose establishment in early life is to be especially recommended.

The object of this paper has been to show that the fundamental and

characteristic feature of rheumatism is a hyperinotic condition of the blood, and that all the other phænomena can be most satisfactorily explained on this basis. I have also endeavoured incidentally to show that Simon's theory goes a long way to explain the method by which this condition is immediately produced, and that it is borne out by some of the symptoms which present themselves in the course of the attack. How far I have been successful, or not, the reader must judge for himself; but I must specially protest against the imperfect manner in which the theory has been treated here being allowed to militate against its reception. It would be impossible, even in a paper of double this length, to discuss in a sufficiently comprehensive manner the many important questions which enter into its consideration: any parsimony, therefore, which may have been exhibited in the treatment of its details has been dictated rather by the necessities of space than by a want of matter. No one can be more conscious than I am myself that even this theory, greatly as I believe it to be in advance of those which at present hold the field in the scientific world, leaves many points unexplained which can only be cleared up by the future progress of science, and many more in obscurity on which perhaps we may never obtain light. Still, however great our darkness may be on some points, it should form no bar to an attempt to methodize our knowledge on others; and there is the strongest ground for asserting that the gleaners in the field of science have already accumulated a sufficient number of facts in relation to rheumatism to enable us to place its pathology in a clearer light than has hitherto been done. Much yet remains to be accomplished; but the work of future explorers will be facilitated in proportion as they are able to rationalize the labours of those who have preceded them, and to interrogate nature by the aid of hypotheses which have a shadow of truth, at least, in their favour. For it is by the copious employment of hypotheses alone that the philosopher can hope to tread successfully the slippery path of scientific discovery. In the words of an able and profound writer, "He who has to discover the laws of nature may have to invent many suppositions before he hits upon the right one; and amongst the endowments which lead to his success we must reckon that fertility of invention which ministers to him many such imaginary schemes, until he finds one which conforms to the true order of nature."* It is hardly necessary to add that the general doctrines upon which this theory is founded are by no means new; but have been developed by some of the first continental authors, and have been warmly endorsed by many authors of great repute in our own country. To the reader who is *au courant* with the literature of the day, it is unnecessary for me to point out how much I am indebted to them; and I trust that any omission to cite

* Whewell: *Philosophy of the Inductive Sciences*; vol. II, p. 34.

chapter and verse on many occasions may not be imputed to an affectation of originality on my part. I must, however, specify one work in particular as one from which much of the present paper has drawn its inspiration: I mean the *Surgical Lectures* of Mr. Paget, to whom, as much as to any living English writer, science owes an inestimable debt of gratitude.*

* Since the above was written, but contemporaneously with the appearance of the first part of the paper, an extremely interesting contribution has been presented to the pathology of rheumatism by Mr. Toynbee (*Medical Times and Gazette*; April 19, 1856), in which he advocates a view of the disease identical in many points with the one supported in the preceding pages. Mr. Toynbee's coincident and unexpected corroboration, independently of his merits as an original observer, will, I trust, give the theory a better claim to examination than it possessed before.

Dr C. J. Parkes
with the Author's
kind regards,

ON THE

DETERMINING CAUSES

OF

VESICULAR EMPHYSEMA

OF

THE LUNG.

BY

WILLIAM JENNER, M.D., F.R.C.P.,

PROFESSOR OF PATHOLOGICAL ANATOMY, UNIVERSITY COLLEGE; PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL, THE HOSPITAL FOR SICK CHILDREN, ETC.

[*From Volume XL of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

J. E. ADLARD, BARTHOLOMEW CLOSE.

1857.

ON

THE DETERMINING CAUSES

OF

VESICULAR EMPHYSEMA OF THE LUNG.

BY

WILLIAM JENNER, M.D., F.R.C.P.

PROFESSOR OF PATHOLOGICAL ANATOMY, UNIVERSITY COLLEGE;
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL, THE
HOSPITAL FOR SICK CHILDREN, ETC.

Received Oct. 22d, 1856.—Read Jan. 13th, 1857.

Since the attention of physicians was fixed on vesicular pulmonary emphysema by Laennec, and the anatomical character of the disease demonstrated by that pathologist, the question as to the determining cause of the dilatation of the air-cells has been a fruitful source of controversy. The question to be decided is one of much practical interest; for as vesicular emphysema is incapable of cure its prevention is of the highest importance, and to determine its cause is to determine the possibility of its prevention, and to some extent the means to be employed for its prevention.[1]

[1] If the advocates of the theory now generally received, and which has been termed the inspiratory theory, be correct, then powerful expiratory efforts, so far as concerns the production of emphysema, are invariably to be encouraged as essentially preservative, the inspiratory efforts only are to be moderated. "Die Causalindication besteht . . . . in Vermei-

That vesicular emphysema may occur in lungs, the texture of which is otherwise perfectly healthy, is beyond doubt; we see examples of this daily in the acute vesicular emphysema of children. But then it is equally beyond doubt that in certain varieties of vesicular emphysema, *e. g.* that in which one or more vesicles at the apex or margin of the lung are greatly enlarged—the texture of the distended vesicles is constantly abnormal. Mr. Rainey[1] has rendered it highly probable that fatty degeneration of the tissue of the lung is one of the anatomical conditions on which loss of its elasticity and contractility depend. While my own observations have led me to the conclusion that the most frequent anatomical change of the lung having this result is fibrous degeneration, the consequence of the exudation of that variety of lymph which escapes from the capillaries where they are seat of slight, but long-continued congestion, in a person of tolerably healthy constitution. So common is the exudation of this variety of lymph in the congestions of old persons, that the changes of tissue which result from its presence are to be reckoned among the degenerations incident to old age. The loss of elasticity and contractility and permanent dilatation of the part which is its seat, if subjected to a distending force, is well illustrated by a reference to the large size so often attained by varicose veins when their walls are the seat of this degeneration.

The contractile power of the lung being impaired, it is evident,—that every single forcible distension of the air-cells must be followed by a certain degree of permanent dilatation; that an amount of expansion of the cells which would

dung inspiratorischer Anstrengungen, welche die gewaltsame Ausdehnung der Luftzellen steigern können." (Canstatt's 'Specielle Pathologie und Therapie, Band ii, p. 636.) If the expiratory theory, which I advocate, be correct, then the great object of the physician, so far as concerns the prevention of emphysema, must be to moderate, in many cases at least, the violence of the expiratory efforts. Persons disposed to emphysema or affected to a slight degree with that disease must avoid occupations which necessitate expiratory efforts with a closed glottis, *e. g.*, carrying great weights, powerful pushing or pulling.

[1] 'Medico-Chirurgical Transactions,' vol. xxxi.

be instantly removed by the normal contractile power of the lung-tissue,—must, if that contractile power be seriously diminished, require only to be frequently repeated to lead to extreme vesicular emphysema. The air-cells, whether their walls be or be not healthy, can be distended, so far as we know, by one power only, viz., the pressure of air on their inner surface, but then the air may be driven against their walls, both during inspiration and during expiration. Hence two theories of the determining cause of emphysema of the lung, termed, respectively, the inspiratory and the expiratory theory.

The inspiratory theory. Atmospheric pressure may be brought to bear with undue force on the inner surface of the air-cells by the enlargement of the capacity of the thorax during inspiration being disproportionate to the capacity of the air-cells. Some portions of the lungs being from collapse or other cause diminished in volume and incapable of dilatation, then as the lungs are kept in contact with the thoracic parietes during inspiration by the pressure of the atmosphere expanding the air-cells, and so increasing the bulk of the organs, the obliteration and diminution in size of one or more air-cells necessitates (*supposing* the enlargement of the capacity of the thorax in inspiration to continue the same as with healthy lungs) abnormal expansion of the unobliterated air-cells.

This theory of the determining cause of emphysema is essentially that of Williams, Elliotson, Watson, Hasse, Rokitansky, and others. It was moulded into its present exact form by Dr. William Gairdner, and strongly supported by him in a series of very able papers published in 1850. Dr. Gairdner maintains, that the inspiratory theory admits of universal application, and that no other reasonable explanation can be offered of the mode in which the dilatation of the air-cells is effected. The views of Dr. Gairdner on this point have been adopted almost without question by the latest writers on the subject, *e. g.*, Rilliet and Barthez 'Maladies des Enfants,' t. i, p. 601; Sieveking (Jones and Sieveking's 'Pathological Anatomy,' p. 411).

Nor do I deny that in some cases a certain amount of dilatation of the air-cells is determined by inspiration.

The expiratory theory.—The object of this paper is to show that by far the most common and efficient cause of vesicular emphysema of the lung is the pressure of the air contained in the lung brought to bear on the inner surface of the air-cells by the expiratory efforts.

The expiratory theory I am about to advance is altogether different from the erroneously termed expiratory theory of Laennec.[1]

Powerful expiration is, it seems to me, infinitely the most frequent determining cause of acute vesicular emphysema and of the chronic vesicular emphysema which accompanies chronic bronchitis. It is probably the constant determining cause of the vesicular emphysema which supervenes on chronic congestion of the lungs and bronchial tubes, and on diseased heart, and of the atrophous emphysema of the aged. In like manner, expiration appears to be the invariable determining cause of vesicular emphysema whenever it is considerable in degree and general, or occupies chiefly or only the apex and border of the lung, and whenever the dilatation of one or more vesicles is extreme.

It has been asserted that during expiration every part of the lung is equally compressed, and, therefore, that the cells of no one part can be over distended. Dr. Gairdner observes:

"But the most serious objection to the expiratory theory

[1] Laennec's is in reality an inspiratory theory. Starting with the assertion that the inspiratory force is greater than the expiratory, he states his theory thus, "l'air, après avoir forcé la resistance que lui opposait la mucosité ou la tuméfaction de la membrane muqueuse bronchique, ne peut la vaincre dans l'expiration, et se trouve emprisonné par un mécanisme analogue à celui de la crosse du fusil à vent. Les inspiration suivantes, ou au moins les plus fortes d'entre elles amenant dans le même lieu une nouvelle quantité d'air, produisent nécessairement la dilatation des cellules aériennes auxquelles se rend la bronche oblitérée." ('Traité de l'Auscultation Médiate,' tom. i, p. 302.) As the temperature of the air in the lungs is considerably above that of the external air, its dilatation after entering the air-cells must tend still further, Laennec fancied, to over distend the air-cells.

is that the expiratory act is mechanically incapable of producing distension of the lung or of any part of it. The act of expiration tends entirely towards emptying the air-vesicles by the uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness by a closed glottis, any further distension of them is as much out of the question as would be the further distension of a bladder blown up and tied at the neck by hydrostatic or equalised pressure applied to its entire external surface. The air-vesicles can sustain no distending pressure from the column of air within the tubes, as that air only becomes compressed in virtue of a force which opposes exactly as much resistance without as it creates pressure within. It is singular that a theory so radically unsound and so devoid of proof should have been allowed to maintain a place in medical literature." ('Monthly Journal of Medical Science,' v. xiii, p. 10.)

But is it true that during expiration every part of the lung is equally compressed? Is it true that every part of the thoracic walls are during powerful expiratory efforts equally unyielding?

We have only to watch a person whose chest is exposed during a fit of coughing to see what a consideration of the anatomical constitution of the thoracic parietes would *à priori* lead one to assert, viz., that the answer to both these questions must be in the negative.

Before coughing a person makes a deep inspiration, *i. e.* he distends as far as possible the air-cells, he then closes the glottis and compresses forcibly the lungs by the thoracic and abdominal parietes, the moment the compression of the lungs has attained a certain point, he opens more or less the glottis, and the air is driven forward by the muscular effort and the elasticity and contraction of the lungs and of the thoracic walls with a force proportionate to the compression to which it was subjected before the opening of the glottis.

Now it is manifest that if there be parts of the thoracic walls which are more yielding, or which during powerful

expiratory efforts with a closed glottis contract less than others, that the air immediately before the opening of the glottis will be driven from the compressed portions of the lung into the air-vesicles of the lung situated under such parts of the walls with a force proportionate to the general muscular and other powers in play, to the local want of compression and to the degree of yielding of the walls at those particular spots. That there are such parts, and that they are exactly those which are most frequently the seat of vesicular emphysema, and the sole seats of extreme dilatation of the air-cells is demonstrable.

Above the first rib the lung is covered only by soft tissues, and certainly, compared with the part of the lung situated below the level of the second rib, it is subjected during ordinary respiratory efforts to very little external compression.

The air-cells of the lung above the first rib being then surrounded by comparatively yielding walls, and being comparatively or absolutely uncompressed during expiration, ought, one would *à priori* suppose, to be distended to the utmost when powerful expiration is made with a closed or imperfectly opened glottis; the air being necessarily driven from the compressed to the comparatively uncompressed parts of the lungs. And that during violent expiration with a more or less closed glottis, the air is actually driven into the lung-vesicles of the apex with power enough to distend them to the utmost, is demonstrated by the supra-clavicular bulging which may be seen during a fit of coughing; and the hand has only to be placed upon the same part to prove that the lung tissue subjacent is, during strong expiration, distended by a considerable force. Percussion proves that the bulging to which I refer is pulmonary. If the apex of the lung be the seat of emphysema then the supra-clavicular bulging during violent expiration is extreme, and percussion on the bulging part elicits an almost tympanitic sound.[1]

[1] The force which is exerted on the walls of the air-cells when the glottis is closed and powerful expiratory efforts made is also shown by the fact that during the expulsive efforts in parturition one or more air-cells occasionally give way, and the air finds its way through the posterior mediastinum into the cellular tissue of the neck and face. A case of this kind fell under my observation when a student. The woman, after a violent detrusive effort, in which necessarily the glottis was closed and the muscles of expiration called into action, suddenly exclaimed, "I am unable to see." Her face and neck were, I now found, the seat of subcutaneous emphysema.

The cells of the anterior margin of the lung are, like those of the apex, forcibly distended when violent expiratory efforts are made with the glottis closed, or imperfectly opened. The expiration of coughing is performed chiefly by the abdominal muscles forcing up the diaphragm, the ribs compressing the lungs laterally, and the sternum and the cartilages of the ribs anteriorly. But the cartilages are to some degree yielding, and the anterior margin of the lungs is to a considerable extent protected from direct pressure by the heart and great vessels; the consequence is, that the air is driven during violent expiration from the parts most compressed (*i. e.*, the central, basic, and lateral parts), into the air-cells of the anterior margin, as well as into the air-cells of the apex.

Again, the margin of the base of the lung, the part of the lung in the vicinity of the root of the organ below the entrance of the bronchus, and the little ridge of lung which lies behind the trachea on the right side forming the posterior margin of what may be termed the tracheal groove on the lung, are in a like manner imperfectly supported and comparatively uncompressed during violent expiration. The base, too, of the left lung generally, is less firmly supported than is the corresponding part of the right lung—the liver being a more unyielding organ than the stomach, and compressing the base of the right lung more uniformly than the spleen and stomach do that of the left lung. The consequence of this want of support and of compression of the parts of the lung last enumerated is, that they, like the apex and the anterior margin, are the chosen seats of

emphysema;[1] the air contained in the parts of the lung most powerfully compressed during expiration with a closed glottis, being forced in undue quantity into such parts of the lung as are less compressed and less perfectly supported.

It is manifest that what has been above advanced, as to the effect on the air-cells of powerful expiratory efforts made when the glottis is partially closed, is equally true of the same efforts when made with one or more bronchial tubes partially closed. Dilatation of some of the air-cells beyond the tubes must ensue, if those cells be not uniformly compressed during expiration.

While, therefore, collapse of the lung is the consequence of complete occlusion of a bronchial tube, dilatation of the air-cells may be the result of imperfect occlusion.

The objection to the theory of Laennec, urged by Louis, viz., that the ordinary seat of catarrh is the base of the lung posteriorly, that of emphysema the apex and anterior margin, is unanswerable. The objection of Dr. Gairdner to the same theory, viz., that impermeability of a bronchial tube is followed by collapse of the pulmonary tissue from which it leads, is equally fatal to it. But then the conclusion of Louis from his facts, viz., that there is no relation between catarrh and vesicular emphysema by no means necessarily follows. The relation between the two is, that catarrh excites frequently repeated violent expiratory efforts, with a more or less perfectly closed glottis, and therefore is one of the exciting causes of repeated over-distension of these air-cells, which during these efforts are the least supported and compressed. Catarrh, then, of the base of the lung posteriorly, may be the exciting cause of vesicular emphysema of the apex and anterior margin, and Louis's conclusion as to the necessary want of relation between catarrh and vesicular emphysema, because they affect different parts of the lung, is manifestly illogical.

[1] Louis found the lower lobe of the left lung twice as often the seat of emphysema as the corresponding lobe of the right lung; while the apex of the right lung was more frequently affected with emphysema than the apex of the left lung.

Dr. Gairdner's conclusion, viz., that obstruction of a bronchus, because it is sometimes followed by collapse of the air-cells, cannot be a determining cause of rarefaction of the part of the lung from which it leads, is equally untenable. For incomplete occlusion of the bronchi may have, and in some cases I believe does have, a result the reverse of perfect occlusion, and in catarrh and bronchitis imperfect occlusion of the tubes is the rule, perfect occlusion the exception.

The notes of three cases will suffice to illustrate the points dwelt on in this paper—the facts are so common and so patent to every observer, that it would be useless to give more.

Mrs. L—, æt. 63, tall, thin; suffered from winter cough; was "asthmatical on exertion;" the chest was small in circumference, but long; the intercostal spaces were wide, and fell in during ordinary inspiration; there was some recession of the supra-clavicular regions during inspiration. On coughing there was great bulging of the supra-clavicular regions, especially the right. On percussion, the bulging part gave out an almost tympanitic sound. Two days after the foregoing observation the lady died suddenly of fatty degeneration of the heart. The apices of the lungs were the seat of extreme local emphysema.

H. C—, æt. 46, a patient under my care in University College Hospital, suffering from dry pleurisy and emphysema. The subjoined note was taken a day or two after admission. The cough is more troublesome, and there is a large amount of sonorous râle, audible during expiration, over both sides of the chest anteriorly. Below the prominence opposite the second costal cartilage the sternum recedes during each inspiration; in the act of coughing it advances with each expiratory succussion; there is marked depression of the supra-clavicular regions during each inspiration, and marked bulging during each expiration. The infra-clavicular regions also advance during the expira-

tion of coughing. The expiration of coughing is chiefly effected by the abdominal muscles, and these are called into play even as low as the pubes.

Catherine C—, æt. 1 year 9 months, was admitted under my care into the Hospital for sick children suffering from Croup. The following note was taken in the morning as she died in the evening.

Respirations 36, noisy; inspiratory and expiratory sound, as heard by the bedside, husky, hoarse. Expiration much longer than inspiration; cough frequent, hoarse, croupy, singing. During inspiration there is recession of the supra-clavicular, infra-clavicular, and supra-sternal regions of the sternum and costal cartilages below the second, and of all the intercostal spaces. During expiration there is bulging of the supra-clavicular region, and prominence of the infra-clavicular and upper sternal regions. No physical signs of bronchitis. The child died the same evening; and on examination of the body, there was found, in addition to the false membrane in the larynx and trachea, vesicular emphysema of the anterior margin of both lungs, especially strongly marked in the parts overlapping the heart and great vessels, and at the apices. There was scarcely a trace of collapse of any part of the right lung, but several small patches of collapsed tissue in the inferior lobe of the left lung. The emphysema was as great of the right as of the left lung.

During the cough, which takes place in a fit of hooping-cough, the same supra-clavicular bulging, advance of the sternum, &c., as were observed in the above case, are well seen, as are, during the hoop, the recession of the supra-clavicular region, of supra-sternal region, &c.

I may sum up the position I have taken in regard of the question as to the determining cause of vesicular emphysema of the lung, thus:

1. The lung during expiration is compressed at different parts with different degrees of force.

2. The parietes of the thorax, in consequence of their

anatomical constitution, yield to the same force at different parts with various degrees of facility.

3. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and the least affected by inspiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure.

4. The power possessed by man of dilating the apex of the lung by inspiratory effort is altogether insufficient to distend the air-cells to the degree to which they are often found to be distended in local emphysema of the apex.

5. That the air-cells of the apex can be distended very considerably and with force during expiration is matter of demonstration; for in violent coughing we may see the elevation of the supra-clavicular region, the advance of the upper part of the sternum, and of the intercostal spaces generally during each expiratory succussion, the bulging parts being resonant on percussion.

6. When an impediment exists to the entrance of the air into the lungs and to its egress from the lungs, as in croup and in hooping-cough, the thoracic walls over the parts of the lungs, which after death are found emphysematous, recede during inspiration and advance during expiration.

7. In the cases last mentioned the more violent the inspiratory efforts the greater the recession of the parts of the parietes referred to, and the more violent the expiratory efforts the greater their advance.

8. When the apex is the seat of calcified or of obsolescent tubercle, lymph is exuded around and into all the tissues of the apex. Some air-cells are obliterated and the elasticity and contractility of the tissues generally is either diminished or lost.

9. Under the circumstances last specified dilatation of the unobliterated cells by the air driven into them during violent expiration with a closed glottis attains its maximum.

10. Under the same conditions in the same situation, the inspiratory force is altogether insufficient to cause dilatation of the air-cells.

11. The atmospheric air moved by the inspiratory effort can exert comparatively little pressure on the inner surface of the air-cells situated at the extreme margin of the base, the root of the lower lobe (*i. e.* that part immediately next the spine and below the primary bronchus) or at the part of the apex situated in the furrow posterior to the trachea on the right side. While violent expiration, being chiefly performed or greatly aided by the abdominal muscles forcing upwards the liver, &c., drives the air (in consequence of the highly arched form of the diaphragm in violent expiration) from the central part of the lung, not only through the bronchi towards the larynx, but also towards the circumference of the lungs, *i. e.*, towards those parts which are the least compressed during expiration.

12. In the parts just named dilatation of the air-cells is extremely common, and the dilated cells are often of large size.

13. While complete obstruction of a bronchial tube is followed by collapse of the pulmonary tissue on its distal side; partial closure of a tube has as its consequence dilatation of some of the air-cells of the part of the lung from which it leads, provided that part be unequally supported and compressed during expiration.

14. Vesicular emphysema is the invariable and necessary consequence of whatever impedes the free exit of air from the lungs, and at the same time excites powerful expiratory efforts; because by such efforts the air is driven from the more powerfully compressed parts of the lungs into those parts which are less powerfully compressed and the walls over which yield more or less readily to pressure.

Postscript.—If the view of the determining cause of emphysema advanced in this paper be correct, then in the lower animals subject to pulmonary vesicular emphysema the disease ought to be found to affect especially those parts of the lungs which in them are, during expiratory efforts with a closed glottis, the least compressed, and which are situated under those parts of the thoracic walls that are the least

capable of offering resistance to pressure. To ascertain whether it is so or not, I examined, a day or two since, with the assistance of Mr. Henderson, the veterinary surgeon of Great Scotland Yard, five horses, the lungs of which were emphysematous. In four of the five horses the anterior lobes only, and that part of the inferior lobe of the right lung which is protected by the trachea were affected; in the fifth, in addition to these parts, the margin of the inferior lobe.

Now the anterior lobe of the lung in the horse is situated above and over the heart, under the four upper ribs. It is placed, that is to say, in a part of the thorax where it can during expiration be subjected to very little external compression. So the part of the right lung, which in all five horses was emphysematous, was protected from compression by the trachea. In the lungs which were the most emphysematous there was no evidence of other disease, so far as I could judge, no trace of recent or of old collapse.

The frequency with which horses suffer from emphysema is of course attributable to their being so often compelled to make (in pulling, &c.) powerful expiratory efforts with a closed glottis. One would expect to find all animals (man among the others), engaged in occupations necessitating continued and repeated expiratory efforts with a closed glottis, more subject to pulmonary vesicular emphysema than others of the same class, *e.g.*, draught oxen rather than milch cows.

ON THE CONNEXION

BETWEEN THE

HEAT OF THE BODY

AND THE

EXCRETED AMOUNTS OF UREA, CHLORIDE OF SODIUM, AND URINARY WATER,

DURING

A FIT OF AGUE.

BY

SYDNEY RINGER, M.R.C.S.,

LATE PHYSICIAN'S ASSISTANT IN UNIVERSITY COLLEGE HOSPITAL.

COMMUNICATED BY

RICHARD QUAIN, F.R.S.

[*From Volume XLII of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

J. E. ADLARD, BARTHOLOMEW CLOSE.

1859.

ON THE CONNEXION

BETWEEN THE

HEAT OF THE BODY

AND THE

EXCRETED AMOUNTS OF UREA, CHLORIDE OF SODIUM, AND URINARY WATER,

DURING A FIT OF AGUE.[1]

BY

SYDNEY RINGER, M.R.C.S.,

LATE PHYSICIAN'S ASSISTANT IN UNIVERSITY COLLEGE HOSPITAL.

COMMUNICATED BY

RICHARD QUAIN, F.R.S.

Received June 6th—Read June 28th, 1859.

The observations on the temperature of the body during ague fits are now numerous and accordant,[2] and may be considered sufficient to indicate the general condition of the animal heat during the several stages of ague.

[1] The following observations were made at the suggestion of Dr. Parkes, from whom, from time to time, I have received assistance. Dr. Parkes has also supplied the entire literature in the paper, and has kindly examined it with care.

[2] Zimmermann; Bärensprung, Müller's 'Archiv,' 1852, p. 217; Michael, 'Archiv für Phys. Heilk.,' 1856, p. 39; Wunderlich, 'Archiv für Phys. Heilk.,' 1858, p. 12.

The increase in the amount of urea and of chloride of sodium during the cold and hot stages has been affirmed by Traube and Jochmann,[1] Moos,[2] Redenbacher,[3] and Hammond.[4] These observations have been criticised, however, and certainly some of the cases were not examined with any great minuteness. In no case yet reported either has any comparison been drawn between the rise in the temperature and the amount of the urea and chloride of sodium.[5] It therefore seemed extremely desirable, on the occasion of a patient with ague being admitted into University College Hospital, under the care of Dr. Parkes, to examine the subject, and to see, first of all, whether the increase of urea and of chloride of sodium really occur in the cold and hot stages of ague, as affirmed by Traube, Redenbacher, and Hammond; and, secondly, whether any close connexion could be traced between the amount of urea and the abnormal temperature. My position as one of the resident officers in the hospital at the time gave me the opportunity of carrying out the inquiry with all the minuteness necessary for accuracy.

The general results may be thus stated—that not only was the increase of urea and of chloride of sodium constant during the cold and hot stages of ague, but that their amount was in very close relation to the temperature. The first case recorded in this paper will, I believe, give little short of mathematical proof of the connexion between these two phenomena, viz., the increase of heat of body and of the excretion of urea and chloride of sodium.

The second case communicated is one in which the phenomena were less carefully observed, but it is valuable as affording another instance of the increase of urea and chloride of sodium during the fit.

[1] 'Deutsche Klinik,' No. 46, Nov., 1855.

[2] Henle's 'Zeitschrift für rat. Med.,' Band vii, p. 291.

[3] Henle's 'Zeitschrift,' Band ii (Dritte Reihe), p. 384.

[4] 'American Journal of Med. Science,' April, 1858.

[5] Some other observations have been made on the excretions of twenty-four hours, but these are of little value as febrile and non-febrile ~~hours~~ are put together.

In addition, a case of hectic fever occurring in phthisis has been narrated, for in this case the phenomena were found to be identical with those presented by malarial ague. The result was the same in both cases, although the causes were so different.

Case 1. *Quotidian ague.*—The patient, a man, was first attacked by tertian in August last, whilst working at Maldon, in Essex. The ague continued on him for three weeks, by which time he was apparently cured; he remained working at the same place till the December following. At that time, moving up to London, he broke his leg, and was carried to Charing Cross Hospital. Upon being discharged from that institution, cured, he caught cold, and an attack of ague immediately followed, and has continued more or less since, being immediately brought on by exposure to cold. This long continuance produced the usual effects of prolonged ague; and in this state he was admitted into the hospital, the disease having changed a day or two previously from the tertian to the quotidian type.

He suffers from slight aortic obstructive disease. His pulse is continually about 120 per minute,[1] exceedingly irregular in both force and rhythm. His arteries are very tortuous and visible. He never suffered from rheumatism, and has no arcus senilis. Except the above heart-disease, he suffers from no organic lesion.

He is fifty-nine years of age, but looks much older. His weight is 144 pounds; his height five feet nine and three quarter inches. His vital capacity (Hutchinson's spirometer) is 140 cubic inches.

The examination was conducted in the following manner. The patient was put to bed, and his urine collected through the night. At 5 a.m. he had breakfast, consisting of two eggs, bread and butter, and tea, the latter being measured. At 6 a.m. he was made to pass his urine, and all passed at

[1] No tables are therefore given of the pulse.

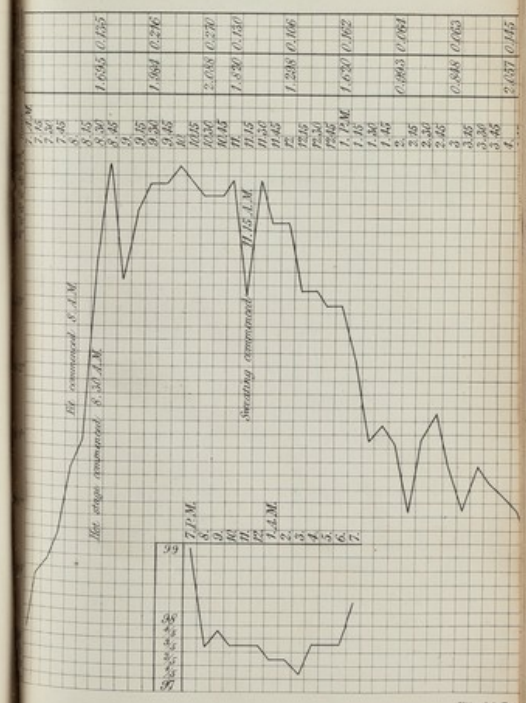
the time was mixed with that passed during the night. The thermometer (a good one, by Negretti and Zambra, and divided into fifths of a Fahrenheit degree) was next placed in his axilla, and kept there during the remainder of the day, the temperature being noted down every quarter of an hour. He was also made to pass his urine hourly, or upon the commencement of another stage, before the completion of the hour, and the next hour was dated from that time; by this means we not only obtained the urine of every hour, but also of each stage, separately. The patient was allowed no food until the completion of the fit, when he had a good meat dinner and six ounces of wine. Lemonade he was permitted to drink *ad libitum*, but the quantity was always measured and noted, with the time at which it was taken. He was weighed immediately after his breakfast, and again at the termination of the fit, and during this time no motions were allowed to be passed. The patient was purposely kept without medicine. In determining the amount of urea and chloride of sodium, Liebig's volumetric method with nitrate of mercury was used.

It was not possible to determine the other urinary constituents in this case. The chloride of sodium was not got rid of before testing for urea, but the usual correction was made.

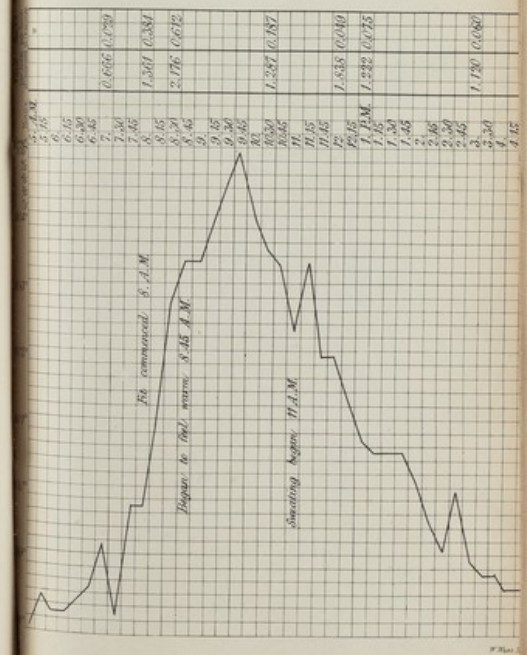
The following charts show the variations in the temperature in fifths of a degree, as taken every quarter of an hour. Above the table are two columns, one showing the amount of urea, the other the amount of chloride of sodium, poured out. The quantity per hour is stated in each case, and put down in the column denoting the time at which it was passed. It indicates, of course, the quantity formed during the previous hour. The amounts of urea and chloride of sodium are given always in French grammes, and the water in cubic centimetres. The commencement of each stage, as judged of in the usual way, by the sensations of the patient, is also noted in the column proper to the time at which it began.

For the purpose of greater clearness and of giving fuller

APRIL 6TH

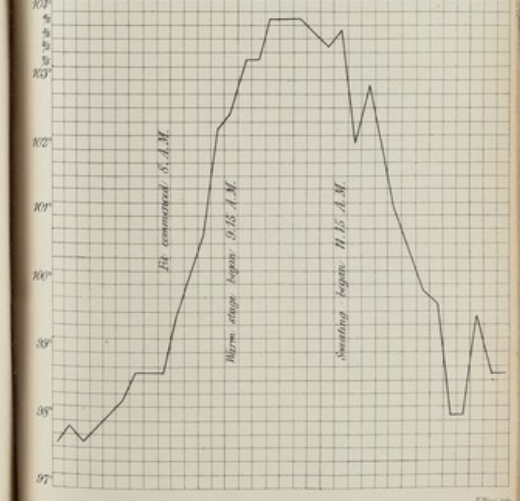


APRIL 7TH

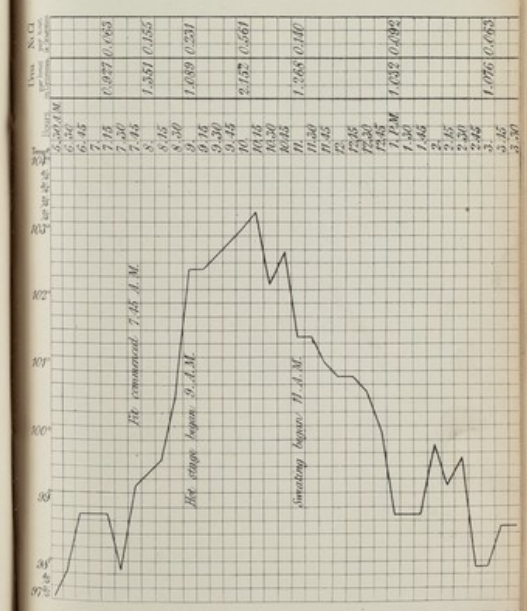

Ice commenced 8 A.M.
Began to feel warm 8.45 A.M.
Steaming began 11 A.M.

APRIL 8TH

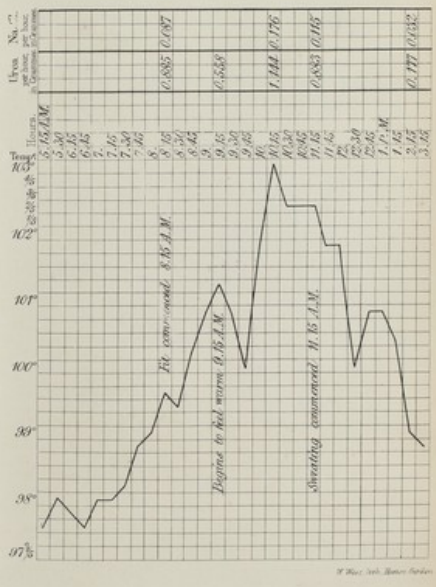
Hours	Temp.	Urea per hour	Urea per cent.	Na Cl per hour	Na Cl per cent.
5.15 A.M.					
5.30		0.664	0.021		
6.30					
6.45					
7.					
7.15		1.425	0.061		
7.30					
7.45		1.478	0.196		
8.					
8.15					
8.30					
8.45					
9.		1.936	0.369		
9.15		3.844	0.620		
9.30					
9.45					
10.					
10.15		0.684	0.030		
10.30					
10.45					
11.					
11.15		1.538	0.111		
11.30					
11.45					
P.M.					
12.15		0.442	0.019		
12.30					
12.45					
1. P.M.					
1.15		0.102	0.045		
1.30					
1.45					
2.15		1.290	0.045		
2.45					



APRIL 9th

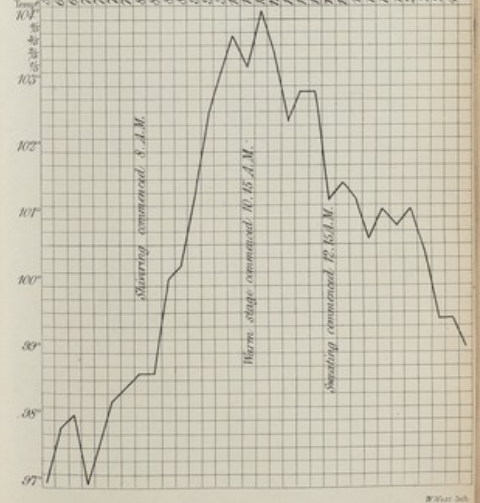

Urea N. Cl
Hours
6.30 A.M
6.45
7.15
7.30
7.45
8.15
8.30
9.15
9.30
9.45
10.
10.15
10.30
10.45
11.30
11.45
12.
12.15
12.30
12.45
1. P.M
1.30
1.45
2.1
2.15
2.30
2.45
3.15
3.30
0.937 0.165
1.351 0.155
1.069 0.231
2.152 0.561
1.265 0.140
1.032 0.092
1.076 0.163
105°
104°
103°
102°
101°
100°
99°
98°
97°
Pit commenced 7.45 A.M.
Hot stage began 9 A.M.
Sweating began 11 A.M.

APRIL 10TH


Urea No. C. Grains
0.685 0.087
0.558
1.144 0.176
0.863 0.145
0.111 0.032
Hours
Temp.
Fit commenced 8.15 A.M.
Began to feel warm 9.15 A.M.
Sweating commenced 11.15 A.M.

APRIL 11TH

Time	Urea per hour, per cent.	NaCl per hour, per cent.
5.30 A.M.		
6.30		
6.35		
7.35	0.514	0.076
7.50	1.954	0.355
7.55		
8.15		
8.30		
8.45	1.118	0.248
9.15		
9.30	1.976	0.361
10.15		
10.35	0.868	0.111
10.36		
11.30		
11.45		
P.M.		
12.53		
1.15	1.045	0.087
1.30		
1.45		
2.30	1.556	0.136
2.45		
4.	1.040	0.089



details, tables are added, recapitulating the amount of the urea, chloride of sodium, and water, separately. In the first column of each table the stages are written down; in the next the hour at which the urine was passed; then follows the total amount of urea contained in the urine passed during the specified time; the next column shows the amount reduced to hours; and, lastly, the average amount of each stage per hour is given.

Table.

April 6th.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Cold	7·45 to 8·30 a.m.	1·356	1·695	1·695
Hot	8·30 to 9·30 a.m.	1·984	1·984	1·964
	9·30 to 10·30 a.m.	2·088	2·088	
	10·30 to 11 a.m.	0·910	1·820	
Sweating	11 to 12 a.m.	1·289	1·289	1·363
	12 to 1 p.m.	1·629	1·620	
	1 to 2 p.m.	0·993	0·993	
	2 to 3 p.m.	0·848	0·848	
	3 to 4 p.m.	2·057	2·057	

CHLORIDE OF SODIUM.

Cold	7·45 to 8·30 a.m.	0·108	0·135	0·135
Hot	8·30 to 9·30 a.m.	0·216	0·216	0·220
	9·30 to 10 30 a.m.	0·270	0·270	
	10·30 to 11 a.m.	0·065	0·130	
Sweating	11 to 12 a.m.	0·106	0·106	0·108
	12 to 1 p.m.	0·162	0·162	
	1 to 2 p.m.	0·064	0·064	
	2 to 3 p.m.	0·063	0·063	
	3 to 4 p.m.	0·145	0·145	

WATER.

Cold	7 45 to 8·30 a.m.	75 c.c.
	8·30 to 9·30 a.m.	80 c.c.
Hot	9·30 to 10·30 a.m.	90 c.c.
	10·30 to 11 a.m.	82 c.c.
Sweating	11 to 12 a.m.	59 c.c.
	12 to 1 p.m.	90 c.c.
	1 to 2 p.m.	54 c.c.
	2 to 3 p.m.	53 c.c.
	3 to 4 p.m.	121 c.c.

APRIL 7th.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
During night	3 p.m. to 5·30 a.m.	10·272	0·684	0·684
Period immediately before shivering	5·30 to 7 a.m.	0·999	0·666	0·666
	7 to 8 a.m.	1·361	1·361	1·361
Cold	8 to 8·30 a.m.	1·088	2·176	2·176
Hot	8·30 to 10·30 a.m.	2·575	1·287	1·287
Sweating	11 to 12 a.m.	0·838	0·838	0·935
	12 to 1 p.m.	1·222	1·222	
	1 to 3 p.m.	2·240	1·120	
	3 to 5 p.m.	1·311	0·655	

CHLORIDE OF SODIUM.

During night	3 p.m. to 5.30 a.m.	0·642	0·029	0·029
Period before shivering	5·30 to 7 a.m.	0·440	0·29	0·29
	7 to 8 a.m.	0·384	0·384	0·384
Cold	8 to 8·30 a.m.	0·306	0·612	0·612
Hot	8·30 to 10·30 a.m.	0·375	0·187	0·187
	11 to 12 a.m.	0·049	0·049	0·064
	12 to 1 p.m.	0·075	0·075	
	1 to 3 p.m.	0·120	0·060	
	3 to 5 p.m.	0·034	0·017	

QUANTITY OF URINE PASSED.

	3 p.m. to 5·30 a.m.	36 c.c.
Beforeshivering	5·30 to 7 a.m.	25 c.c.
	7 to 8 a.m.	89 c.c.
Cold	8 to 8·30 a.m.	136 c.c.
Hot	8·30 to 10·30 a.m.	62 c.c.
	11 to 12 a.m.	33 c.c.
	12 to 1 p.m.	50 c.c.
	1 to 3 p.m.	50 c.c.
	3 to 5 p.m.	28 c.c.

APRIL 8th.—UREA.

Period immediately before shivering	5 p.m. to 6·30 a.m.	8·832	0·654	0·654
	6·30 to 7·30 a.m.	1·425	1·425	1·452
	7·30 to 8 a.m.	0·738	1·478	
Cold	8 to 9 a.m.	1·926	1·926	2·700
	9 to 9·15 a.m.	0·868	3·474	
Hot	9·15 to 10·15 a.m.	0·644	0·644	1·011
	10·15 to 11·15 a.m.	1·378	1·378	
Sweating	11·15 to 12·15 p.m.	0·044	0·044	0·498
	12·15 to 1·15 p.m.	0·102	0·102	
	1·15 to 2·45 p.m.	1·830	1·220	

CHLORIDE OF SODIUM.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Night	5 p.m. to 6·30 a.m.	0·576	0·021	0·021
Before fit	6·30 to 7·30 a.m.	0·064	0·064	0·095
	7·30 to 8 a.m.	0·063	0·162	
Cold	8 to 9 a.m.	0·369	0·369	0·381
	9 to 9·15 a.m.	0·167	0·670	
Hot	9·15 to 10·15 a.m.	0·070	0·070	0·090
	10·15 to 11·15 a.m.	0·111	0·111	
	11·15 to 12·15 p.m.	0·019	0·019	0·027
	12·15 to 1·15 p.m.	0·045	0·045	
	1·15 to 2·45 p.m.	0·045	0·030	

QUANTITY OF URINE PASSED.

Night	5 p.m. to 6·30 a.m.	42 c.c.
Before fit	6·30 to 7·30 a.m.	54 c.c.
	7·30 to 8 a.m.	60 c.c.
Cold	8 to 9 a.m.	86 c.c.
	9 to 9·15 a.m.	172 c.c.
Hot	9·15 to 10·15 a.m.	26 c.c.
	10·15 to 11·15 a.m.	52 c.c.
	11·15 to 12·15 p.m.	13 c.c.
	12·15 to 1·15 p.m.	38 c.c.
	1·15 to 2·45 p.m.	50 c.c.

APRIL 9TH.—UREA.

Immediately before shivering	6·15 to 7·15 a.m.	0·927	0·927	0·927
	7·15 to 8 a.m.	1·081	1·351	1·351
Cold	8 to 9 a.m.	1·089	1·089	1·089
Hot	9 to 10 a.m.	2·152	2·152	1·710
	10 to 11 a.m.	1·268	1·268	
Sweating	11 to 1 p.m.	2·164	1·032	1·129
	1 to 3 p.m.	2·353	1·076	

CHLORIDE OF SODIUM.

Before fit	6·15 to 7·15 a.m.	0·063	0·063	0·063
	7·15 to 8 a.m.	0·124	0·155	0·155
Cold	8 to 9 a.m.	0·231	0·231	0·231
Hot	9 to 10 a.m.	0·561	0·561	0·351
	10 to 11 a.m.	0·140	0·140	
Sweating	11 to 1 p.m.	0·184	0·092	0·078
	1 to 3 p.m.	0·127	0·063	

QUANTITY OF URINE PASSED.

Before fit	6·15 to 7·15 a.m.	35 c.c.
	7·15 to 8 a.m.	61 c.c.
Cold	8 to 9 a.m.	55 c.c.
	9 to 10 a.m.	117 c.c.
	10 to 11 a.m.	52 c.c.
	11 to 1 p.m.	44 c.c.
	1 to 3 p.m.	53 c.c.

APRIL 10TH.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Before fit	6·30 to 8·15 a.m.	1·420	0·885	0·885
Cold	8·15 to 9·15 a.m.	0·558	0·558	0·558
Hot	9·15 to 10·15 a.m.	1·144	1·144	1·013
	10·15 to 11·15 a.m.	0·883	0·883	
Sweating	11·15 to 2·15 p.m.	0·532	0·177	0·177
	CHLORIDE OF SODIUM.			
Before fit	6·30 to 8·15 a.m.	0·140	0·087	0·087
	8·15 to 9·15 a.m.	Urine lost.		
Hot	9·15 to 10·15 a.m.	0·176	0·176	0·145
	10·15 to 11·15 a.m.	0·115	0·115	
Sweating	11·15 to 2·15 p.m.	0·070	0·032	0·032
	QUANTITY OF URINE PASSED.			
Before fit	6·30 to 8·15 a.m.	50 c.c.		
Cold	8·15 to 9·15 a.m.	20 c.c.		
Hot	9·15 to 10·15 a.m.	44 c.c.		
	10·15 to 11·15 a.m.	32 c.c.		
	11·15 to 2·15 p.m.	22 c.c.		
	APRIL 11TH.—UREA.			
During night ...	2·15 p.m. to 6 a.m.	9·785	0·609	0·609
	6 to 7·30 a.m.	0·921	0·614	0·614
Immediately before fit	7·30 to 8 a.m.	0·962	1·924	1·924
Cold	8 to 9 a.m.	1·113	1·113	1·476
	9 to 10 a.m.	1·839	1·839	
Hot	10 to 11 a.m.	0·868	0·868	0·868
Sweating	11 to 1 p.m.	2·090	1·045	1·147
	1 to 2 p.m.	1·356	1·356	
	2 to 4 p.m.	2·090	1·040	
	CHLORIDE OF SODIUM.			
	2·15 p.m. to 6 a.m.	0·139	0·087	0·087
	6 to 7·30 a.m.	0·115	0·076	0·076
Before fit	7·30 to 8 a.m.	0·355	0·355	0·355
Cold	8 to 9 a.m.	0·248	0·248	0·306
	9 to 10 a.m.	0·364	0·364	
Hot	10 to 11 a.m.	0·111	0·111	0·111
Sweating	11 to 1 p.m.	0·114	0·057	0·097
	1 to 2 p.m.	0·136	0·136	
	2 to 4 p.m.	0·119	0·099	

QUANTITY OF URINE PASSED.

Stage.	Hour.	Total Quantity in this time.
Night	2·15 p.m. to 6 a.m.	...
	6 to 7·30 a.m.	24 c.c.
Before fit	7·30 to 8 a.m.	74 c.c.
Cold	8 to 9 a.m.	64 c.c.
	9 to 10 a.m.	76 c.c.
Hot	10 to 11 a.m.	31 c.c.
	11 to 1 p.m.	38 c.c.
	1 to 2 p.m.	57 c.c.
	2 to 4 p.m.	95 c.c.

CONCLUSIONS FROM THE PREVIOUS FACTS.

I. *Temperature.*

The temperature during the several fits, amid much general resemblance, presented numerous partial differences, all of which are, however, reducible to order.

(*a*) In every case, the temperature commenced to rise previous to the cold stage,[1] as experienced by the patient, that is to say, before any feeling of cold or illness of any kind. The time that it commenced, previous to the cold stage, varied from an hour and a half to three quarters of an hour, and no connexion between the time and the severity of the fit could be traced. The temperature commenced to rise—

April 6th..1½ hour before any feeling of cold or illness.
„ 7th..1 „ „
„ 8th..1½ „ „
„ 9th..1½ „ „
„ 10th..1½ „ „
„ 11th..¾ „ „

A close connexion between the severity of the fit and the character of the rise before the cold stage existed, the severity being in proportion to the continuousness of the rise, and also to the extent of each rise. As the fit became less severe, the temperature had a tendency either to oscillate, rise slowly, or remain stationary, and these tendencies

[1] The same fact is noted by Michael, op. cit., p. 43.

increased as the fit decreased in severity, and eventually all combined.

(*b*) During the cold stage the temperature rose throughout, the rise being greater than during any other period; but here also great variations existed, having their counterpart in variations in the severity of the fit.

Thus, in the cold stage, the temperature rises continuously when the fit is severe; then, as the severity lessens, there is a tendency for the rise at the termination of the cold stage to flag, and eventually to become stationary; then the range of each rise becomes less; and lastly, it oscillates. It also appears that the alteration first affects the commencement and termination of the cold stage, an oscillation in its middle indicating a greater diminution of the severity of the fit than at either end.

There was no connexion between the duration of the stage and the severity of the fit, as measured by the temperature. Thus, it lasted—

On the 6th ..... ¾ hour.
„ 7th..... ¾ „
„ 8th.....1½ „
„ 9th..... ¾ „
„ 10th.....1 „
„ 11th.....2 hours.

The fits were most severe on the 6th and 7th, and declined in severity till the 11th, when the fit was again more severe.

(*c*) During the hot stage the temperature continued to rise, and in its early part closely corresponded to the former periods.

From a careful examination of the charts, it appears that in the severer cases the temperature in the hot stage ran up at once to its acme, and had a tendency to remain permanent; the permanency not, however, being obtained at once nor retained throughout, the temperature oscillating both at the commencement and termination. In less severe cases it ran up slowly, and did not remain stationary, and when least severe, oscillated in its rise.

The temperature in those fits becoming less severe first lost its permanency, and next the temperature rose to a less extent

and again became permanent; then, when they became still less severe, it rose to the same extent, but again lost its permanency. Again, in those cases where the temperature remained permanent, the hot stage ended at the termination of the permanent period in an oscillation, these cases being the severest at their own temperature. In all other cases the temperature fell before the sweating stage commenced, ending in an oscillation, and the less severe the fit the greater was the portion of the hot stage occupied by the fall of the temperature. Also the fit was less severe, and the fall greater, when the temperature in falling every now and then stood still for some time, and was still less severe when it oscillated.

(*d*) The temperature continued to fall through the sweating stage, and was often a long time before it reached the point from which it started. The temperature fell most rapidly in those cases in which the oscillations occurred, and least rapidly where the fall was continuous. Towards the termination of the sweating stage a rise occurred to a slight extent (perhaps followed by oscillations) in four of the six fits. Thus, on April 6th, sweating commenced at 11·15, when the thermometer marked 103½°; at 2·15 the temperature had fallen to 100°; it then rose to 101° at 2·30, to 101⅘° at 2·45; then fell to 100⅗° and 100° at 3 and 3·15; then rose to 100½° at 3·30, and then finally fell regularly to 97⅘°, a point not reached till seven o'clock. The charts show, at a glance, these slight but perfectly definite rises at this late period of the sweating stage.

II. *Urea.*

As the type of the disease in this man was quotidian, there was no opportunity of comparing the amount of urea on a fever and on a fever-free *day*. But the amount of urea passed in the fever-free *hours* was decidedly much smaller than might have been anticipated. He excreted only 0·650 grm. on an average per hour in the apyrectic period, which would give in twenty-four hours 15·600 grms., had the excretion remained at the same amount during

the whole day. Now, a man of the same weight, between twenty and forty years of age, on a good diet, as this man was, would have secreted 32 grms. in the twenty-four hours, if he passed the average amount. Our patient was, however, older (fifty-nine), and would, no doubt, form less urea than a man at a more vigorous period of life. But it can hardly be supposed that the amount would be reduced so low as 15½ grms. in the twenty-four hours by this difference of age. It may, therefore, be concluded that, in accordance with Redenbacher's observations, the excretion of urea in the fever-free period was below the healthy amount.

The observations made by Traube, Redenbacher, and Hammond on the increase of urea during the cold and hot stages are entirely confirmed by this case, but a more minute statement of the kind and amount of the increase can now be given.

It must, however, be premised, that the only obvious causes of the increase of the urea in this case during the fit are, either the food taken at breakfast at 5 a.m., the fluid drunk during the fit, or the fit itself. No other known causes existed which could have had the effect of increasing the urea. The following objections to the idea of the food being the cause may be urged. That the amount of food was not great, and that the increase in the urea was far larger than has yet been noted after even the heaviest meal. For example, on the 7th, the amount per hour rose from 0·684 grm. to 2·176, being an increase of more than 200 per cent.; on the 8th, from 0·654 to 3·474, being an increase of nearly 500 per cent.; on the 9th and 10th, when the fits were slighter, the increase was less marked, though still considerable; while on the 11th, when the fit was severe, it was again 200 per cent. Such an amount is greater than has yet been found after an ordinary amount of food.[1] The time, however, at which the urea increases after food accords with our case; for, augmenting even

[1] Dr. Parkes has noted in one person without fever a rise from 0·665 grm. in a fasting hour to 1·554 grm. in a food hour, but this was after a hearty dinner.

during the first hour after food,[1] it attains its maximum sometimes in the third hour;[2] sometimes, however, not till the seventh hour; usually, however, it reaches its maximum at the fourth hour.

Now, in this case food was taken between 5 and 6 a.m., and the maximum amount of urea secreted was during the cold stage, from 8 to 9·30 a.m., or in the fourth and fifth hours. But it will probably be conceded, after it has been shown how closely the amount of the urea was associated with the variation in the temperature, that its increase in the fourth and fifth hours after food was merely a coincidence, and was not owing to the very moderate breakfast, but to the fact of the highest temperature occurring at this time. On one day, moreover, he took no food, having no appetite, and on this day the usual increase occurred. Again, after the fits were stopped by the quinine, the food was given him as usual, and the urine being collected on the same day, in the same way, comparatively little rise took place in the urea. These two last points, I conceive, set the question quite at rest.

With respect to the amount of fluid drunk, this could have no effect on the urea, as very little fluid was taken till after the time when the urea had commenced to rise. The urea, moreover, reached its maximum often at the termination of the cold stage, whilst he seldom drank anything between his breakfast and the hot stage. I believe, then, that I am justified in concluding that the rise in the amount of urea was not owing either to food or liquid. It must, therefore, have been owing to the fit.

The urea begins to increase in amount *before* the cold stage, as judged of by the first feeling of shivering, in four of the five fits. Thus, it rose—

On the 7th, from 0·666 to 1·361 per hour.
„ 8th, from 0·654 to 1·425 „
„ 9th, from 0·927 to 1·351 „
„ 11th, from 0·614 to 1·924 „

[1] Voit, quoted by Meissner, "Report on Phys. for 1857," in Henle's 'Zeitschrift,' p. 352.
[2] Becker, Henle's 'Zeitschrift,' 1855, p. 549.

The time it commenced to rise before the subjective fit varied. Thus—

On the 7th.... ¾ hour before.
„ 8th....1½ „
„ 9th....1¾ „
„ 11th.... ½ „

It often began to increase, indeed, even before the temperature began to rise. Thus, as the temperature on the 7th rose before the fit ¾ of an hour, on the 8th 1¼, on the 9th ¾, the urea on those days commenced to rise previous to any similar change in the temperature.

Great apparent irregularity existed in the rise of urea, but a close correspondence is observed between these variations and similar ones in the temperature.

The alteration in the above case of urea and the temperature did not always exactly agree in time, though this was the rule, but sometimes the alteration in the temperature did not occur till after the alteration in the urea; the reverse never happened.

The quantity of urea continued to increase, and reached its highest point either at the termination of the cold stage, as on the 8th and 11th, or at the commencement of the hot, as on the 6th, 9th, 10th, and 12th. It then began to fall in quantity, slowly at first. On the 6th and 10th the temperature and urea commenced to fall simultaneously. On the 7th, 8th, 9th, and 11th, the temperature continued to rise, notwithstanding the fall in the urea. Up to the period when the urea commenced to decrease, the temperature rose rapidly, each rise being extensive, but after that, at the point where the urea commenced to fall, the temperature either oscillated, or every rise remained stationary for a short time, the rise being always slow and by small additions.

The urea continued to fall slowly from the end of the cold, or from the commencement of the hot stage, till the sweating stage began; and the temperature corresponding in time to the latter part of this slow decrease, after reaching its height, fell also slowly and slightly.

From the following table it appears that no close correspondence existed between the fall in the temperature towards the close of the hot stage and the decrease in the amount of urea.

Date.	Fall in Urea.	Fall in Temperature in same time.	Temperature oscillated.	Tendency to be stationary.	Temperature fell continuously.	Fall of Urea reduced to quantity for 1° Fahr.
6th	0·268	1°		—		1·346
7th	0·830	1½°			—	0·533
8th	1·452	½°			—	...
9th	0·884	1°			—	0·884
10th	0·261	½°		—		0·435
11th	1·000	2½°	—			0·357

In the first column the date is given; in the second the fall in the amount of urea, corresponding in time to the fall in the temperature, from its highest point to the commencement of the sweating stage, is given; next, the number of degrees the temperature fell in the same time; then, in three columns, the character of the fall is given; and in the last column the fall in the amount of urea is reduced to the quantity corresponding to each degree. But though there is no intimate connexion, still the greater the fall in the temperature the greater is the decrease in the quantity of urea. At the same time the character of the fall varied; thus, sometimes the temperature showed a tendency to remain stationary, then the urea fell but little, as on the 6th and 10th. On other days the temperature fell gradually, and then the fall in urea rather increased. The greatest fall occurred on those days when the temperature oscillated greatly. Thus, the least decrease in urea corresponded to that temperature which has a tendency to remain stationary, more when it fell slowly but continuously, and much the most when it oscillated, as on the 11th; and it is

possible to judge of the rapidity of the decrease of the urea by the character of the temperature.

The temperature has been shown to fall either at the close of the hot or the commencement of the sweating stage. A similar fall occurred in the urea, and the amount of the decrease of urea corresponded to the length of the oscillation, and the subsequent rise in the urea corresponded to the rise in the oscillation, though the whole oscillation always occurred in an hour previous to that in which the subsequent rise in the urea occurred.

Date.	Fall in Temperature.	Fall in Urea.	Rise in Temperature.	Rise in Urea.
6th	1f°	1·820 to 1·289	1f°	1·289 to 1·620
7th	1°	1·287 to 0·838	1°	0·838 to 1·222
8th	1f°	3·474 to 0·644	f°	0·644 to 1·378
11th	1f°	1·839 to 1·045	f°	1·045 to 1·356

A slight rise, however, took place at the same period, even when the temperature remained permanent, but then the increase was small in amount.

On the 9th, the temperature fell, but did not oscillate; the fall in the urea was not so great as when the temperature oscillated.

A relationship in the latter part of the sweating stage between the variation in the urea and temperature existed, corresponding to what is stated above. Thus, on the 6th, after the usual oscillation, the temperature had a tendency to fall, and then to remain stationary for some time, and again to fall and remain a second time stationary. During this time the urea fell in quantity slowly. The temperature then took a great fall, and oscillated greatly, and at the same time the fall in urea was very great.

On the 7th the temperature fell slowly, and often remained stationary; at the same time the urea fell slowly, and, like the temperature, did not reach the normal amount till late in the afternoon.

The same close correspondence exists between the other cases.

In order to test still further the relationship between the urea and the temperature, the following method was adopted. The total amounts of urea excreted during the fit, the quantity excreted during the rise and fall of temperature, during the period immediately before the fit, and during the cold and hot stages, respectively, have been added up and divided by the number of degrees the temperature rose or fell in the corresponding periods. By this means the amounts of the urea secreted during the whole and each of the divisions of the fit were reduced to a common standard, and could thus be compared more easily with one another.

Table[1] *to show the amount of Urea corresponding to each degree of Fahrenheit of abnormal heat.*

Date.	Entire fit. The amount of urea for each degree of abnormal heat.	Rise in the temperature, that is, amount of urea to each degree.	Fall in the temperature and the urea to each degree.	The quantity of urea to each degree of the urea before the fit.	The same in the cold stage.	The same in the hot stage.
6th	1·557	0·626	0·930	...	0·339	3·560
7th	1·413	0·615	0·798	0·972	0·391	1·609
8th	1·508	0·903	0·722	2·163	0·735	1·444
9th	1·460	0·675	0·710	0·566	0·246	2·793
0th	1·298	0·718	0·746	0·657	0·617	1·381
11th	1·781	0·712	1·280	0·601	0·590	0·868

[1] In this table each of the quantities of urea correspond to 1° of temperature. In the first column the date is given; in the second, the whole amount of urea passed during the entire fit (the fit being considered to

On comparing the different days, the closest correspondence existed when the whole fit was taken. The extreme in column two being taken, the difference only amounted to one fifth of a degree. The rise and fall also closely corresponded. The extreme again being taken, a difference amounting to one third of a degree was found.

As the duration of the fit might have varied greatly, and so the above mode of comparison of the different days one with the other have been invalidated, the following tables have been worked out.

The first shows the duration of the fit on each day.

6th	.....	$7\frac{1}{4}$ hours' duration.
7th	.....	8 "
8th	.....	$8\frac{1}{2}$ "
9th	.....	$7\frac{3}{4}$ "
10th	.....	$8\frac{1}{2}$ "
11th	.....	$7\frac{1}{2}$ "

commence with the rise of the temperature and the increase of the urea and chloride of sodium). In the third column the rise (in the temperature) only is taken, and when the temperature remained stationary at its highest point for some time the time of the rise is calculated up to the middle of the stationary part. In the next column the fall, calculated in the same way, is given. Then the cold and hot stages are given separately.

The amount of urea varied in this patient; for the night urine varied on one day greatly from the quantity passed during a similar period on other nights. This day, the 9th (the urine of the night before not having been saved), the amount of urea corresponding to each degree was greatly in excess; but on assuming that the quantity passed during the first hour 0·900 grm.) was the quantity normal to him for that day, and so deducting the excess, a close correspondence to the other days resulted. Thus, when the whole amount of urea was calculated as it stood, to each degree 2·043 grms. were found to correspond; but when the additional 300 grms. were deducted, then the quantity per degree was 1·460; and throughout all the different comparisons it will be then found closely to agree. On the 10th, again, the normal excretion was below par (0·170 grm. per hour), which also prevented any comparisons from being made. When this was raised to 0·600 grm., then a much closer correspondence to the other days was found.

Another calculation must now be made. On three days the urine was collected through the previous night; taking that hourly amount, and assuming that to be the amount normal to the man through that day, if there had been no disturbing influences, and deducting that quantity from the quantity passed hourly through the fit, the increase only is obtained, and this divided by the number of degrees the temperature rose, the comparison is then found to be very close.

April 7th	.....	0·638	urea to each degree of abnormal heat.
" 8th	.....	0·618	" "
" 11th	.....	0·624	" "

If this table be compared with the former one, the comparison will be found to be even much closer. Unfortunately, the amount during the night was not determined except on these three days, so that this mode of calculation is not applicable to all the fits.

On looking at the second column of the table given at page 377, where the total amounts are compared, it is found that on the 6th, 8th, and 11th, the quantity in excess is somewhat over other days. The temperature on the 6th and 8th remained permanent after reaching its highest point. Thus, on the 6th it remained at 105° for two hours and a half; on the 8th at $103\frac{5}{8}$° for one hour and a quarter; but when the amount passed during these permanent periods is deducted, then the urea on the 6th fell to 1·277 grm., and on the 8th to 1·366 grm., bringing each on a level with the 10th. On the 11th, the increase in urea occurred entirely in the fourth column, that is, during the fall of the temperature, the amount passed during the rise corresponding closely to the other days; and on examining the sweating stage on the 11th, there is found a great and sudden increase at its very termination, and this excessive quantity being replaced by the quantity passed on other days at the same period, the amount corresponding to each degree was 1·510, closely coinciding with the other days.

That the increase on the 6th and 8th was due to the above is rendered probable by the following statement: The whole quantity of urea passed on the 6th, during the stationary period of two hours and a half, was 1·957 grm. On the 8th, for one hour and a quarter, the quantity was 1·155. When compared to equal time, that is, the two hours and a half reduced to one hour and a quarter, then, on the 6th, the amount of urea was 0·978, corresponding closely to the quantity passed in the same time on the 8th. From this it would appear that the same amount of urea corresponds to 1°, when the temperature is permanent, whether it be at a high temperature or not, whilst the second table shows that *every degree in the hot stage corresponds to a much greater amount of urea than in the cold or any other stage, whilst the temperature is rising;* thus showing, that the higher the temperature, the more urea corresponds with each rise of a degree than formerly, and thus accounting in some measure for the greater quantity of urea to each degree, when the whole fit is compared, in the more severe fits.

The very close correspondence between the temperature and the urea seems to be thus placed beyond doubt; in fact, the one may be calculated from the other. An example or two will illustrate this.

Thus, on—

April 6th, the amount of urea excreted during the fit was 11·097 grammes; taking 1·503 gramme as the average quantity (all the degrees being taken) corresponding to each degree, this gives a rise of 7°, or a little over; the actual rise was from 98½ to 105, or 6½°.

April 7th, the total amount of urea during the fit was 10·635 grammes. The rise in the temperature here should be 7°; the actual rise was 6½°.

April 8th, the total amount of urea excreted was 9·353 grammes, which would imply a rise in the temperature of 6°; the actual rise was from 97½ to 103½, being exactly 6°.

III. *Chloride of Sodium.*

The chloride of sodium, to a very great extent, agreed with the urea in its relationship to the temperature. Traube's statement on this point is therefore confirmed. For the most part, the remarks above made on the urea hold good for the chloride of sodium; still some differences occurred.

On the 8th, the urea between 7·30 and 8 a.m. remained stationary, whilst the chloride of sodium rose continuously.

9th.—Between 8 and 9 a.m. the urea fell, the temperature corresponding, but the chloride of sodium rose to double its quantity.

Again, during the decline of the fit, they differed on the 8th and 9th; the urea at its termination rose somewhat, whilst the chloride of sodium continued to fall.

The following table corresponds to one given for the urea, and shows the intimate connexion between the severity of the fit and the amount of chloride.

Date.	Entire fit. The amount of sodium chloride for each degree.	Rise in the temperature taken, and the amount of chloride to each degree.	Fall in the temperature taken, and the amount of chloride to each degree.	The quantity of chloride to each degree of the temperature during the cold stage.	The same during the hot stage.	The same during the period before the cold stage.
6th	0·195	0·085	0·086	0·027	0·293	...
7th	0·225	0·159	0·066	0·085	0·259	0·640
8th	0·171	0·107	0·078	0·131	0·162	0·134
9th	0·241	0·150	0·062	0·175	0·702	0·163
10th			Lost.			
11th	0·180	0·149	0·041	0·102	0·176	0·785

Here, as occurred in the urea, the patient on two days

passed a different quantity normally from what he had on former days; so that on one day, the 8th, it was necessary to add 0·022 grm. to each hour to bring it up to the other days. On the 11th it was necessary to subtract 0·044 grm.

On the 6th the urine was not collected before the commencement of the cold stage, so that the amount for each degree is less than it would otherwise have been in the third and fourth columns.

When the extremes in the third column were compared, a difference corresponding to one third of a degree of temperature occurred. When the fourth column was compared, excluding the 6th, a difference of one third of a degree occurred. In the fifth column, a difference corresponding to less than one third occurred, excluding the 11th, on which day the chloride fell to its normal amount before the fit had ended.

In the cold stage an enormous difference was found.

In the hot the difference corresponded to half of a degree.

Beyond these above differences, all that has been said regarding the urea applies equally to the chloride of sodium.

IV. *Water of the Urine.*

In rising and falling in quantity, the water has a very close correspondence to the urea, though there is very little proportion between the different rises and falls in the two. On the 6th, 8th, and 9th, they corresponded in rising and falling at the same periods. On 11th and 7th the quantity of urine in the hot stage falls so considerably, that in the commencement of the sweating stage a slight rise occurs, this being followed, however, by a decided fall during the second hour.

The quantity of urine thus corresponding to the urea, must, like it, correspond somewhat to the variations in the temperature. That the quantity stands in close relation to the intensity of the fit is seen from the following table.

Date.[1]	Quantity of urine in the fit.	Number of degrees temperature rose.	Quantity of urine to each degree.
6th	608 c.c.	5½	117 c.c.
7th	411 c.c.	5¾	71 c.c.
8th	449 c.c.	6¼	72 c.c.
9th	382 c.c.	5¾	71 c.c.
11th	435 c.c.	5¾	75 c.c.

It has been shown that some correspondence exists between the oscillation of temperature at the commencement of the sweating stage and the fall and subsequent rise in the amount of urea. The following table shows the relationship between the same oscillation and the fall in the amount of urine passed.

On the 6th and 11th it has been said that the amount of urine secreted was so small during the hot stage that it rose somewhat at the commencement of the sweating, so that no comparison can be given on those days between the fall of the oscillation and the quantity of urine. In the next table, in the first column, as usual, the day of the month is given; in the second, the fall of the temperature in the oscillation; in the third, the fall in the amount of urine; in the fourth, the amount reduced to a degree; in the fifth column, the rise of the temperature in the oscillation is given; in the sixth, the rise in the amount of urine in the hour subsequent; and in the seventh, the quantity is reduced to that corresponding to a degree:

[1] In the first column the date is given. In the second, the total quantity of urine passed during the fit. In the third, the number of degrees the temperature rose during the fit. In the fourth, the number of cubic centimetres corresponding to each degree. Excluding the first day, a very close correspondence existed.

6th	Urine rose		...	1½°	0·030	0·020
7th	1°	0·030	0·030	1°	0·017	0·017
8th	1½°	0·039	0·025	½°	0·025	0·030
11th	Urine rose		...	½°	0·007	0·035

From this limited table, no great correspondence can be traced.

But though the urea, chloride of sodium, and water, thus constantly show a close correspondence to the temperature, the relative amount of rise was different in these three ingredients.

This indeed was shown well during the analysis, in which 10 c.c. of urine were taken; the amount of mercury solution required for the amount of urea varied greatly from hour to hour, showing no regularity of rise; whilst, on the other hand, the amount required in testing for the chloride gradually rose and then gradually sank, often again rising somewhat at the very close of the fit. Thus, to take the 7th of April:

UREA.

Hour.	Amount of mercury solution required for 100 pints of urine.	Same for NaCl.	Hourly amount of water passed.
7 a.m.	298 c.c.	48 c.c.	25 c.c.
8 a.m.	198 c.c.	144 c.c.	80 c.c.
8·30 a.m.	188 c.c.	135 c.c.	136 c.c.
10·30 a.m.	224 c.c.	90 c.c.	62 c.c.
12 noon.	272 c.c.	45 c.c.	33 c.c.
1 p.m.	268 c.c.	45 c.c.	50 c.c.
3 p.m.	248 c.c.	36 c.c.	50 c.c.
5 p.m.	258 c.c.	18 c.c.	28 c.c.

The urea varies in its proportion to the water; thus, during excessive diuresis, the per-centage amount falls, whilst, on the other hand, the per-centage amount of chloride even then increases, up to the time the greatest amount is poured out, then it as steadily falls, even when the water fluctuates greatly. These remarks apply to the per-centage of chloride of sodium in the urine.

It appears that the amount of urea undergoes an increase definite in amount, independent of the water. The chloride of sodium also undergoes a definite increase, which, also, is independent of the amount of water; but the water being increased, the same per-centage of chloride is poured out as would have been the case if a smaller amount of water had been voided, the per-centage not being lowered by an excess of water, as is the case with urea.

The chloride has thus a tendency to rise and fall steadily, not observing the various alterations corresponding to temperature that the urea does, but the water, corresponding to the urea in this respect, causes variations of the same character in the total amount of chloride poured out.

The time of greatest per-centage excretion of chloride of sodium does not always correspond to the greatest hourly excretion.

Thus, on April 6th, the greatest amount of saturation was at 8 a.m., whilst the greatest hourly excretion was at 8·30, when the water was at its greatest amount.

7th.—The hourly excretion and the per-centage amount agreed in the time at which they occurred, and so also did the water.

8th.—The same occurred on this day.

9th and 10th.—The same occurred also on these days.

11th.—The per-centage amount of chloride was greatest at 9 a.m., whilst the hourly amount excreted was greatest at 10, the water being most abundant during the last period.

The urea, on the other hand, often decreased in per-centage during the fit, especially if the increase in the water was great, its highest per-centage amount corresponding to the lesser amount of water. Thus, on April 6th, the per-centage amount decreased through the entire fit. The decrease was, however, much more gradual at its commencement.

7th.—The greatest per-centage amount was at 7 a.m., the amount of water being 25 c.c. for the hour. The water

then rose to 80 c.c., and the per-centage amount of urea fell; the amount of mercury solution required for 10 c.c. of urine fell from 29 c.c. to 19 c.c. The water then again rose to 136 c.c., and the solution of mercury required fell in amount to 18 c.c. The water next fell to 62 c.c., and the solution of mercury rose to 22·6 c.c. The water again fell to 33 c.c., and the amount of the solution required rose to 27·4 c.c. The water next rose to 50 c.c., and the solution required fell to 26·4 c.c. The water then remained at 50 c.c., but the solution fell to 24 c.c. Then the water fell to 28 c.c., and the quantity of the solution rose to 25 c.c.[1]

From these facts it is evident that both the urea and chloride increase, independent of the influence of the amount of water; that any increase in the latter does not modify the aggregate amount of urea, but that it does that of the chloride.

No connexion exists between the quantity of urine passed during the entire fit and the amount of water drunk.

This is seen in the following table:[2]

Date.	Water drunk.	Urine passed.
6th	2135 c.c.	648 c.c.
7th.	1335 „	550 „
8th	1050 „	428 „
9th	500 „	499 „

The urine was passed much more equally as regards the different periods than the water drunk, for the entire quantity was mostly drunk during the hot stage only.

Thus, the urine, urea, and chloride of sodium, were independent, to a large extent, of the quantity drunk,

[1] Every c.c. of the mercury solution corresponds to 10 milligrammes of urea.

[2] As usual, the date is given in the first column; in the second, the quantity of water drunk during the entire fit, and in the third, the amount of urine passed during the entire fit, are given.

though some connexion appeared to exist, as, when the patient drank copiously, the next time the urine was collected it was found to be somewhat in excess; but this might be a mere coincidence.

TABLE.

APRIL 12.—UREA.

Stage.	Hour.	Total Quantity in this time.	Quantity per hour.	Average Quantity per hour during period.
		Grammes.	Grammes.	Grammes.
Before fit	6·15 to 7·15	1·868	1·868	1·630
	7·15 to 8·15	1·815	1·815	
	8·15 to 8·45	0·604	1·209	
Cold	8·45 to 9·45	0·530	0·530	2·310
	9·45 to 10	0·445	1·780	
Hot	10 to 11	2·287	2·287	2·219
	11 to 11·30	1·075	2·150	
Sweating	11·30 to 12·30	0·756	0·756	0·442
	12·30 to 3·30	0·384	0·128	
	CHLORIDE OF SODIUM.			
Before fit	6·15 to 7·15	0·153	0·153	0·153
	7·15 to 8·15	0·225	0·225	0·225
	8·15 to 8·45	0·340	1·080	1·080
Cold	8·45 to 9·45	0·240	0·240	0·321
	9·45 to 10	0·100	0·403	
Hot	10 to 11	0·625	0·625	0·467
	11 to 11·30	0·154	0·309	
	11·30 to 12·30	0·090	0·090	0·173
	12·30 to 3·30	0·708	0·256	
	QUANTITY OF URINE PASSED.			
Before fit	6 to 7·15	73 c.c.		
	7·15 to 8·15	75 c.c.		
	8·15 to 8·45	54 c.c.		
Cold	8·45 to 9·45	25 c.c.		
	9·45 to 10	84 c.c.		
Hot	10 to 11	125 c.c.		
	11 to 11·30	86 c.c.		
	11·30 to 12·30	32 c.c.		
	12·30 to 3·30	128 c.c.		

On the 12th, everything being conducted in the same manner, upon the temperature commencing to rise, the patient was given ℈j of quinine, which caused the tempera-

ture to fall again. It continued to fall for half an hour, and then began to rise; the cold stage came on an hour later than on the previous day. The fit, however, was afterwards as severe as usual. The effects on the urea corresponding to this fall cannot be ascertained, as, for some reason, the amount secreted was three times as great as on previous days, for the first two hours it was collected; it then fell somewhat, but continued high throughout the fit, the quantity to each degree being 0·980 grammes.

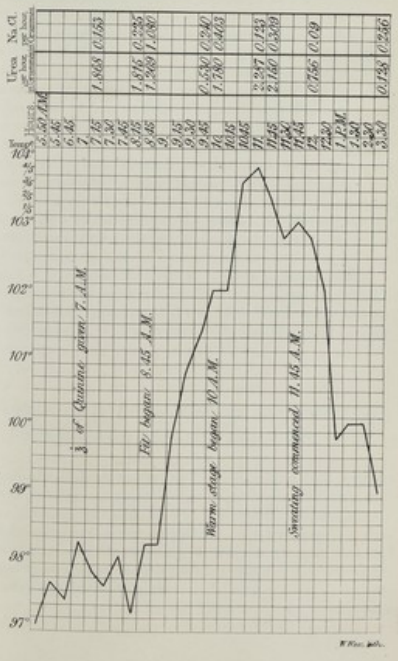
The temperature obeyed all the rules laid down previously—rising to the commencement of the cold stage; then remaining permanent for half an hour; then rising through the whole of the cold stage, the latter being shorter than the former; and just at the commencement of the hot stage, it again remained stationary for a quarter of an hour; then rose, but began to fall before the sweating stage commenced, and at that point oscillated, the oscillation being small; it then fell rapidly.

The urea corresponded also to these variations, after the manner described. Thus, it fell where the temperature remained stationary, but the fall was out of proportion to the alteration in the temperature, being very great. Possibly, this was due to the quinine. It then rose rapidly, reaching its highest point at the same time as the temperature. It fell slowly during the latter part of the hot stage, then fell greatly during the oscillation at the commencement of the sweating stage; but as the urea did not subsequently rise, the rise in the oscillation of the temperature was only one fifth; then the urea fell greatly.

The chloride of sodium differed somewhat from the urea at the commencement and termination. Thus, though an unusual quantity was passed in the first two hours, like the urea, still it did not fall when the temperature began to rise a second time, but continued rising throughout; just before the cold stage it reached its highest point, arriving at the very unusual quantity of 1·080 grm.

APRIL 12TH

Phenomena of a Fit modified by Quinine.



After this it corresponded in its variations to the urea, except in the second hour of the sweating stage, when, instead of continuing to fall, it rose considerably. The rise in the hour subsequent to the oscillation in the temperature was very extensive.

Considering the fit to have commenced on this day, at 7·15 a.m., the amount for each degree was 0·396, a larger quantity (as is also the case with the urea) than occurred on other days; but as the amount passed for some hours before the fit was unusually large, it is probable that the quantity normal for that day was in excess.

The water in its variations corresponded to the urea, except at its very termination (that is to say, the second hour of the sweating stage), when it rose considerably.

The amount for each degree was 105 c.c. It is rendered most highly probable that the reason for the amount for each degree being greater than usual in urea, salt, and water, is, that the normal quantity was in excess on this day, for it is found that the increase in each is proportionate.

The quantity he drank was not taken down on this day. Thus the only influence that can be ascribed to the quinine is the lowering of the temperature and the postponement of the fit.

Examination of the Urine on April 13th, ℈ij *of Quinine having been taken the day before, the fit being entirely absent.*

On the 13th no fit occurred. The patient took, without leave, another scruple of quinine the evening before. The temperature remained at 97° the entire day, with very slight variations, never amounting to more than $\frac{1}{5}$°. But, notwithstanding this equality of temperature, the urea and chloride of sodium underwent an increase. The urine was collected through the night.

UREA.		CHLORIDE OF SODIUM.	
Hour.	Quantity per hour.	Hour.	Quantity per hour.
	Grammes.		Grammes.
3·30 p.m. to 6·30 a.m.	0·610	3·30 p.m. to 6·30 a.m.	0·057
6·30 to 7·30 a.m.	1·340	6·30 to 7·30 a.m.	0·060
7·30 to 8·30 a.m.	1·122	7·30 to 8·30 a.m.	0·082
8·30 to 9·30 a.m.	1·150	8·30 to 9·30 a.m.	0·096
9·30 to 10·30 a.m.	1·412	9·30 to 10·30 a.m.	0·138
10·30 to 11·30 a.m.	0·803	10·30 to 11·30 a.m.	0·086
11·30 to 12·30 p.m.	0·999	11·30 to 12·30 p.m.	0·091
12·30 to 1·30 p.m.	0·606	12·30 to 1·30 p.m.	0·027

Thus, though the patient experienced no change as regards his sensations, and his temperature remained the same throughout the day, *a similar change to that occurring on the previous days was observed in the urea and chloride of sodium*. The fit, as judged by the urine, commenced at 6·30 a.m. The urea then fell somewhat during the next hour, rising again steadily up to 10·30 a.m., when it had reached its highest point. It then fell greatly, and this was followed by a slight rise, and then again fell to the amount normal to this man. The fall, immediately after the first rise, probably corresponded to the termination of the period prior to the cold or the commencement of the cold stage; and had the temperature risen, it would probably at this point have either oscillated or remained permanent for some time.

The next great fall, at 11·30 a.m., corresponded to the sweating stage, and would have been marked in the temperature by a great fall, and then, probably, the temperature would have remained stationary for half an hour, and not have risen, as the subsequent rise in the urea is small in amount. The urea after this small rise again falls to the normal amount.

The chloride also strictly followed the principles laid down, for, as has been shown, it has a tendency to rise continuously, an equal amount of urine being taken each hour, though, if the urine be much altered in

quantity, this salt also falls and rises. Thus, the chloride rises continuously, and is marked by no fall after the first hour, but, corresponding to the great fall of urea at 11·30, it also falls and again rises slightly in the next hour, and then again falls to the amount normal.

The urea and chloride increase *pari passu*, and each reaches its height at the same hour.

The amount of urine, again, corresponded to the urea and chloride of sodium; it fell slightly after the first hour, then rose, reaching its highest point at the same time as the urea; it then fell, but again rose somewhat, and then permanently fell.

3·30 p.m. to 6·30 a.m. ..... 40 c.c.
6·30 a.m. to 7·30 „ ..... 50 „
7·30 „ 8·30 „ ..... 46 „
8·30 „ 9·30 „ ..... 46 „
9·30 „ 10·30 „ ..... 66 „
10·30 „ 11·30 „ ..... 41 „
11·30 „ 12·30 p.m. ..... 51 „
12·30 p.m. to 1·30 „ ..... 30 „

Taking the mean quantity of grammes corresponding to a single degree on other days, the increase of urea would correspond to a rise of 5½° in temperature. Thus, as far as the urea, &c., are concerned, the fit was as severe as on previous days.

The urine was again collected hourly during the 14th.

Here the urea and chloride of sodium were again in great excess during the whole day, and also during the night previous.

Hour.	Urea per hour.	Chloride per hour.	Water per hour.
1·30 p.m. to 6 a.m.	1·620	0·168	85 c.c.
6 to 7 a.m.	2·715	0·585	150 c.c.
7 to 8 a.m.	1·332	0·405	90 c.c.
8 to 9 a.m.	2·545	0·877	172 c.c.
9 to 10·30 a.m.	1·106	0·329	67 c.c.
10·30 to 11·30 a.m.	1·107	0·421	78 c.c.
11·30 a.m. to 1·30 p.m.	1·118	0·312	60 c.c.
1·30 to 3·30 p.m.	1·180	0·254	77 c.c.

On the second and fourth hours the urea rose to nearly double the quantity of other hours. After the second rise it remained stationary for two hours, and then rose. This corresponds to some extent with the urea the day before; but after the second rise the fall was not so great, and the subsequent rise was very small in amount.

The chloride more closely resembled the day before. Like the urea of the same day, it rose twice, but the last rise exceeded the first, and was followed by a considerable fall, and this again by a rise of some extent, and then it again fell. The second rise and fall corresponding to the commencement of the sweating stage, the first fall probably would have corresponded (had the temperature risen) to an oscillation or a permanency at the termination of the period prior to the cold stage.

The water closely corresponded to the chloride.

Detracting 1·000 gramme from each hour, that being the necessary quantity to reduce the urea in the night urine to the amount normal to this man, and again, as on the day before, calculating what height the temperature would have reached had it risen, it is found to be 3°.

This fit was therefore less severe than that on the previous day. It thus appears that *variations in the urea and chloride of sodium continue to occur at those periods when, if the fit had continued, the temperature would have risen.* The same fact has been noted by Redenbacher; and it would show that the cure of the fit by quinine, in this man, was followed by a much larger excretion of urea during what would have been the apyrectic hours than had been noted at the corresponding time when he was suffering from the disease.

Case 2.—*Tertian ague.*—The following case of ague occurred in a boy, æt. 19, strong, and in every respect healthy with exception of the attack of ague. The boy had not been out of London for twelve months before, but at that time came over from Dantzig, having been there for some months.

He was admitted into the hospital with the second fit, which was very slightly marked, the fit increasing subsequently in severity till he took four grains of quinine every two hours, which prevented its continuance. It was tertian in type.

Everything was conducted exactly after the manner of the former case. The urine, however, was collected (on one day only) by the stage, and not by the hour. His pulse was taken each quarter of an hour, with the temperature.

The two following charts of the temperature will be found to correspond closely with those given before, the correspondence holding mostly with the severer fits. In the first, the temperature rises slowly up to the cold stage; it then rises rapidly, running up continuously to the commencement of the hot stage, at which point it remains stationary for half an hour; it then runs up to its highest point, remains stationary for a short time, and sinks till the sweating stage commences, when, instead of oscillating, it remains stationary for half an hour, then falls and oscillates, but remains high even at 9 p.m., thus differing from the former and subsequent tables.

This further illustrates the slow rise of the stage previous to the cold one; then its rapid rise, the rise being here also greater than at any other period; also its tendency to be affected first at either termination as the fit becomes less severe. It shows that the temperature falls before the commencement of the sweating stage, and that at this point it either oscillates or remains stationary; and in the severe fit, that the temperature has a tendency to remain stationary at its highest point.

The next chart (that of April 4th) shows the temperature on the day the fit was severer, and consequently we find indications of this in the character and fall of the temperature. It shows at the commencement of the cold stage a tendency to be stationary, but after this it runs up continuously with no permanent period at the termination

of the cold stage, though an approximation is seen in the rise at this point being less than the previous ones. It then remains permanent for some time, and then very slowly sinks, remaining permanent for half an hour just where the sweating commences; the temperature here also falling before the sweating stage commenced. Thus, the continuous rise, the permanency when at its height, and the slow and gradual fall, all indicate the severity of the fit, besides the great height to which the temperature reaches.

The urea, here only estimated for each stage, corresponds to the results before stated. Taking the average amount per hour in the sweating stage as the nearest approach to the quantity normal to the boy, it is found that there is an increase in the period previous to the fit; this continues increasing, is highest during the cold stage, then falls somewhat in the hot, and again falls still lower in the sweating.[1]

The same is exactly the case with the chloride of sodium and water.[2]

The patient drank—

At breakfast	285 c.c.
Before cold stage	200 „
Cold stage	200 „
Hot stage	700 „
Sweating stage	500 „

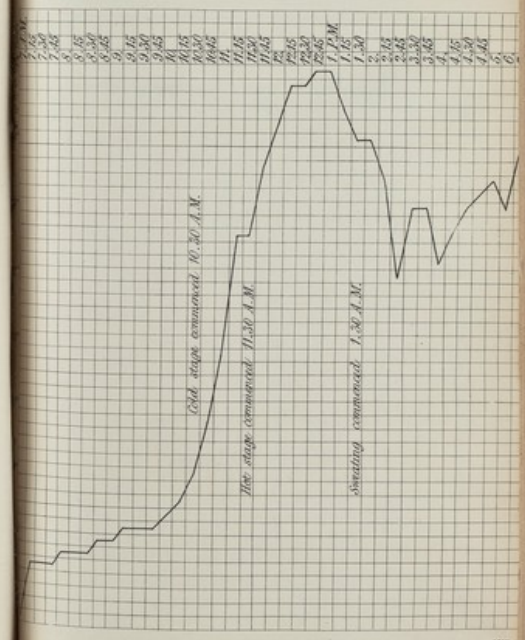
He passed the following quantity of urine:

Before fit (3 hours)	162 c.c.	= 54 c.c.	per hour.
Cold stage (1 hour)	100 „	= 100	„
Hot stage (2 hours)	180 „	= 90	„
Sweating stage (4 hours)	150 „	= 37½	„

[1] The quantity of urea to each degree was 1·977 grm., showing a close correspondence to the amount for each degree in the former case. The correspondence would probably be closer still if the normal amount of this patient was reduced to that of the former.

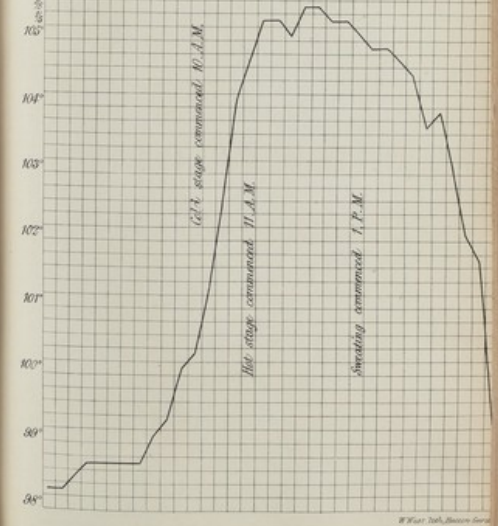
[2] The amount of chloride of sodium corresponding to each degree was 0·502.

APRIL 2ND



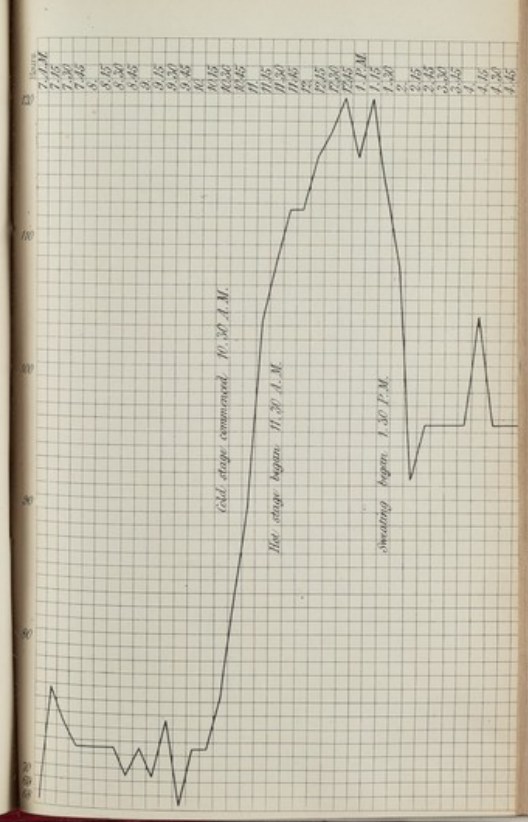
APRIL 4TH

Time	Temp.	Urea per hour	Urea per minute	Na. Cl per hour	Na. Cl per minute
7 P.M.					
7.15					
7.30					
7.45					
8.15					
8.30					
8.45					
9.15					
9.30					
10.15		1.344	0.934		
10.30					
10.45					
11.		2.340	1.090		
11.15					
11.30					
11.45					
12.					
12.15					
12.30					
12.45					
1. P.M.		1.710	0.972		
1.15					
1.30					
1.45					
2.					
2.15					
2.30					
2.45					
3.15					
4.		1.087	0.840		



APRIL 2ND

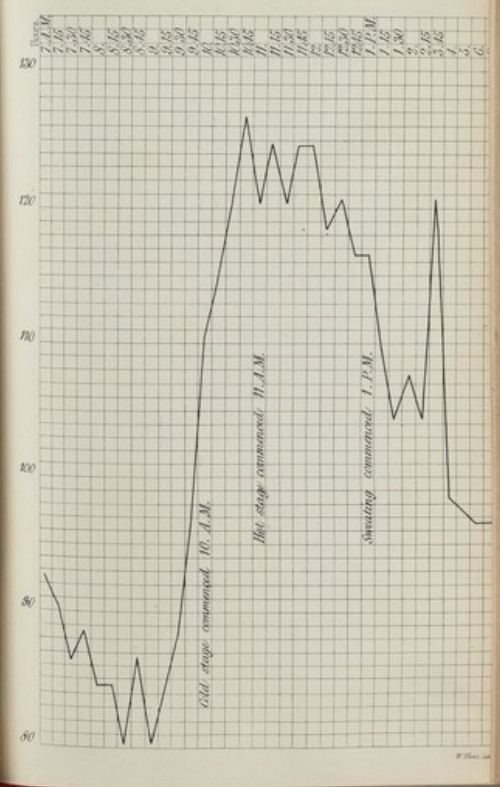
Pulse.



T. 594 N°

APRIL 4TH

Pulse.



Thus, during the period before the cold stage he drank 323 c.c. in excess over the quantity of urine passed.

Cold stage, he drank	100 c.c.	more	than he passed;
Hot stage, „	520 „	„	
Sweating, „	350 „	„	

making a total of 1293 c.c. in excess of the total amount of urine voided. In this respect, again, he corresponds to the previous patient.

The pulse was taken every quarter of an hour with the temperature in the two fits, and the result is shown in the two following charts. In the first the pulse at the very commencement falls somewhat, and then oscillates. Just before the commencement of the cold stage it commences rising, and then remains stationary for half an hour; then runs up enormously and rapidly during the whole of the cold stage, and at its termination remains again stationary for half an hour; then runs up slightly, oscillates when it has reached its highest point, and immediately on the sweating stage setting in it falls rapidly. In the second chart, at the very commencement, the temperature falls more than it did in the previous one; in the stage previous to the cold it rises more rapidly than in the previous one; runs up still more rapidly during the cold stage, reaches its highest point, and then immediately oscillating, before the sweating stage commences; after oscillating for some time, it falls, and at the commencement of the sweating stage it stands stationary for half an hour, and then falls greatly. During the entire rise there is no tendency for it to remain stationary, still less to oscillate.

This last fit was more severe than the former.

Thus, on both days the pulse commences by falling somewhat, then on both it rises before the fit commences, thus corresponding to the urine and its constituents and also to the temperature. At the commencement of the cold stage, in the less severe fit, the pulse remained stationary for half an hour, and again at the termination of the same stage, whilst nothing of this is seen in the severer one, thus cor-

responding to the temperature, &c. Again, on reaching the climax on both days the pulse oscillates, and the temperature also was unsteady at the same time, though to a much less extent. The pulse commences falling before the sweating stage, and then falls rapidly, more so than the temperature. The pulse, however, fell most rapidly in the less severe case.

Thus, a close correspondence exists between the pulse and the temperature. Like it, the character of the rise varies with the intensity of the fit; it rises before the cold stage commences; then, if the fit is not very severe, remains stationary; again rises through the entire cold stage, the rise being more considerable than during any other period; then falls before the sweating stage, and just at the commencement of this stage stands still for half an hour in the severer cases, and falls subsequently slower than in the less severe cases, in which also there is no stationary period.

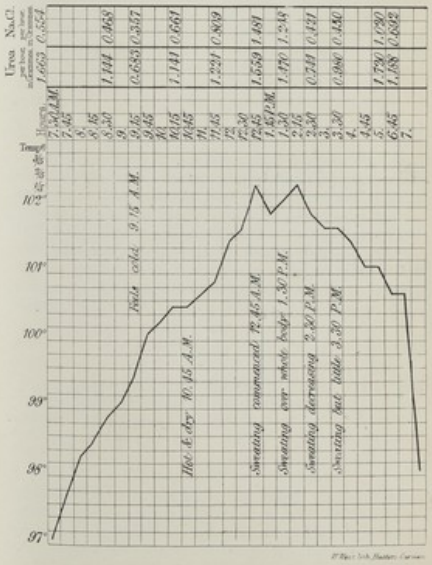
Case 3.—*A case of hectic fever.*—The following well-marked case of hectic is subjoined, on account of its close correspondence to the cases above given.

It occurred in a phthisical patient, æt. 45, under Dr. Walshe. It was particularly obstinate in resisting all measures for its removal, and was also peculiar in its occurring always during the day, and its very long continuance, as it commenced at 7 a.m., and continued to 5 p.m. All the stages were unusually well marked, consisting in severe rigors, accompanied with great pain about the loins and legs, the rigors coming on gradually, the sensation of cold being first observed about the feet; the hot and especially the sweating stages followed regularly. The observations were conducted in all respects after the manner of those already detailed.

Temperature.

Here, as in the other cases, the temperature began to rise before the rigors set in, ran up slowly through the

Case of Hectic.



whole of the cold stage, at its termination remained stationary for half an hour, then again ran up slowly, remaining stationary once, till it reached its highest point, when it oscillated, the rise in the oscillation occupying half an hour, then slowly and gradually fell, every now and then remaining stationary. A close connexion exists between this temperature and those given formerly; the rise, however, before the shivering set in was peculiarly great, and the cold stage very short, the rise in this stage not being so much in excess over the other stages as in the cases of ague.

Urea.

Unlike the cases of ague, the urea falls during the period preceding the rigors, and reaches its minimum immediately they set in; the quantity then rises rapidly, and becomes greatest during the hour immediately preceding the sweating stage. The quantity then falls rapidly through the sweating stage, reaching its minimum when the sweating is most severe; then rises again even higher than it was during the hot stage, and after that falls to its normal amount.

Chloride of sodium.

This corresponds to the urea in all respects, except that the rise at the termination of the fit does not equal the amount secreted during the hot stage.

Water

Again corresponds to both the above, except that its greatest quantity was poured out an hour later than the greatest amount of urea and chloride of sodium (showing the independence of these substances one of the other).

The patient was permitted to drink *ad libitum*; the quantity, however, was not measured.

The following table shows the variations in the amount of urinary water:

Hour.	Amount of Water.
7·30 a.m.	93 c.c.
8·30 „	52 „
9·15 „	35 „
10·15 „	58 „
11·15 „	71 „
12·30 p.m.	97 „
1·30 „	104 „
2·30 „	39 „
3·30 „	50 „
5 „	100 „
6·45 „	70 „

The pulse in this patient remained about 80 throughout the day.

General Conclusions.

The following are the conclusions deducible from the facts noted in the cases of ague.

Temperature.

1. This rises before the commencement of the subjective fit.
2. The time before the cold stage at which the rise commences varies.
3. It continues to rise during the entire cold stage.
4. The rise during this stage is greater than during any other.
5. It reaches its highest point during the hot stage, but falls again before the sweating stage, the fall being gradual.
6. The fall is more rapid during the sweating stage. The rapidity is in proportion to the slightness of the fit.
7. Definite variations occur in the rise and fall of the temperature indicative of alterations in the severity of the fit.
8. These variations first appear at the commencement and termination of the cold stage.
9. Their earliest indication is seen in a tendency for the later rises in the cold stage to become less extensive.

10. The temperature next becomes stationary for a variable time at either or both extremities of the cold stage.
11. Should the fit become still less severe, the temperature oscillates at these points.
12. An oscillation in the middle of the cold stage indicates a still further diminution in the severity of the fit.
13. Variations also occur during the hot stage.
14. In the severest fits there is a tendency for the temperature to remain stationary at its highest point.
15. If less severe, the temperature immediately falls on reaching its extreme height.
16. When the fits become still less severe the temperature next fails to reach to such a height as previously, but at this point again has a tendency to be stationary.
17. Should the severity of the fit be still less, it just touches the extreme point, and immediately falls.
18. Variations are also observed during the fall of the temperature.
19. Thus, at the junction of the hot and sweating stage, an oscillation, or a tendency to remain permanent, always occurs.
20. In continuing to fall, the temperature either falls gradually and continuously; this occurs in the severe fit—or,
21. Remains stationary every now and then; this indicates a somewhat less intensity of the fit—or,
22. It oscillates; this accompanies the most rapid fall, and occurs during the least severe fit.
23. After sinking to its extreme point, the temperature has a tendency to rise again, the rise being often considerable.

Urea, Chloride of Sodium, and Water.

24. The urea, chloride of sodium, and water, also begin to increase in quantity before the commencement of the cold stage.
25. They continue to rise rapidly, and become most

abundant either at the termination of the cold or the commencement of the hot stage.

26. These urinary constituents commence to fall in amount before the temperature reaches its highest point.

27. During the latter part of the hot stage they decrease in amount slowly.

28. They fall rapidly during the sweating stage, the rapidity of the fall being proportionate to the slightness of the fit.

29. These constituents exhibit variations corresponding to the variations in the temperature.

30. Thus, when the temperature remains stationary at either end of the cold stage, these urinary constituents also remain stationary, or fall somewhat. When the temperature oscillates, the fall in these constituents is proportionately greater.

31. A fall occurs in these constituents corresponding to the oscillation in the temperature at the termination of the hot stage, the fall in their amount corresponding to the depth of the oscillation.

32. A greater diminution in the amount of these constituents is observed when an oscillation occurs, than when the temperature decreases gradually, or tends to remain stationary.

33. The increase in the urea and water is definite, the same amount of increase corresponding to a single degree each day.

34. A greater increase in these constituents corresponds to a single degree at a high than at a low temperature.

35. No connexion existed between the various rises of the urea and chloride of sodium when equal periods were compared together.

36. The urea appeared to be independent entirely of the influence of the amount of water.

37. The chloride of sodium also underwent a definite increase, but this increased per-centage remained the same whatever the amount of water was, so that the total amount of chloride was greatly under the influence of the urinary water.

38. The chloride rose steadily and constantly, and did not observe the variations corresponding to temperature which were seen in the urea, but the water varying with the urea and influencing the chloride, as seen above, caused similar variations in the hourly amount of the chloride.

39. The quantity of water drunk in no way influenced the total amount excreted.

40. The increase in the above constituents often commenced before a corresponding rise in the temperature occurred.

41. The variations in the temperature above described often followed similar variations in the urea, but never preceded them.

42. Quinine given in a single, but large dose, when the temperature commenced to rise, lowered the temperature and postponed the fit for an hour, but had no other effect on that fit, though it prevented its recurrence next day, another scruple having been taken.

43. The pulse corresponded closely with the temperature.

ON
THE RELATIVE AMOUNTS
OF
SUGAR AND UREA IN THE URINE
IN
DIABETES MELLITUS.

BY

SYDNEY RINGER,

LATE PHYSICIAN'S ASSISTANT AT UNIVERSITY COLLEGE HOSPITAL.

COMMUNICATED BY

RICHARD QUAIN, F.R.S.

[*From Volume XLIII of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

J. E. ADLARD, BARTHOLOMEW CLOSE.

1860.

ON

THE RELATIVE AMOUNTS OF SUGAR AND UREA IN THE URINE

IN

DIABETES MELLITUS.

BY

SYDNEY RINGER,

LATE PHYSICIAN'S ASSISTANT AT UNIVERSITY COLLEGE HOSPITAL.

COMMUNICATED BY

RICHARD QUAIN, F.R.S.

Received May 28th.—Read June 30th, 1860.

The observations were made on two patients, inmates of University College, under the care of Dr. Parkes.

In both cases the urea was estimated quantitatively by Liebig's volumetric method. The usual allowance was made for the chloride of sodium, except in the case of Harriet Meader, in whom it was so small in quantity that no allowance was made. The sugar was estimated quantitatively by the copper test. The water is given in cubic centimètres, and the urea and sugar in grammes.

The following points are attempted to be determined.

1. The relation between the excreted amounts of sugar and urea.

2. The influence of starch and sugar on the excretion of urea.

3. The time at which the sugar reached its maximum after a meal.
4. The duration of the influence of a meal.
5. The temperature of the body.

Case 1.—George Hudson, æt. 30; height, five feet nine inches and a half; weight in health, eleven stone nine pounds. For six months previous to his admission into the hospital he had suffered from symptoms of diabetes, which had increased rapidly in severity, and were strongly marked at the time of his admission. During the first sixteen weeks they continued with unabated activity. After this he improved, and his weight and strength increased. He had some tubercular deposit in both lungs, which had advanced to excavation on the left side. During the early part of the investigation he perspired greatly. During the whole of these observations the patient was purposely kept without medicine. The observations were commenced on October 13th, 1859.

Case 2.—Harriet Meader, æt. 30, of very diminutive stature. She had suffered ten years previously from diabetes, and was greatly emaciated, though she had been in much the same state during the greater part of her illness. She was almost totally blind from double cataract, and her intellect was greatly impaired. She also suffered from a slight leucorrhœal discharge, so that her urine contained a *minute* trace of albumen, which was removed by amylic alcohol before testing for the sugar, but was not removed previous to testing for urea. During her whole illness she was subjected to no treatment, with the exception of having brown bread in the place of white. Nor was any medicine given her whilst these observations were being made.

1. *On the relation between the excreted amount of sugar and urea.*

After the influence of food on the urine had disappeared

by long abstinence, a constant ratio was found to be maintained between the sugar and the urea. This ratio was 1 of urea to 2·2 of sugar. The ratio is most strictly observed when several hours are compared together in preference to single hours, as the sugar and urea do not exactly correspond in the exact time of elimination. The following tables show the above ratio clearly:

George Hudson.

Urine in inanition.

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
9th	4·881	2·582	...
10th	4·272	2·415	...
11th	5·350	2·127	...
12th	5·350	2·127	...
13th	4·545	1·598	...
14th	5·025	2·202	...
15th	3·260	1·560	...
Total	33·211	14·612	1:2·2

The same ratio was observed on several other occasions; not, as above, in several consecutive hours, but for a single hour only. For, on any day that observations were made, a fasting hour was first taken for comparison. Thus:

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
14th	2·453	1·428	1:1·7
14th	5·319	2·430	1:2·1
13th	3·977	1·680	1:2·3
12th	4·825	1·909	1:2·5
			Mean 1:2·1

Harriet Meader.

In the following table three consecutive hours are given.

Urine in inanition.

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
15th	1·631	0·727	1 : 2·2
16th	1·631	0·727	1 : 2·2
17th	1·041	0·456	1 : 2·2

But not only was the above ratio observed during inanition hours, but also after a non-amylaceous and non-saccharine diet; for though both the urea and sugar rose greatly, the same ratio was maintained. Here, again, though, when separate hours are compared, the ratio varies slightly, still the mean ratio is the same as that above given.

Hudson.

December 30th.—At 10 a.m. he breakfasted on eggs and mutton. He took tea the evening previous at 5, and had nothing between that time and breakfast.

Urine of nitrogenized food.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
9 to 10 a.m.	4·324	1·676	1 : 2·5
10 to 11 „	3·856	1·583	1 : 2·4
11 to 12 „	3·604	1·972	1 : 1·8
12 to 1 „	3·604	1·972	1 : 1·8
1 to 2 „	4·181	2·618	1 : 1·6
2 to 3 „	8·214	3·832	1 : 2·1
3 to 4 „	5·851	2·970	1 : 1·9
4 to 5 „	4·427	2·380	1 : 1·8
5 to 6 „	3·437	2·042	1 : 1·6
			Mean 1 : 1·97

January 2d.—He dined at 6.30 on fish, mutton, and eggs. He had taken nothing since the evening previous.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
6 to 7 p.m.	0·750	0·583	1 : 1·2
7 to 8 „	3·846	2·080	1 : 1·8
8 to 9 „	7·937	3·048	1 : 2·6
9 to 10 „	9·523	3·744	1 : 2·5
10 to 11 „	8·522	3·450	1 : 2·4
11 to 12 „	7·283	3·510	1 : 2·0
12 to 9 a.m.	3·945	2·034	1 : 1·9
			Mean 1 : 2·1

The hourly mean, and not the total amount, is given between 12 p.m. and 9 a.m.

4th.—He dined at 11 p.m. on fish and meat.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
11 to 12 p.m.	8·695	3·520	1 : 2·4
12 to 1 a.m.	6·818	3·300	1 : 2·0
1 to 2 „	6·607	3·293	1 : 2·0
2 to 7 „	6·666	2·688	1 : 2·4
			Mean 1 : 2·3

Between the hours of 2 and 7 the hourly, and not the total, amount is given.

That some such ratio existed was anticipated on *à priori* grounds, for the sugar, under the above conditions, is probably derived from the nitrogenous elements of the body, and the nitrogen of the albuminous matters which are resolved into sugar must apparently pass off as urea: thus some ratio was probable; for the sugar could only be derived from the nitrogenous or oleaginous constituents of the body. And though reasons have been advanced tending to show that sugar and fat are inter-convertible in these cases, I think it must be admitted that the sugar came from the nitrogenous elements. The fact of the urea

maintaining a constant ratio with the sugar in all its variations, under these circumstances, is strong proof of this. For though it may be said that a certain amount of tissue would be consumed in the conversion of fat into sugar; also that urea being the exponent of this, as the tissue used would be in proportion to the sugar formed, so the urea would hold a constant ratio to the sugar; still, the increase of the urea is much too great to be accounted for in this manner, being often double and treble the amount normal to the patient, which is the exponent of all the changes (secretion, &c.) taking place in the body, and would surely more than equal the loss of tissue required to convert so much fat into sugar.

2. *On the influence of amylaceous or saccharine food on the excretion of urea.*

The excretion of urea appears to be increased after amylaceous or saccharine food, and this independent of the kind of sugar used. The following tables render this probable.

HARRIET MEADER.

December 17th.—The patient dined at 4 on mixed diet. At 12 p.m. eight drachms of cane sugar were given to her.

Hour.	Quantity.	Sugar.	Urea.
9 p.m.	261 c.c.	14·085	1·357
12 „	220 „	11·000	1·166
1 a.m.	235 „	13·976	1·222
2 „	282 „	14·100	1·381
3 „	225 „	10·416	1·125
9 „	140 „	7·954	0·840

The above (as is the case with the following) experiment is not so perfect as could have been desired. As it was impossible to keep her without food till the influence of the previous meal had disappeared. Still, the sugar was never given her till the urinary sugar and urea had commenced to decline.

In the above table it is seen that the urea was increased in the second hour after the sugar was taken by 0·220 gramme. The actual increase was much more than this; for the urea would have continued to decrease in amount during these two hours if no sugar had been given, the influence of the previous meal during that time becoming less.

19th.—The patient had taken no food since 11 p.m. the evening previous; at 10 a.m. eight drachms of sugar of milk were given to her.

Hour.	Amount.	Sugar.	Urea.
8 to 9 a.m.	238 c.c.	11·053	1·528
9 to 10 „	107 „	6·079	0·920
10 to 11 „	150 „	8·333	1·120
11 to 12	165 „	9·375	1·270
12 to 1 p.m.	102 „	6·071	0·683

19th and 20th.—She dined heartily at 4 p.m. on mixed diet. At 12 p.m. she had eight drachms of sugar of milk given her.

Hour.	Amount.	Sugar.	Urea.
9 to 10 p.m.	101 c.c.	5·295	0·671
10 to 12	195 „	9·461	1·482
12 to 1 a.m.	255 „	11·590	1·785
1 to 2 „	230 „	10·865	1·610
2 to 3 „	158 „	8·977	1·216

20th and 21st.—The patient dined, as usual, on a mixed diet at 4 p.m., and at 12 p.m. eight drachms of sugar of milk were given to her.

Hour.	Amount.	Sugar.	Urea.
7 to 8 p.m.	305 c.c.	16·576	1·830
8 to 10 „	250 „	12·019	1·550
10 to 12	187 „	10·190	1·325
12 to 1 a.m.	210 „	10·744	1·449
1 to 2 „	234 „	10·833	1·544
2 to 3 „	195 „	9·375	1·267

22d.—She breakfasted at 9 on mixed diet. At 1 p.m. eight drachms of glucose were given her.

Hour.	Amount.	Sugar.	Urea.
10 to 11 a.m.	286 c.c.	16·388	1·510
11 to 12	258 „	13·913	1·522
12 to 1 p.m.	55 „	3·197	0·368
1 to 2 „	195 „	11·079	1·092
2 to 3 „	187 „	12·331	0·990
3 to 4 „	130 „	9·027	0·728

On the same day she dined, as usual, at 4 p.m. At 12 p.m. she had eight drachms of glucose given her.

Hour.	Amount.	Sugar.	Urea.
8 to 9 p.m.	243 c.c.	13·522	1·849
9 to 12	253 „	11·727	1·570
12 to 1 a.m.	280 „	12·096	1·820
1 to 3 „	...	5·062	1·582
3 to 9 „	...	6·713	1·203

George Hudson.

January 5th.—The evening previous he took a non-amylaceous dinner; consequently the sugar and the urea had nearly reached the ratio observed during inanition hours, and the influence of the meal was much more completely worked off than in the previous tables, thus more correctly showing the actual increase after sugar was taken. Four ounces of cane sugar were given him at 9 a.m.

Hour.	Amount.	Sugar.	Urea.
8 to 9 a.m.	60 c.c.	4·166	1·600
9 to 10 „	126 „	9·834	2·102
10 to 11 „	142 „	12·241	2·101
11 to 12	127 „	11·759	1·778
12 to 1 p.m.	82 „	8·200	0·984

In all the above tables, when more than an hour elapsed before the urine was examined, the total amount of water,

sugar, and urea is not given, but the mean hourly amount.[1]

This fact is also well shown on other occasions, in other experiments not devised for this special purpose. Thus, after a hearty non-amylaceous meal, the urea was found to be much less than after a mixed diet, though it may be fairly granted that the amount of nitrogenized food taken was greater in the former than the latter kind of meal. Thus, on December 30th, he took a non-amylaceous breakfast; the highest hourly amount of urea was 3·832 grammes (59 grains). On January 2d he had a similar dinner, and the highest hourly amount of urea was 3·744 (58 grains). On both these occasions he ate most heartily. On October 24th, after a mixed breakfast, the highest hourly amount of urea was 5·3 grammes (82 grains). After a similar tea on the same day, 9·2 grammes (142 grains). On October 28th, after a mixed breakfast, the urea reached 5·355 grammes (83 grains). On the same day, after a similar tea, 7·7 grammes (119 grains) per hour. These are enough for examples, though many others might be given.

The facts above attempted to be established—namely, that during inanition, and also after a meal containing nitrogenous matter, whether combined with amylaceous matter or not, albuminous matter is converted into sugar and urea, and that this is greatly increased by the administration of sugar, and therefore by an amylaceous diet—are quite sufficient to account for the great loss of weight and

[1] In the following case, a patient, æt. 11, under the care of Dr. Hare, half a pound of sugar was given at 12 p.m., the urine having been collected for the two hours previous, and again from 12 p.m. to 7 a.m.

Hour.	Sugar.	Urea.
10 to 12 p.m.	4·049	1·322
12 p.m. to 7 a.m.....	12·668	3·230

As usual, the hourly amount is given in the above table.

During the above experiment, the patient took no food between 5 p.m. and 8 a.m. next morning.

In the above table the hourly amounts of sugar and urea are given.

strength in these patients, and the general degeneration of the functions of the body.

It may, perhaps, be said that the increase of the urea, after the administration of sugar, was due to this causing an increase in the amount of urinary water, under which circumstances it is known that an increase of urea takes place.[1] But the increase then depends on previous retention, probably due to a deficiency in the amount of urinary water. Now, it cannot be said that in diabetic patients there is any deficiency in this.

Again, if the increase in the amount of urinary water were the cause of the increase in the amount of urea, there should be a relationship between the two, but nothing of the kind holds, as is seen from the following table, in which the water and urea are given for the twenty-four hours, and the ratio between them also. On all these days the patient took ordinary mixed diet.

HUDSON.

Date.	Water.	Urea.	Ratio of urea to water.
October 17th	5575 c.c.	82·339	1 : 68
18th	5354 „	83·037	1 : 64
19th	5129 „	74·258	1 : 69
February 14th	3805 „	33·454	1 : 115
15th	4042 „	32·336	1 : 126
16th	4400 „	30·800	1 : 146
18th	4000 „	25·600	1 : 160

The first three closely correspond; but, as the water is obviously under the control of the sugar and the urea also, when there is a ratio between the two latter there will of necessity be a relation between the water and urea, and this occurred on the first three days given.

[1] Thus, in a healthy subject, of average height and build, by drinking a pint of water hourly, the amount of urea rose from 2·0 grms. per hour to 4·040 grms. for three hours. During the fourth hour, however, it fell to 3·026. It is true that the usual allowance was not made for the NaCl; but the amount of urinary water was increased enormously, whilst the NaCl suffered no such increase.

The above table, I think, shows indubitably that the increase in the urea is independent of the increase of urinary water, as the ratio between the two varies from 1 : 68 to 1 : 160.

On referring to this table, showing the increase in the amount of urea and sugar after a non-saccharine and non-amylaceous meal, it will be seen that an enormous increase in the amount of the urea resulted. It has also been shown that there is probably an increased formation of urea in the body, due to the ingestion of saccharine matter; but the amount of urea eliminated by one or both of these causes may vary in different patients, and in the same patient in different periods of the disease. From the above cases it appears that the condition of the patient depends on the amount of urea, and not on the amount of sugar, eliminated; that, in fact, the improvement of the patient is due to a diminished amount of urea formed, whilst, at the same time, the amount of sugar eliminated may remain the same or be greatly increased.[1] Hudson's case illustrates this. At first he lost flesh with great rapidity, all his symptoms being proportionately severe. The disease continued with unabated severity for nine months. He then apparently remained in *statu quo* for a few weeks, but towards the latter part of the investigation he improved greatly in strength and weight. These variations are well seen in the fluctuations in his weight. Thus, in health he weighed 163 lbs.; January 3d, 107 lbs.; January 26th, 103 lbs.; February 19th, 113 lbs.; February 25th, 113 lbs.

On referring to the following table, it will be seen that coincident with this improvement was an increase in the amount of excreted sugar (this increase was accounted for by an increase in his appetite). But at the same time that this increase in the sugar occurred, a large diminution in the amount of urea took place. Thus, during October, November, and December, he rapidly declined; but in January and February he improved greatly.

[1] Of course, in judging of the increase of the urea in these cases, the amount of nitrogenous food ingested must be considered.

Date.	Sugar.	Urea.
October 17th	324·840	82·339
18th	342·413	83·037
19th	313·091	74·258
January 26th	228·947	29·928
27th	339·285	34·960
28th	231·761	32·637
30th	366·935	41·860
31st	340·357	40·026
February 1st	389·224	30·702
2d	327·307	19·743
3d	281·774	33·542
4th	562·500	42·120
6th	457·900	28·901
7th	429·600	41·639
8th	446·354	44·564
9th	512·000	29·696
10th	456·250	42·705
11th	609·659	40·237
13th	541·666	32·240
14th	413·586	33·454
15th	459·318	32·336
16th	392·857	30·800
18th	313·500	25·600

That the severity of the symptoms is greatly due to the urea is also rendered probable from three other cases, one of which is given above, the other two Dr. Garrod kindly allowed me to take. In all these the amount of sugar excreted was nearly as large as the amount in the case of Hudson during the most severe part of his disease (October), but the amount of urea was very much less, and at the same time their condition was stationary, and had been so over a period of about ten years.

The following table shows the daily amount of sugar and urea in the case of Harriet Meader, on mixed diet.

Date.	Sugar.	Urea.
December 16th	189·435	23·300
17th	248·504	26·098
18th	238·712	33·653
19th	257·475	30·196
23d	247·589	33·885

Dr. Garrod's cases were taken for only one day each; they gave the following amount of sugar and urea:

	Sugar.	Urea.
1st	295·454	39·009
2d	264·309	32·374

The variation in the amount of urea in different patients, and in the same patient at different periods of the disease, may be due either to a lessened influence of the ingested sugar or to a diminished amount of nitrogenous matter resolved into sugar and urea, independent of the influence of sugar (which we have seen occurs), or, as is probable, it may be due to both causes combined.

This twofold origin of urea will readily account for their enormous appetite; for the chief part of the albuminous matter ingested being resolved into urea and sugar, proportionately less will be left to nourish the tissues, and if the entire amount of nitrogenous matter should be eliminated, then we can understand how it is that they experience a sense of hunger immediately after a meal. And if it be established that the ingestion of sugar has the power to increase the resolution of albuminous matters into urea and sugar, it is conceivable that the amount thus resolved may be even greater than the amount taken in as food; then the blood or other tissue will be attacked, and the meal will then leave the patient in a worse condition than it found him.

These facts also explain how it is that abstinence from amylaceous matter, as food, is often followed by such good results.

The urea in Hudson's case increased after a meal, corresponding, in this respect, to the increase that occurs in health, except as regards amount; and there can be little doubt, I think, that the increase in his case was due to the same causes that operate in health. But as the sugar increased also in his case, maintaining the ratio mentioned above, after a non-amylaceous meal, if it can be shown from

whence the sugar is derived it will also show the origin of this rise in the urea.

Now, as regards the increase of the sugar, there are two conceivable sources—1st, the tissues (retrograde metamorphosis); 2d, some organ, by its function, resolving nitrogenous matter into sugar and urea, which function, in diabetes, is altered, so that the sugar is less highly elaborated, and thus non-consumed.

Again, the sugar during inanition periods, and the increase after a non-amylaceous meal, are probably derived from the same source.

The chief difficulty in settling this question lies in the difficulty of proving whether any of the sugar, either taken in *ab externo*, or formed in the body, is consumed, or whether all is thrown off (as far as these cases are concerned).

There are reasons for thinking that some is consumed; thus, the enormous rise in the temperature after a meal would seem to point to this conclusion, though, as the diet in all these cases was mixed, it might have been due to oleaginous matters, taken in in unusually large amount. On the other hand, there are some rather strong reasons for thinking that all the sugar was thrown off. For, though the temperature rose so highly after food, this did not stand in proportion to the amount of amylaceous food taken; thus the temperature reached its highest after dinner, when the nitrogenous and oleaginous elements were in excess, the patient then taking much less bread. On October 24th, after dinner, it reached 101°; the same evening the temperature rose nearly as high as 100⅘°, but he had a meat tea; he ate, however, much more bread than at dinner. On October 27th he had a very good breakfast, but only toast and tea; the temperature reached 99⅘°. And on the 28th, after a very good tea, it reached only 99⅕°, and it must be remembered that he took butter with his bread at breakfast and tea, so that would, perhaps, account for the great rise, even then.

Again, as there is a constant ratio between the sugar and

the urea during inanition and after a non-amylaceous diet, it is obvious that if any is consumed it must be complete up to a certain point, and after that in a constant ratio; if this applies to sugars formed in the body, it probably applies to all. If so, it is evident that there should be a relationship between the sugar excreted and the temperature (if the former is the cause of the rise of the latter); or if the temperature is due to the sugar consumed, and this again holds a constant proportion to that excreted, the excreted amount will hold a proportion to the temperature; but there is not the slightest relationship between the two. It may be said that both the sugar and the oleaginous matters were concerned in the elevation of the temperature, and as the proportion of the two to one another, in the food, varied, so no relationship would exist between the sugar and the temperature. But admitting the force of this, still, if any were due to the sugar, there could not be so great a discrepancy as constantly exists between the temperature and the sugar, no vestige of a relationship existing between them. This is well seen by looking at the diagrams, where it will be observed that, on October 24th, at 3 p.m., the temperature stood at 101°, whilst the sugar amounted to 17 grammes per hour. At 10 p.m. the temperature stood at 100⅖°; the sugar had reached 37 grammes per hour.

27th.—The temperature at 11 a.m. was 99⅗°, and the sugar stood at 30 grammes.

28th.—At 9 p.m. the temperature showed 99°. The sugar reached 31 grammes per hour.

It thus appears to me that the temperature cannot be taken to prove that any of the sugar is consumed, indeed, the inference is in the other direction.[1]

It may be that some of the sugar taken in *ab externo*

[1] There is, however, another conceivable cause of the rise in the temperature. Thus it is known that at least some of the sugar ingested is converted into lactic acid in the alimentary canal, and being absorbed as such would be consumed, and cause an elevation in the temperature. (See Lehmann's 'Physiological Chemistry,' translated by the Cavendish Society.)

was consumed, whilst all that formed in the body was excreted. If this be granted, it is all that is required for the present purpose.

When abstinence of food was continued long enough, in both cases the ratio between the sugar and the urea was lost, that is to say, the urea ceased to fall, whilst the sugar still did so. Thus, in Hudson's case, on January 2d, the following occurred:

Hour.	Sugar.	Urea.
1·30	2·884	1·320
3	1·384	1·100
5	0·635	1·000
7	0·750	0·583

MEADER.

1·30	1·291	0·574
2·30	0·852	0·567
3·30	0·961	0·700

Now, if no sugar is consumed, to what is the excess, just at this point due? If the sugar is due to some organ acting on the albuminous constituent of the blood, and not due to the retrograde metamorphosis of the tissues, we should have a small amount of urea to be accounted for in this manner; and whilst the sugar and urea are very large in quantity, this would be insufficient to affect the ratio to any appreciable degree; but when the sugar and urea become much reduced, then the urea would be found in excess, as we find it here. For if the sugar is derived from the retrograde metamorphosis of the tissues, then they should fall, *pari passu*, maintaining the usual ratio. Thus I think we are driven to admit that the sugar is due, in these cases, to some organ which is endowed with this function; and even if some of the sugar was derived from the tissues by retrograde metamorphosis, the liver must have been at fault likewise. As it is known that it is here that ordinary sugar

is rendered available for the body, and as it is also known that the liver has the power of forming sugar from nitrogenous substances, it is more compatible with reason to ascribe the entire disease to the liver than to distribute it between it and the rest of the body.[1]

Thus, having shown it probable that the sugar in these cases was due to the function of the liver, it is of course in an equal degree probable that the rise of urea after a meal is due to the same organ.

From what has gone before in this paper, I think it appears that the excretion of urea from the influence of amylaceous food, and the excretion due to nitrogenous food, though mostly combined, are not necessarily so; that the sugar taken in may pass off unchanged, whilst the nitrogenous matters remain unattacked; but whether the reverse may occur is, perhaps, doubtful,[2] though conceivable. But besides these two possible forms of diabetes, there is probably a third, namely, the tissues which by altered retrograde metamorphosis, yield diabetic sugar and urea instead of their ordinary products.

Any theoretical consideration as to the nature of the lesion (in cases similar to those given above), based solely on the non-consumption of the sugar, if not erroneous, must be incomplete, as it is necessary to consider the excessive formation of sugar in the body. Thus we may reject as at

[1] Again, the liver (as is well seen in carnivora) forms sugar at the expense of the albuminous constituents of the body, the nitrogen of which must pass off as urea; and as we find an increased formation of sugar by the liver after a meal, so should we expect a corresponding increase in the amount of urea in the urine. Dr. Parkes, moreover, has endeavoured to show that in cases of abscess of the liver, with great loss of liver-tissue, the amount of urea is diminished. Another probable cause of part of the increase is the products of retrograde metamorphosis taken in with muscle, &c., as food, for we know the juice of flesh contains creatine and creatinine, &c. These would probably be absorbed to some extent, and some would pass on to the formation of urea.

[2] Since the above was written I have met with three cases which render it possible that such may be the case.

least insufficient Miahle's view, that the disease is due to an insufficient amount of alkali in the blood; also any views that look to the respiratory organs as at fault; as also any view that merely considers the disease due to mal-assimilation of sugar in the intestinal canal; and further, any system of treatment founded on such views must be received with caution.

3. *On the time at which the sugar reached its maximum in the urine after a meal.*

This varies according to the nature of the food and the stage of the disease.

Mixed diet:

HUDSON.

During the early experiments it reached its maximum during the third or fourth hour.

Date.	Hour.		
	Breakfast.	Dinner.	Tea.
October 17th	3d	—	—
18th	4th	2d	—
21st	—	—	3d
24th	—	—	4th
26th	3d	—	—
27th	4th	—	—
28th	4th	—	—

Later in the disease it reached its highest during the sixth hour.

January 6th.....6th.
January 13th.....6th.

Sugar in solution (both cases) reached its maximum at the second hour, with one exception, when the maximum was reached during the first hour.

In these observations the time occupied in eating the meal is included.

Pure albuminous diet:

Date.	Hour.		
	Breakfast.	Dinner.	Tea.
November 2d	—	4th	—
December 29th	—	4th	—
January 2d	—	4th	—

4. *Duration of the influence of food (mixed diet).*

The duration of the influence of mixed diet was longer towards the latter part of the observations.

October 19th.—Tea between 5 and 6 p.m. All effect of the meal had disappeared at 2 next morning, that is to say, the starvation ratio was reached by that time; but on December 28th the influence still existed after thirteen hours had elapsed, and might have continued longer, but the patient could wait no longer. In this woman the influence lasted fifteen hours. Thus it would appear, as might have been expected, that the digestive powers became impaired.

The urea had a tendency to pass off sooner somewhat than the sugar, possibly because it is more pernicious.

The increase of the sugar in the urine due to the influence of the meal lasted longer than the increase in the urea.

The above statements will be found to be correct on referring to the tables appended, in which the entire observations are given.[1]

[1] Besides the sugar and urea, the amount of urinary water in all cases is given; also in many cases the chloride of sodium, and in a few cases the specific gravity. No reference has been made in the previous pages to these constituents, as the observations were not sufficiently numerous.

HUDSON.

Till October 20th he was allowed to take what food he chose. This consisted of bread and butter, tea or cocoa, with sugar and milk, for breakfast. Meat, bread, and potatoes, with half a pint of porter, for dinner. His tea corresponded to his breakfast.

October 14th.—Breakfasted at 8·30; dined at 1; tea at 5.

15th.—Breakfasted at 9; dined at 1; tea at 5.

16th.—Breakfasted at 9·15; dined at 1; tea at 6.

17th.—Breakfasted at 8·30; dined at 1.

18th.—Breakfasted at 9; dined at 1; tea at 6.

19th.—Breakfasted at 9; dined at 1; tea at 5·30.

20th.—Breakfasted at 8.

Hour.	Quantity.	Sugar.	Urea.	NaCl.	Sp. gr.
October 13th.					
9 to 10 p.m.	390 c.c.	...	4·524	...	1042
10 to 11 „	355 „	...	4·473	...	1042
October 14th.					
11 p.m. to 8 a.m.	1115 c.c.	...	23·192	6·244	1050
8 to 9 a.m.	60 „	...	1·710	0·300	
9 to 10 „	205 „	...	3·895	0·820	
10 to 11 „	310 „	...	4·030	1·054	
11 to 12	330 „	...	2·640	1·386	
12 to 1 p.m.	295 „	...	2·006	0·914	
1 to 2 „	367 „	...	3·596	1·388	
2 to 3 „	335 „	...	3·886	1·239	
3 to 4 „	247 „	...	3·507	0·666	
4 to 6 „	377 „	...	7·351	1·508	
October 15th.					
6 p.m. to 8 a.m.	2600 c.c.	...	36·920	8·840	
8 to 9 a.m.	50 „	...	1·300	0·185	
9 to 10 „	80 „	...	2·136	0·320	
10 to 11 „	150 „	...	2·775	0·330	
11 to 12 „	265 „	...	3·445	0·530	
12 to 1 p.m.	265 „	...	3·445	lost	
1 to 2 „	353 „	...	3·996	1·198	1042
2 to 3 „	315 „	...	4·095	1·102	1043
3 to 4 „	265 „	...	4·081	0·715	1047
4 to 5 „	180 „	...	3·366	0·480	1051
5 to 6 „	225 „	...	4·320	0·832	1048
6 to 7 „	280 „	...	4·844	1·164	1047
7 to 8 „	350 „	...	4·865	1·120	1043
8 to 9 „	275 „	...	3·575	0·825	1045
October 16th.					
9 p.m. to 8 a.m.	1480 c.c.	...	23·680	6·600	
8 to 9 a.m.	50 „	...	1·270	0·225	
9 to 10 „	88 „	...	2·041	0·440	
10 to 11 „	220 „	12·200	3·580	0·572	
11 to 12 „	290 „	16·111	3·915	0·870	
12 to 1 p.m.	295 „	17·987	3·953	1·357	
1 to 2 „	360 „	25·714	4·080	1·476	
2 to 3 „	250 „	15·625	3·025	1·075	
3 to 4 „	397 „	20·253	5·161	3·295	1041
4 to 5 „	215 „	15·808	3·289	1·258	1050
5 to 6 „	215 „	14·527	3·504	1·225	1051
October 17th.					
6 p.m. to 8 a.m.	2225 c.c.	163·603	36·295	17·132	1049
8 to 9 a.m.	127 „	7·434	2·870	1·143	1051
9 to 10 „	225 „	14·423	3·487	1·755	1045
10 to 11 „	265 „	16·562	2·982	2·252	1040
11 to 12 „	590 „	26·339	6·608	3·304	1040

October 17th—*continued.*

Hour.	Quantity.	Sugar.	Urea.	NaCl.	Sp. gr.
12 to 1 p.m.	515 c.c.	23·842	6·025	3·399	1038
1 to 2 „	400 „	18·181	4·880	2·600	1036
2 to 3 „	465 „	16·146	5·673	3·441	1035
3 to 4 „	330 „	16·176	4·851	1·710	1040
4 to 5 „	215 „	13·109	4·178	0·838	1045
5 to 6 „	215 „	9·045	4·493	1·075	1050
October 18th.					
6 p.m. to 8 a.m.	2535 c.c.	147·384	43·095	9·126	1046
8 to 9 a.m.	52 „	2·453	1·428	0·273	1050
9 to 10 „	172 „	12·647	3·698	0·584	1045
10 to 11 „	255 „	16·346	3·493	0·994	1039
11 to 12 „	380 „	25·000	4·408	1·444	1040
12 to 1 p.m.	407 „	28·208	4·354	1·465	1039
1 to 2 „	443 „	24·611	4·562	1·993	1042
2 to 3 „	400 „	33·333	4·520	1·200	1045
3 to 4 „	270 „	15·000	3·591	1·215	1044
4 to 5 „	325 „	18·460	5·037	1·430	1040
5 to 6 „	315 „	13·815	4·851	1·795	1040
6 to 7 „	195 „	10·156	3·315	0·936	1048
7 to 8 „	270 „	21·094	3·051	0·864	1047
8 to 9 „	327 „	26·435	4·152	1·113	1046
October 19th.					
9 p.m. to 8 a.m.	1510 c.c.	92·037	26·275	6·946	1049
8 to 9 a.m.	100 „	5·319	2·430	0·750	1050
9 to 10 „	95 „	5·325	2·004	0·351	1050
10 to 11 „	237 „	17·426	3·578	0·616	1045
11 to 12 „	327 „	18·055	4·087	1·012	1042
12 to 1 p.m.	345 „	19·166	3·795	1·301	1040
1 to 2 „	388 „	22·558	4·362	1·590	
2 to 3 „	440 „	22·000	5·325	2·288	
3 to 4 „	395 „	21·944	4·891	2·409	
4 to 5 „	290 „	16·122	3·944	1·524	
5 to 6 „	250 „	15·244	4·050	1·175	
6 to 7 „	228 „	17·272	3·898	0·661	
7 to 8 „	313 „	22·357	4·695	0·939	
8 to 9 „	380 „	29·688	4·332	1·140	
9 to 10 „	435 „	21·750	5·176	1·696	
10 to 11 „	383 „	21·277	4·251	1·532	
11 to 12 „	342 „	21·625	4·856	1·675	
October 20th.					
12 to 1 a.m.	248 c.c.	12·917	2·429	1·288	
1 to 2 „	207 „	11·500	2·607	1·304	
2 to 3 „	123 „	4·881	2·583	1·210	
3 to 4 „	105 „	4·772	2·415	0·787	
4 to 6 „	197 „	10·701	4·255	1·635	
6 to 7 „	72 „	4·545	1·598	0·597	
7 to 8 „	97 „	5·052	2·201	0·834	
8 to 9 „	60 „	3·260	1·560	0·510	

For the tables on Oct. 26th, 27th, and 28th, the reader is referred to the diagrams.

December 28th.—Dined between 12 and 1; ordinary diet. Tea at 5·15; ordinary diet. At 6·45 he took some milk.

29th.—6 a.m., he took a non-amylaceous breakfast. Tea at 5.

30th.—Breakfasted between 9 and 10, on a non-amylaceous meal. He took nothing from that time till 6, when he dined on a mixed diet.

31st.—He took only breakfast and dinner, the latter at 6 p.m.

January 2d.—Breakfasted at 9, on a non-amylaceous diet; but scarcely ate anything till 6 p.m., when he ate a hearty dinner, composed of non-amylaceous matter.

4th.—He took nothing from breakfast till 6 p.m., when he took a pint of milk. At 11 p.m. he ate heartily of meat alone.

5th.—9 a.m., he took two ounces of cane sugar.

December 28th.

Hour.	Quantity.	Sugar.	Urea.
4 to 5 p.m.	308 c.c.	22·000	4·312
5 to 6 „	312 „	22·941	4·368
6 to 7 „	510 „	36·428	5·304
7 to 8 „	305 „	22·426	3·233
8 to 9 „	252 „	20·321	2·973
9 to 10 „	394 „	30 781	4·275
December 29th.			
10 p.m. to 5 a.m.	1860 c.c.	122·368	26·412
5 to 6 a.m.	122 „	8·714	2·293
6 to 7 „	107 „	7·037	2·204
7 to 8 „	105 „	6·907	2·268
8 to 9 „	98 „	6·447	2·254
9 to 6 p.m.	2160 „	154·285	35·424
6 to 7 „	363 „	23·881	5·445
7 to 8 „	345 „	20·535	5·106
8 to 9 „	190 „	11·046	3·268
9 to 12·30 a.m.	567 „	35·437	11·907
December 30th.			
12·30 to 8 a.m.	580 c.c.	45·312	13·456
8 to 9 a.m.	62 „	5·000	1·475
9 to 10 „	64 „	4·324	1·676
10 to 11 „	57 „	3·856	1·363

Hour.	Quantity.	Sugar.	Urea.
December 30th—*continued.*			
11 a.m. to 1 p.m.	124 c.c.	7·208	3·944
1 to 2 p.m.	77 „	4·181	2·618
2 to 3 „	138 „	8·214	3·832
3 to 4 „	110 „	5·815	2·970
4 to 5 „	85 „	4·427	2·380
5 to 6 „	66 „	3·437	2·042
January 2d.			
9 p.m. to 7·30 a.m.	2320 c.c.	45·238	26·216
7·30 to 8·30 a.m.	78 „	4·642	1·255
8·30 to 9·30 „	124 „	9·687	1·996
9·30 to 10·30 „	120 „	8·571	1·920
10·30 to 11·30 „	110 „	7·432	1·980
11·30 to 12·30 p.m.	87 „	5·178	1·705
12·30 to 1·30 „	60 „	2·884	1·320
1·30 to 3 p.m.	64 „	2·077	1·651
3 to 5 p.m.	61 „	1·270	2·000
5 to 7 „	72 „	1·500	2·332
7 to 8 „	80 „	3·846	2·080
8 to 9 „	127 „	7·937	3·048
9 to 20 „	160 „	9·323	3·744
10 to 11 „	150 „	8·322	3·450
11 to 12	134 „	7·283	3·510
12 to 9 a.m.	625 „	35·511	18·312
January 4th.			
7·30 to 8·30 p.m.	503 c.c.	26·198	9·355
8·30 to 11 p.m.	500 „	22·727	8·950
11 to 12	160 „	8·695	3·520
January 5th.			
12 to 1 a.m.	150 c.c.	6·818	3·300
1 to 2 „	185 „	6·607	3·293
2 to 7 „	640 „	33·333	13·440
7 to 8 „	153 „	10·066	3·457
8 to 9 „	60 „	4·166	1·600
9 to 10 „	126 „	9·834	2·102
10 to 11 „	142 „	12·241	2·101
11 to 12	127 „	11·759	1·778
12 to 1 p.m.	82 „	8·200	0·984

MEADER.

On December 15th, the urine was saved for the entire day, having been kept also through the night previous, from 12·45 a.m. At 5 p.m. of this day the patient ate a hearty tea; from this time she took no food till 11 a.m. December 16th, when we ceased collecting the urine for a few hours.

The same evening (December 16th) the patient ate a bad tea, at 5 p.m., composed of ordinary diet. The urine was collected from 8 p.m. The patient took no food till 12 a.m. next morning (December 17th). Thus she had no food for 18 hours. At 4 p.m. she took dinner and tea together. The urine was now continuously collected (from 8 p.m.); sometimes hourly; at others, during various periods, to 3 a.m. December 21st. December 17th, at 12 p.m., the patient took eight drachms of cane sugar, in 275 c.c. of water. At tea, the previous evening, she drank 570 c.c., and another 180 c.c. at 1 a.m.; beside this, she drank nothing till breakfast, at 9 a.m. the following morning.

December 18th.—At 11 p.m., took a mug of tea and a round and a half of toast.

19th.—At 10 a.m., took eight drachms of sugar of milk in 275 c.c. of water, the patient having taken no food since the tea and toast on the previous evening. On the same day she dined at 4 p.m., on fish, mutton chop, bread, and tea; during the rest of the evening she took nothing but water. At 12 p.m., eight drachms of sugar of milk, in the usual quantity of water, was given her.

20th.—At 3 a.m., she took some bread and butter and tea. Breakfasted at 10. Dined at 4, on a similar diet to that of the day before. 12 p.m., she drank eight drachms of sugar of milk, in 275 c.c. of water.

Hour.	Quantity.	Sugar.	Urea.
December 14th.			
5 to 9·15 p.m.	125 c.c.	2·976	1·975
9·15 to 10·45 p.m.	83 „	1·921	1·045
10·45 to 11·45 „	155 „	3·799	1·813
11·45 to 12·45 a.m.	117 „	3·052	1·240
December 15th.			
12·45 to 9 a.m.	2090 c.c.	96·759	15·048
9 to 2 p.m.	1205 „	37·631	7·230
2 to 12 „	1890 „	98·437	11·340
December 16th.			
12 to 9 a.m.	1025 c.c.	53·385	6·563
9 to 11 „	44 „	2·291	1·410

Hour.	Total Quantity.	Total Sugar.	Total Urea.
December 16th and 17th.			
8 to 10 p.m.	503 c.c.	25·150	4·024
10 to 11 „	272 „	12·362	2·067
11 to 1 a.m.	345 „	16·273	3·036
1 to 2 „	210 „	4·687	1·680
2 to 9 „	635 „	23·694	6·350
9 to 11 „	107 „	3·262	1·458
11 to 12 „	30 „	1·041	0·456
12 to 4 p.m.	570 „	29·688	4·104
4 to 9 „	1302 „	70·924	6·786
9 to 12 „	660 „	33·000	3·496
December 18th.			
12 to 1 a.m.	235 c.c.	13·976	1·222
1 to 2 „	282 „	14·100	1·381
2 to 3 „	225 „	10·416	1·125
3 to 9 „	840 „	47·727	5·040
9 to 11 „	217 „	12·616	2·039
11 to 3·30 p.m.	680 „	35·836	3·400
3·30 to 7 p.m.	1200 „	60·000	5·520
7 to 9 p.m.	375 „	20·833	2·875
December 19th.			
9 p.m. to 9 a.m.	2865 c.c.	132·638	18·336
9 to 10 a.m.	107 „	6·079	0·920
10 to 11 „	150 „	8·333	1·120
11 to 12 „	165 „	9·375	1·270
12 to 1 p.m.	102 „	6·071	0·683
1 to 4 „	1035 „	49·759	7·969
4 to 10 „	610 „	31·770	4·026
10 to 12 „	390 „	18·923	2·964
December 20th.			
12 to 1 a.m.	255 c.c.	11·590	1·785
1 to 2 „	230 „	10·865	1·610
2 to 3 „	158 „	8·977	1·216
3 to 5 p.m.	2873 „	149·719	16·683
5 to 7 „	375 „	23·439	1·987
7 to 8 „	305 „	16·576	1·830
8 to 10 „	500 „	24·038	3·100
10 to 12 „	375 „	20·380	2·625
December 21st.			
12 to 1 a.m.	210 c.c.	10·744	1·449
1 to 2 „	234 „	10·833	1·544
2 to 3 „	195 „	9·375	1·267

An intermission then occurred, and the urine was again collected from 9 a.m., December 22d, and kept from this time till 1 p.m., December 24th, over various periods.

December 22d.—She breakfasted at 9; breakfast consisted of bread and butter, with cocoa, with milk and sugar. At 1 p.m. she took eight drachms of glucose, in 275 c.c. of water. Dinner between 4 and 5, consisted of usual food. At 12 p.m. took another eight drachms of glucose, in same amount of water.

23d.—Breakfasted at 9; had three eggs, and tea without milk or sugar, thus solely albuminous food. Dined at 2, good; usual food. Tea at 5; bread and butter; then had nothing till next morning.

24th.—At 9 a.m. drank a mugful of tea; another at 10·20, and then had four eggs. The tea, on both occasions, was without milk or sugar. Dined at 2; usual food.

25th.—Took no starch food on this day, from 6 p.m. (nor milk).

26th.—Breakfasted, at 9·30, on egg and tea, without milk or sugar. Between 1 and 2, had meat solely. At 5 p.m. drank a solution containing three drachms of glucose. At 9 p.m. had meat and a very small piece of bread.

27th.—Breakfasted on eggs and tea, without sugar or milk, at 9·30. Dined, at 1·45, on chop and beef tea solely.

Hour.	Quantity.	Sugar.	Urea.
December 22d.			
9 to 11 a.m.	472 c.c.	32·777	3·020
11 to 12	258 „	13·913	1·522
12 to 1 p.m.	55 „	3·197	0·368
1 to 2 „	105 „	11·079	1·092
2 to 3 „	187 „	12·331	0·991
3 to 4 „	130 „	9·027	0·728
4 to 9 „	1217 „	67·611	9·249
9 to 12	760 „	35·183	4·712
December 23d.			
12 to 1 a.m.	280 c.c.	12·069	1·820
1 to 3 „	255 „	10·125	3·162
3 to 9 „	145 „	40·277	7·221
9 to 10 „	54 „	2·596	0·540
10 to 11 „	75 „	3·472	0·795
11 to 12	54 „	2·410	0·631
12 to 1 p.m.	87 „	3·625	1·061

December 24th.			
Hour.	Quantity.	Sugar.	Urea.
1 to 9 a.m.	3350 c.c.	154·296	31·235
9 to 10 „	110 „	4·741	1·001
10 to 11 „	135 „	5·443	1·593
11 to 12	90 „	3·629	1·116
12 to 1 p.m.	78 „	2·635	0·982

Another intermission then occurred in the urine, which was again collected from 4 p.m. to 8 p.m., December 26th. Then again it was collected from 11 a.m. to 3·30 p.m., December 27th. On this last day the amount of urine was very small, and then the chloride of sodium was allowed for. The amount of sugar also was so small that it was difficult to tell when all trace of blue colour was gone, and some mostly remained.

December 26th.			
Hour.	Total Quantity.	Total Sugar.	Total Urea.
4 to 5 p.m.	120 c.c.	5·769	1·224
5 to 6 „	128 „	6·666	1·190
6 to 7 „	205 „	9·318	1·640
7 to 8 „	154 „	6·875	1·185
December 27th.			
11 a.m. to 1·30 p.m.	133 c.c.	3·228	1·436
1·30 to 2·30 p.m.	45 „	0·852	0·567
2·30 to 3·30 „	50 „	0·961	0·760

5. *Temperature.*

Charts are given to show the variation in the temperature each quarter of an hour, judged of by a thermometer placed in the axilla. In the columns above, the amount of sugar, urea, chloride of sodium, and water, is given hourly, and written down opposite to the time at which the urine was collected. The hourly amount is always given.

Hudson.

October 24th.—The patient finished dinner at 1·10. He ate heartily of fish, bread, and potatoes, and took half a pint of porter. He took nothing more till tea, which he completed by 6·45, and ate heartily of meat and bread and butter; after this he took nothing till after 10 p.m.

26th.—Breakfasted exactly at 9; ate very heartily. At 4 a.m., previous, he had taken some tea and a little bread and butter.

27th.—He commenced his breakfast at 7; it consisted of tea and toast, the tea with milk and sugar; breakfast very good.

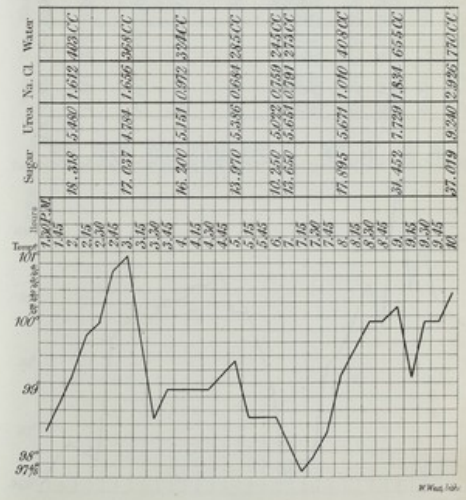
28th.—Breakfasted at 7·30; he ate six eggs for breakfast, and less bread than usual. Dined, at 1, on meat, bread, and potatoes, and half a pint of beer; he ate heartily. Tea at 5, very good.

At 6 p.m. the temperature rose suddenly to $100\frac{4}{5}^{\circ}$, and, in a few minutes, fell again to $99\frac{1}{5}^{\circ}$. The rise followed after a cup of tea, by no means hot. A similar rapid rise, and subsequent fall, after drinking, was noticed on another occasion.

These charts require no remarks, as they speak for themselves. Suffice it to say, that the temperature of the axilla was taken by an accurate thermometer, graduated into fifths.

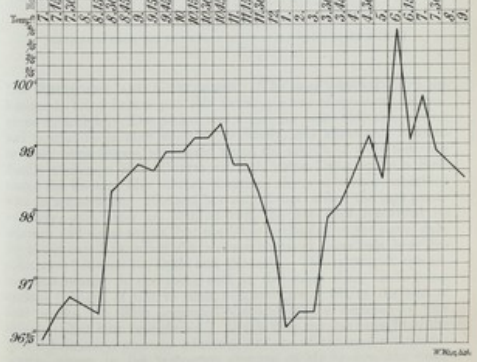
TEMPERATURE IN DIABETES MELLITUS.

OCTOBER 24TH



OCTOBER 28TH

Hours	Sugar	Urea	Na.Cl.	Water
AM.	4.825	1.869	0.686	80CC.
2.15				
2.30				
8.	3.384	2.010	0.738	84CC.
8.15				
8.30				
8.45				
9.	11.857	2.954	0.650	166CC.
9.15				
9.45				
9.45	13.404	3.685	0.811	254CC.
10.15				
10.45				
11.	18.206	4.723	1.340	335CC.
11.15				
11.30				
12.	16.203	5.535	2.065	350CC.
1.	12.500	4.475	1.900	230CC.
2.	11.136	4.116	1.048	196CC.
3.	20.640	6.180	2.342	412CC.
3.30				
3.45				
4.	24.506	7.091	4.880	600CC.
4.30				
5.	23.157	6.705	3.462	528CC.
6.	20.402	5.040	2.662	458CC.
6.15				
7.	25.223	7.514	3.390	665CC.
7.30				
8.	28.428	7.452	3.069	698CC.
9.	32.250	7.700	4.260	710CC.



ON

SPONTANEOUS GANGRENE FROM ARTERITIS

AND

THE CAUSES OF COAGULATION OF THE BLOOD IN DISEASES OF THE BLOOD-VESSELS.

By JOSEPH LISTER, Esq., F.R.C.S., Eng. and Edin.,
ASSISTANT-SURGEON TO THE ROYAL INFIRMARY OF EDINBURGH.

(*Read before the Medico-Chirurgical Society of Edinburgh, March* 17, 1858.)

[REPRINTED FROM THE EDINBURGH MEDICAL JOURNAL FOR APRIL 1858.]

Mr President and Gentlemen.—The case which I have the honour to bring before you this evening is one of spontaneous gangrene in a child. The patient was a girl, six years old, who, having had scarlet fever nine months previously, and afterwards suffered severely from dropsy, was seized about the middle of July last with deadly whiteness and coldness of both lower limbs up to a little above the knees. Her mother describes them as having resembled wax in appearance. She and her neighbours rubbed the legs perseveringly, and, after an hour or two, the left limb recovered its warmth and usual aspect, but the other continued in the same state for about two days, when the mother observed some pale blue discoloration between the ankle and the calf. This increased, spreading downwards to the foot; and, at the same time, undergoing various changes of tint to pink, red, and green, till, at the end of four weeks, the limb presented the appearance depicted in this sketch. [Not here reproduced.] At this time she was brought to the Royal Infirmary, and was admitted into one of the senior surgeon's wards, where, in the temporary absence of Mr Spence, she came

under my care. Her general health appeared remarkably good, considering all that she had gone through: her tongue was clean and moist, and her appetite good, and though her pulse was very quick, viz. 148, this was not to be regarded as a serious symptom in one so young, with such a source of irritation present. Accordingly, three days later, a line of demarcation having distinctly declared itself, I performed amputation immediately above the knee. Very little blood was lost in the operation, which did not accelerate the pulse or impair the appetite even for a day. Three days later, the constitution was evidently experiencing relief from the removal of the disease: the pulse was reduced to 112, and her general aspect improved. The stump healed kindly, and the result was in all respects satisfactory.

The amputated limb having been laid open on its posterior aspect, the gangrene was found to have extended somewhat higher in the deep parts than in the skin; the mortified tissues, including the posterior tibial nerve, were congested in a manner closely simulating inflammation, but of a duller tint, and exactly as far as this congestion extended, the posterior tibial venæ comites were turgid, and evidently contained coagulated blood. In the upper part of the limb the veins were flaccid and empty, and all the tissues appeared healthy, except that the popliteal artery, for an inch and a half from its lower end, was the seat of intense congestion, which also implicated slightly the cellular tissue about the vein and nerve. [These appearances were represented in a coloured drawing.] On laying open the vessels, the vein was found pervious and healthy, except that its coats seemed a little thicker than natural, but the artery was filled at the congested part with a coagulum, an inch and a quarter in length, partly pinkish and partly dark in colour. There was no appearance of any inflammatory exudation having taken place into the interior of the vessel, but the clot lay everywhere in contact with the internal coat, to which it was firmly adherent, so much so, as to tear away a portion of it when removed. Beneath the lining membrane were to be seen transverse red streaks, which appeared due to congestion of the circular coat of the vessel. [A sketch of the vessel and its contained clot was now exhibited.] The coagulum extended down the anterior tibial artery as far as to the commencement of the gangrene, but the companion veins were empty and perfectly natural in appearance, as also was the posterior tibial artery.

These pathological appearances clearly indicate that the primary disease was inflammation of the arteries, accompanied by coagulation of the blood within them, obstructing the supply of the nutrient fluid, and so inducing death of the lower part of the limb. This conclusion is in harmony with the previous history, which was from the first that of arterial obstruction.

This case is of practical interest, as illustrating the principle that spontaneous gangrene may be entirely local in its cause, and that in such cases the greatest benefit may be anticipated from removal of

the mortified part, provided the constitution be in a state fitted, as regards age and in other respects, for bearing the operation.

But my chief reason for bringing the case now before the Society is, because it appears a distinct example of inflammation of the coats of a vessel determining coagulation of the blood within it, without the exudation of lymph into its interior. Not that there is any novelty connected with such an occurrence, but because this effect of arteritis and phlebitis, long recognised by all sound pathologists, appears to be of peculiar interest at the present juncture, in connection with the recent publication of the last Astley Cooper Prize Essay, in which Dr Richardson of London propounds the theory, that the coagulation of the blood is due to the escape of a minute quantity of ammonia, which he believes holds the fibrine in solution. I propose, therefore, on the present occasion, to consider how far this new theory accounts for the phenomenon of coagulation in inflammation and other diseases of the blood-vessels.

So short a time having elapsed since the publication of the volume alluded to, it may be well to mention, as briefly as possible, the main facts by which the arguments of the author are supported:—And first I may relate the startling observation made by Dr Richardson, that if a current of air is passed through two successive portions of freshly drawn blood, contained in two Wolfe's bottles, while that in the first bottle, as might have been expected, has its coagulation accelerated, that in the second bottle is prevented from coagulating for several minutes after the time at which it would have solidified, had it been left in the vessel without interference. In other words, the air has had its properties so modified in passing through the first mass of blood, that it afterwards retards instead of promoting coagulation; whence Dr Richardson infers, that it has obtained in its passage some volatile solvent of the fibrine. Secondly, Dr Richardson has discovered that a very minute quantity of ammonia added to freshly drawn blood keeps it fluid for an indefinite period in a stoppered bottle, but that if exposed to air it coagulates as usual, though at a later period, in proportion to the amount of ammonia employed. He also finds that, by careful management, a fresh clot may be redissolved by means of ammonia, and that after the escape of the ammonia it will again coagulate, and afterwards contract in the usual manner, though more feebly. Next, he finds that ammonia is always to be obtained from the halitus of freshly drawn blood, and although the alkalinity of the blood through soda renders the ammonia excessively prone to escape, so that a good deal is necessarily lost from unavoidable exposure to air, yet he has succeeded in collecting about a third by weight of the smallest amount which he has found sufficient to keep the blood permanently fluid outside the body. Lastly, he has observed that all those circumstances which are known to promote the coagulation of blood outside the body, such as an elevated temperature, free admixture with air, a vacuum, etc., also hasten the process in blood mixed with ammonia, or, in other words,

favour the escape of the volatile alkali; while, on the other hand, those things which check coagulation, such as cold and occlusion from air, prevent or retard the evolution of the gas. To the latter class he has added the remarkable fact, that blood remains fluid for many hours under a high mercurial pressure, but coagulates when relieved from it. I confess that, although I was by no means prepossessed in favour of this theory, these facts appear to me to prove irresistibly that the cause of the fluidity of blood, after it has been drawn from the body, is a minute portion of free ammonia holding the fibrine chemically in solution, and that the coagulation of such blood is the result of the escape of the alkali. The only point on which the evidence appeared deficient was the effect of occlusion from air in tubes of dead matter, and this defect I endeavoured to supply by the experiment which I mentioned at last meeting of the Society, by which I succeeded in keeping the blood of a sheep fluid for three hours within a vulcanised Indian rubber tube, the blood coagulating in about two minutes when let out just as if freshly drawn from the veins of the animal.[1] Hence it appears to me that the medical profession is deeply indebted to Dr Richardson for his laborious and able investigations, which have, as I think, removed much mystery from this long vexed question.

But Dr Richardson aims at much more than the explanation of coagulation outside the body. He believes that the fluidity of the blood within the healthy living vessels is due simply and solely to the presence of free ammonia, which he supposes to be generated either in the systemic or pulmonary capillaries, and he denies that the walls of the arteries or veins have any effect on the blood by virtue of their vitality, or exercise any other influence upon it than that of checking

[1] This experiment was performed in the following manner:—One of the jugular veins of a sheep having been exposed, it was emptied of blood by passing the finger along it while pressure was applied by an assistant at its anterior part. The vessel was then opened at two places about three inches distant from each other, and into each opening was tied one end of a piece of vulcanised India rubber tube, a quarter of an inch in diameter, and about eighteen inches long, filled with water, to prevent the introduction of air into the circulation. The pressure was then removed from the upper part of the vein, so as to allow the blood to flow through the tube. It was now easy to ascertain, by observing the collapse of the lower part of the vein, when a part of the tube was momentarily obstructed by pressure, that the circulation was going on freely through the new channel. This having been determined, ligatures of waxed string were tied as tightly as possible round the tube, at intervals of about two inches, beginning at the end next the head and proceeding backwards, so as to avoid all tension upon the enclosed blood, which was, of course, displaced freely in the direction towards the thorax. By this means a number of portions of blood were obtained enclosed in receptacles nearly, though not absolutely impermeable to gases. The various compartments were opened at different intervals, and up to three hours some of them contained fluid blood which coagulated on exposure, whereas there was in others a considerable portion of coagulum. After four hours, coagulation was almost complete, but a slender thread of fibrine was still obtained from the fluid part in one of the divisions a few minutes after it had been let out.

the evolution of ammonia, just as would be the case were they tubes of dead matter of the same degree of permeability. And all cases of coagulation within the living body are supposed, by him, to be explicable on simple chemical principles. Here, however, I find myself quite unable to follow him. Thus, he believes that the coagulation in an aneurism is the result of the blood which is at rest in the tumour giving up its ammonia to the current which is flowing past the mouth of the sac. This theory was suggested to him by the following circumstance:—In one of the experiments of transmitting air through successive portions of blood, the longer tube in the last Wolfe's bottle was accidentally too short, so that it did not reach farther down than to about an inch from the bottom of the vessel. The result was, that while the upper part of the blood in this last bottle was retained fluid for a considerable time, that below the level to which the tube reached speedily coagulated. Dr Richardson infers that the lower portion of blood gave up its ammonia to the air which was bubbling through the upper part sufficiently charged with the alkali to retain that part in a state of fluidity. But, surely, this implies a mistake in chemistry. The lower portion of blood coagulated, I imagine, for the same reasons that it would have done had it been put in a stoppered bottle[1] after passing through the air, though probably not quite so rapidly, while the other part was prevented from coagulating by the ammoniacal vapour bubbling through it.

But if the ammonia theory fails to explain the coagulation that occurs in aneurism, still more inadequate does it appear to account for the phenomenon in arteritis or phlebitis. How can the fact that the wall of the vessel is inflamed determine, on simply chemical principles, the evolution of ammonia from the blood within it? Being convinced that in these and other cases of coagulation of the blood in local diseases of the vessels something remained quite unexplained, I have, during the last fortnight, made several experiments, with a view to throwing further light upon the subject, and will now communicate to the Society the results at which I have arrived.

In reflecting upon the matter, some circumstances in physiology and pathology appeared to me to indicate that, on the hypothesis that the blood does contain free ammonia in the living body, the healthy vessels must have a special power of preventing its escape. Thus, the blood in the capillaries of the lungs, is separated from the air in the air-cells only by an excessively thin partition of permeable living tissue; yet Dr Richardson's experiments have shown that there are times in the day, as, for instance, early morning, in which not a trace of ammonia is given off in the breath. Again, in surgical emphysema, the tissues of the body may be enormously distended with air, without any special tendency to coagulation of the blood in the vessels, such as might be anticipated unless

[1] This occurrence I have frequently observed.—J. L.

their parietes have a special power of preventing the escape of ammonia. It is true that in this case, the blood being in constant circulation, a perpetual supply of lost ammonia might be maintained from the capillaries; but it occurred to me that some information might perhaps be gained upon the point in question, by producing emphysema artificially in a limb in which the circulation had been arrested. For this purpose I applied a tourniquet firmly to one of the fore legs of a sheep, just above the elbow, and then injected air, by means of a condensing syringe, into the tissues of the lower part of the limb. The struggling of the animal, however, caused repeated displacement of the tourniquet, which I did not succeed in retaining in position for longer than an hour at a time. But, though the experiment was so far a failure, it yielded fruit in an unexpected manner. Having amputated the limb and preserved it, though with little expectation of learning anything from it, I was surprised to find, on examining it six hours later, that, although the cellular tissue about the vessels was still fully distended with air, the blood within them was perfectly fluid, and coagulated in about two and a half minutes, when shed into a saucer. Still greater was my surprise on finding next day, sixteen hours after the amputation, that the blood was still fluid in the vessels; and though it took longer to coagulate when let out from them, viz. five minutes, did so as fully as before. The muscular irritability, as tested by a powerful galvanic battery, had been found, on the previous evening, to be entirely lost. I next obtained four other feet, with the veins turgid with blood, by applying bandages firmly to the limbs below the joints where the butcher removes them, and amputating above the constricting band, after the sheep had been killed in the usual manner, by the knife. I examined veins in these limbs, day after day, till all the vessels were exhausted, and found at the end of the sixth day after their severance from all connection with the vascular and nervous centres, that the blood from a deep vein was still perfectly fluid, and coagulated when shed, though the time occupied by the process was now half an hour,—the length of the period having gradually increased, from day to day, since the time of the amputation. The feet, in the meantime, continued perfectly sweet, the coldness of the weather at the time being very favourable for the experiments. Some blood from a subcutaneous vein of the same foot, where decomposition might be expected to occur somewhat earlier, contained, at the same period (the end of the sixth day), some minute portions of coagulum. The fluid part of this blood remained liquid for an hour, but then coagulated well. Hence it was evident that so long as the tissues retained their freshness, the blood within the vessels was kept in a state of fluidity by some agency utterly inexplicable by the ammonia theory. I also found that the same thing occurs in the cat. In one such animal, killed under chloroform, by a knife passed into the great vessels of the neck, the blood in the veins of the extremities remained perfectly

fluid after forty-eight hours, and coagulated when shed. In another cat, killed by asphyxia, the same was the case as regards the posterior extremities; but the veins of the fore legs contained particles of coagulum, like the subcutaneous vessel of the sheep's foot. This difference I am inclined to attribute to the fact, that the animal made violent and protracted exertion with the fore legs during the death struggle, thus exhausting their vital energies more than those of the other limbs. After four days, however, the blood in the hind legs, though still fluid, with the exception of very minute particles of coagulum, had lost its power of coagulation. This increasing slowness and final absence of coagulation in blood long kept within the vessels, is curious, and must, I imagine, depend upon some gradual change in the properties of the fibrine.

We have seen that in two classes of the higher animals, differing from one another as widely as the carnivora and herbivora, and after modes of death so various as hæmorrhage, asphyxia, and an operation performed under chloroform, the blood remains fluid in the vessels, though perfectly at rest, for days after death. It may appear almost incredible that a fact of such fundamental importance, and at the same time so easy of demonstration, should have escaped the observation of all the eminent men who have made the coagulation of the blood a subject of special study; yet such appears to be the case. Dr Richardson speaks of occasional instances of fluidity of the blood after death, and coagulation on exposure, but considers it quite essential for such an occurrence that the vascular system should not have been opened by wound, though it is difficult to see how such a circumstance could affect the question, according to his theory, except on the supposition that the blood vessels were impermeable to gases in solution. Again, Dr Davey, in his "Researches," writes as follows:—"The blood, after death, I have often found liquid, and that many hours after death, when cold, but still retaining its power of coagulating;" but he had no idea of fluidity and coagulability lasting for days after death, or even for hours, except in rare instances. The nearest approach which I have been able to find to such an observation is contained in that inexhaustible treasury of original observation and profound reflection, the works of John Hunter, where the following passages occur:—"As a proof that blood will not coagulate in living vessels, in a perfect and natural state, and ready to act when powers were restored to it, I found that the blood of a fish, which had the actions of life stopped for three days, and was supposed to be dead, did not coagulate in the vessels, but, upon being exposed or extravasated, soon coagulated." "The blood of a lamprey-eel, which had been dead to appearance some days, was found fluid in the vessels, because the animal was not really dead: there had, however, been no motion in the blood, as the heart had ceased acting; but upon its being exposed, or extravasated into water, it soon coagulated." (*Palmer's Edition*, vol. iii., p. 32.) Hunter, however, does not

seem to have drawn any inference with regard to the higher animals from these cases. He speaks of "the very speedy coagulation of the blood which usually takes place in all the vessels after death" (vol. iii., p. 27); and though he believed that "where there is a full power of life, the vessels are capable of keeping the blood in a fluid state," he also supposed that some motion, though "very little, is required to keep up its fluidity" (ib., p. 32). Indeed, the expression, "full power of life," just quoted, is quite inconsistent with the state of a sheep's foot, six days after muscular irritability has been lost. I had myself frequently made experiments on inflammation upon the amputated limbs of frogs, and observed that the blood remains fluid for more than twenty-four hours after death; but muscular irritability, ciliary action, etc., also last in those creatures to a very much later period than in the higher animals, so that I never ventured to infer that fluidity of the blood was likely to continue long after death in mammalia.

Further observations on the feet of the sheep and limbs of the cat, proved even still more strikingly, the influence of the vessels upon their contained blood. If the skin be reflected from over a subcutaneous vein full of blood, and lightly replaced, so as to protect the subjacent parts from evaporation, without excluding the air, the vessel will be found, in two or three hours, changed from a dark venous colour to a scarlet arterial tint; yet no coagulation will occur in the blood, although the oxygen of the atmosphere has evidently penetrated freely through the coats of the vessel, showing that abundant opportunity has occurred for evolution of ammonia, provided any tendency to such an occurrence existed. Again, if such a vein be cut across with fine sharp scissors, without disturbing its connections, or inflicting much injury on its coats, the blood will be found, after about six hours, perfectly fluid in the vein, up to within perhaps 1-20th inch of the wound, where a small clot is perhaps seen, utterly insufficient to obstruct the progress of ammoniacal vapour. Hence it appears to me to follow, as a matter of demonstration, that, if free ammonia really exists in the blood within the vessels, the circumstance of its being in those vessels deprives it entirely of its volatility; and that, whether the ammonia be free in the blood or not, its chemical tendencies, such as it exhibits outside the body, are in some manner entirely modified by the vicinity of the vascular tissue. With regard to the nature of the modifying influence, no other explanation appeared to offer itself than that it depended upon residual vitality in the tissues.

In order to prosecute the investigation of the cause of coagulation in arteritis or phlebetis, I endeavoured to produce artificially, as nearly as practicable, in a living animal, the condition in which the vessels are when inflamed. Having proved, as I think I may venture to say—by investigations an account of which will shortly appear in the *Philosophical Transactions*—that inflammation consists in an impairment of the vital energies of the tissues of the part

affected, I resolved to destroy the vitality of a vein, and then permit the blood to flow through it for some time, and ascertain whether coagulation would occur in spite of the current, as it must do in phlebitis.[1] The agent which seemed best adapted for inflicting the lesion was strongest liquor ammoniæ, both on account of its rapid action, and also from the circumstance that, as Dr Richardson has shown, its chemical effect upon the blood, whether applied concentrated or diluted, is to prevent coagulation. On the 8th inst., having exposed one of the jugular veins of a sheep, and isolated it from surrounding connections for six inches of its length, carefully avoiding even momentary obstruction of the flow through it, I placed a plate of glass beneath the vein, to protect the neighbouring tissues from the action of the alkali, and at 3·13 P.M., emptied the portion of vein of its blood, by stroking the finger along it, while an assistant exerted gentle pressure on the anterior part, and then at once applied the liquor ammoniæ thoroughly, with a camel's hair brush, to all sides of the vessel throughout the length exposed. The application of ammonia occupied three quarters of a minute, and three quarters of a minute later the blood was again allowed to flow through the vessel, having been arrested altogether a minute and three quarters. A short time having been allowed for the evaporation of the ammonia, the edges of the wound were brought together with stitches. At 4.58, or an hour and three quarters later, the wound having been opened, the flow was again obstructed as before, and the vein was rapidly slit up. A small amount of dark coagulum escaped with the fluid blood. The interior of the vessel was now immediately examined. A valve with three flaps, about the middle of the opened portion, was rendered conspicuous from the fact that a black coagulum existed between each of the flaps and the wall of the vessel; and on careful observation of the lining membrane of the vein in other parts, it was seen to be dotted over in patches with fine granular deposits of pink fibrine, which could only be detached by firmly scraping with the edge of the knife, reminding me precisely of the close adhesion of the clot which occurred in the popliteal artery in the case which I related at the commencement of this paper, and which is known to be characteristic both of arteritis and phlebitis. Here it is clear that the coats of the vessel having been deprived of their vitality, the blood flowing through it assumed the same chemical tendencies as we have seen it to possess when removed from the body; and those parts of the fluid which re-

[1] Sir Astley Cooper performed experiments to show the effect of mechanical injury of the coats of a vein upon the coagulation of the blood at rest within a portion of the vessel contained between two ligatures; and he came to the general conclusion, that loss of vitality in the vessel greatly accelerated the process of coagulation. Dr Richardson alludes to these experiments, but says they have been invalidated by subsequent investigations by Scudamore. I have not as yet seen Sir Astley's own account of his researches on this subject; but, from a notice of them by Palmer, in his edition of Hunter's works, I suspect that they do not deserve to be set aside so lightly.

mained at rest under these conditions, namely, the motionless layer of liquor sanguinis next to the lining membrane, and the portions of blood in the sinuses of the valve, underwent coagulation, yielding up their ammonia through the permeable coats of the vein. And I think we need not hesitate to admit that similar occurrences take place in the early stages of arteritis and phlebitis, the coats of the vessels being in those cases not dead, but impaired in vital energy by inflammation.

A similar explanation appears to account for the early formation of coagula in the vicinity of a ligature placed upon an artery. It has been seen how utterly the usual explanation, that of the quiescence of the blood, fails to account for the phenomenon; but the fact that lymph is afterwards exuded from this part of the vessel, shows that the case is really one of limited traumatic arteritis.

But if the coagulation within inflamed vessels thus receives a solution from the results of the last mentioned experiment, still more unequivocally, at least to most of my hearers, is the coagulation in gangrene explained, such as occurred, for instance, in the case which has been described.

Again, it is well known that contused wounds bleed very little, the ends of the divided arteries becoming speedily plugged with a long coagulum. The only explanation which Sir Charles Bell could offer of this remarkable provision of nature was, that the living vessels had a special faculty of preventing the blood from exercising friction upon their lining membrane, but that the contused artery, having lost its vitality, the blood became arrested by friction, and coagulated. We now see that there was much more truth in this theory than has been generally supposed, though the loss of vitality in the vessel does not operate in the manner which Sir Charles imagined.[1]

It has been found difficult to understand why the fact of the arteries being converted into calcareous tubes should impress upon the blood within them a tendency to coagulate in atheromatous degeneration of the vessels. The impairment, or entire loss of vitality connected with such a condition, will now be found a sufficient explanation.

The coagulation in aneurism is now equally comprehensible, the walls of the sac consisting either of degenerated or torn coats of the vessel, of inflamed surrounding tissues, or of layers of fibrine, each of these constituents being in a state of very low vitality.

The rapid coagulation of lymph, which appears to be neither more nor less than the fibrine of effused liquor sanguinis, contrasts,

[1] I find I have not done justice to Sir C. Bell's views upon this subject. In his later works he expresses the opinion that the lining membrane of the living vessels possesses the power of "preserving the blood fluid," and that the cause of coagulation in a contused artery is the loss of this power in consequence of the injury.—*Vide* Sir C. Bell's *Institutes of Surgery*, vol. i., p. 52, and vol. ii., p. 277.

in a very striking manner, with the lengthened period during which blood extravasated into the cellular tissue may retain its fluidity. But the fact that the liquor sanguinis is exuded among tissues that are in a state of inflammation, and so impaired in their vital energies, renders the circumstance in question easily intelligible.

With regard to the nature of the influence exercised by the living vessels upon the blood within them, it might be conceived to be either of a positive or negative character. It might be imagined, either that the blood has a natural tendency within the vessels to comport itself as it does when outside the body, and that this tendency is counteracted by an active operation of the living tissues, or, that the vital fluid tends to no such change except when prejudicially acted on by surrounding objects, which in that case might be supposed to exert upon it attractive forces such as tend to group the molecules of dead matter together in aggregation, while the living tissues were destitute of such action, and simply neutral in their conduct towards the blood. Of these, the former has always appeared to me the more likely, *a priori*, but I had not expected to have met with any facts to give distinct evidence either in one direction or the other upon a subject so recondite. A simple observation, however, made on the sheep's foot, appears to throw clear light on the matter. I have frequently observed, that when a vein has been opened and has remained patent, the blood has continued fluid in the aperture for a very much longer time than is necessary to produce coagulation of a portion of that blood placed in a saucer. When the wound in the vein has been a narrow one, I have seen the blood remain fluid between its lips for three hours together, though perfectly at rest. I have even observed where a portion of fluid blood has been pressed from a vein into a groove in the tissues, formed by muscle below, periosteum on one side and tendon on the other, this portion of blood has remained fluid for an hour, while another drop removed into a saucer at the same time from the same vein, has coagulated in a quarter of an hour. Now, in all these cases the blood was fully exposed to the influence of the atmosphere; and if the air had been an active agent, promoting the tendency to coagulate, and the tissues merely neutral in their operation, coagulation must have occurred rapidly. On the other hand, if we admit that the tissues exert an active influence on the blood, and that air, oil, and other inorganic matter is inert with regard to it, the retarded coagulation follows naturally.[1] Here, then, it appears to me, we

[1] Since this paper was read, I have obtained further evidence which will, I trust, appear quite conclusive regarding the entirely negative influence of the atmosphere upon the blood, with respect to promoting the tendency to coagulation. Just forty-eight hours after the death of the sheep which furnished preparations exhibited to the Society, I exposed a vein of one of the feet and injected air forcibly into it, by means of a condensing syringe with a fine injection pipe adapted to it. Seven hours later I again examined the foot, and on reflecting the skin from the opposite aspect of the limb, found there a large subcutaneous vein distended with a mixture of blood and air; the latter, which

have a sure, though imperfect glimpse, of the operation of mysterious but potent forces, peculiar to the tissues of living beings, and capable of reversing the natural order of chemical affinities; forces which I suspect will never be fully comprehended by man in the present state of his existence, and the study of which should always be approached with humility and reverence.

Having thus obtained evidence of the active operation of the living tissues upon the blood, it occurred to me that the walls of the vessels might probably act to greater advantage upon their contents when of small than of large calibre, and that, in that case, the blood might be found fluid in the small vessels of the human body after death, although coagulated in the heart and large vessels. Accordingly, I have examined three human bodies with regard to this point, and in every case have found my idea confirmed. One of these was a woman, aged 70, who had been a patient under Dr Gillespie's care at the Infirmary, with senile gangrene. The right cavities of the heart were full of blood, and contained large clots buffed on their upper surface, and the large vessels also contained abundant soft coagula, but a small vein from one of the thighs yielded fluid blood, which coagulated slowly in a saucer. The body was examined about thirty-six hours after death. The other two had been patients under Dr Gairdner's care, also in the Royal Infirmary. One of these was a man about 30, who had died of meningitis. The heart had been removed before I saw the body, but the large vessels, such as the external iliac vein, contained coagula, whereas all the small veins which I observed contained perfectly fluid blood, which, however, had lost the power of coagulation. The third case was that of a young man, aged 21, who died of a complication of medical and surgical complaints, nearly forty-eight hours before the body was examined. The cornæ were perfectly clear, and there was no appearance of any incipient decomposition. This case was investigated very carefully; and as the subject is novel, it may be well to give the results in detail:—I was not present when the heart was removed, but Dr De Fabeck

had evidently passed through an anastomosing channel, being present in the form of very numerous large and small bubbles. Having secured the ends of a long piece of this vein, I dissected it out and shed its blood into a saucer. Not a particle of clot existed in the vein, and complete coagulation took place within a quarter of an hour. The vein which had been exposed, in order to inject the air, contained here and there portions of clot in the exposed part, the vitality of the vessel having doubtless been impaired by the mechanical violence to which it was subjected in the dissection, or by the drying influence of the atmosphere.

In order to illustrate the effect of mechanical violence applied to a vessel in promoting the coagulation of the blood within it, I pinched a vein of the same foot severely with dissecting forceps in about an inch of its length, at the same time that I injected the air into the other vein. On examining the foot, seven hours later, the vein which had been pinched contained coagulum in the part which had been so treated, but fluid blood in the rest of its extent, both above and below the injured portion.—J. L., 19th March 1858.

(resident physician under Dr Gairdner) informs me that the cavities contained coagula buffed on their upper surface. The vena cava, the right iliac veins (common, internal and external), and the femoral vein for about four inches down the thigh, contained soft coagula, mixed with thick dark fluid blood. The upper part of the axillary vein and the internal jugular of the same side, also contained some soft dark coagula, but the deep epigastric, the femoral vein below the part before mentioned, the internal saphena, and a smaller venous branch in the thigh, the axillary, except at the upper part, the cephalic and a subcutaneous vein of the throat, all contained fluid blood, which coagulated in about half an hour after being shed; and I noticed in the thigh that the blood from a small venous branch coagulated more quickly than that from the saphena. In the veins of the lower limb, both large and small, there were curious strings of highly elastic tawny fibrine, but these had evidently been deposited long before death. Similar threads were also present in the veins of the neck and in the aorta, external iliac and femoral arteries, which, however, contained but little blood, and no *post mortem* coagula. I did not test the coagulability of the blood in the arteries, nor in a branch of the internal iliac vein, which also contained fluid blood.

I am aware of one source of fallacy in these experiments, namely, that the abdominal viscera are subject to decomposition before the limbs; and as soon as decomposition does set in, the blood coagulates in the parts which are the seat of it; as, for example, in small veins of the intestines. This cause of error was, however, I think, guarded against in the last case;[1] and considering the almost universal occur-

[1] Through the kindness of my friend Mr John Gamgee, of the New Veterinary College, I have had the opportunity of making further observations regarding this point, upon an animal with very large blood-vessels, so soon after death as to avoid the risk of incipient decomposition. A healthy horse having been killed by pithing, at 11·30 P.M. on the 22d inst. (March 1858), I examined the body just twelve hours later, while it was still warm. The cavity of the thorax, when opened, smelt perfectly fresh. Both auricles contained large masses of coagulum, buffed on their upper surface. There were also soft dark clots in both ventricles, together with a good deal of fluid blood, which, however, scarcely coagulated at all, a considerable portion from the right ventricle yielding, after many minutes, only a minute thread of fibrin. I suspect this was chiefly serum and corpuscles, which had passed in from the auricles on relaxation of the ventricles. There was a considerable amount of firm coagulum in the aorta, and the large veins at the anterior part of the chest were loaded with firm buffed clot. A small branch beneath the pleura, where it is reflected over the pericardium, contained perfectly fluid blood, as also did a coronary vein of the heart, about as large as the saphena of the human thigh; whereas the concomitant artery, which was very large (bigger than the human femoral), had the blood a good deal coagulated. That from the coronary vein having been shed into a saucer, yielded, after some time, threads and lumps of fibrine. An intercostal vein, from beneath the pleura, as big as a crow-quill, furnished fluid blood, which coagulated. The superficial veins of both fore-legs yielded perfectly liquid blood, which began to coagulate in about four minutes, and set into a solid mass. But, just below the axillary, small portions of

rence of coagulum in the heart of the human subject twenty-four hours after death, compared with the universal absence of it in the small veins of healthy parts, so far as I have yet examined them, both in man and the lower animals, I think the fact must be admitted, that where a large mass of blood exists within a cavity of the heart or a blood-vessel, it experiences coagulation sooner than if in a small vessel of the same body. If this be admitted, it becomes a strong argument in favour of the active operation of the tissues, for the blood is more exposed to the influence of the air in a subcutaneous vein than in the heart, and the only conceivable reason for the greater persistence of fluidity in the latter than in the former, is that the influence of the tissues operates to greater advantage upon the smaller mass of blood.

Again, supposing it to be admitted that free ammonia exists within the blood vessels, maintaining the fibrine in solution, a hypothesis which, I confess, appears to me very probable,—granting the ammonia theory, I say, as far as it can possibly be granted, it is clear that no merely neutral action of the tissues could check the evolution of the alkali in the manner above described; and nothing can tend to convince us more of the potency of the vital forces than to consider what new powers must be impressed upon the chemically inert constituents of the tissues, in order to enable them securely to chain down the alkaline gas, in spite of its excessive volatility.

There is one other experiment upon the sheep's foot which I do not like to omit mentioning. Having exposed a subcutaneous vein, six hours after the death of the animal, I pressed out the blood from an inch of it, and treated the empty part with caustic ammonia, the adjacent parts being protected by olive oil. When the smell of ammonia had passed off, I let the blood return, and, two or three hours after, found that the portion which had had its vitality destroyed by the ammonia, was full of clot, while the blood in the adjacent parts of the vein was fluid, and coagulated on exposure.[1]

This, however, was not the only result of the application of the ammonia. The surrounding tissues had not been thoroughly protected from its action by the oil, and next morning all the parts on which it had acted were the seat of the most intense congestion, accompanied with exudation of glary matter into the cellular tissue; in fact, there were all the appearances of the most severe inflammation. Some of the exuded matter had trickled down on a board beneath, and had there coagulated, showing that genuine exudation

coagulum made their appearance in the vessels, which here attained a size about equal to the femoral in man; and both the axillary trunks were plugged with firm clot.

[1] Two feet of a sheep, killed six hours before the Society met, were exhibited in illustration. One of these was prepared in the manner described in the text. The portion of vein which had been treated with ammonia, contained a cylindrical coagulum, while the blood in the adjacent parts of the same vessel was fluid. The other foot was for the purpose of showing the fluidity of the blood so many hours after death. A considerable amount having been shed into a saucer in the liquid state, soon assumed the solid form.

of lymph had been the result of this *post mortem* inflammation, then, I believe, for the first time observed in one of the mammalia.[1] I cannot avoid expressing the satisfaction that it has given me to find what I had inferred from other circumstances, in my investigations on inflammation, now established as a matter of observation. I had found that the blood-corpuscles, both red and white, were perfectly free from adhesiveness within the vessels of a healthy part, but that in an inflamed region they stuck together just as they are seen to do between two plates of glass. Having thus observed that the corpuscles of the blood comport themselves in an inflamed part in the same manner as in blood drawn from the body, I inferred that the liquor sanguinis was, in all probability, similarly affected, although coagulation is not observed in the capillaries, in consequence of the movement of the blood; and I gave the same explanation of the speedy coagulation of lymph, and of the formation of clots in inflamed vessels, as has been substantiated by independent facts this evening. In the paper before alluded to, the following passage occurs:—"The non-adhesiveness of the red and white corpuscles, and the fluidity of the blood, seem to be due to one and the same mysterious and wonderful agency,—the tissues of a healthy body appearing to extend over the blood near them, a part of the same influence by which they are themselves protected from the action of chemical affinities tending to their decomposition." We now see that when an agent capable of producing inflammation acts upon a part in which the blood is at rest, coagulation of the blood does really occur in the vessels.

There is an error of observation into which Dr Richardson has unaccountably fallen, which it appears important to correct. In speaking of the coagulation of a portion of blood enclosed between two ligatures in the jugular vein of a dog or cat, he mentions the formation of a large bubble of air within the vessel, a little prior to the occurrence of coagulation. I have frequently seen the pellucid appearance he describes, but find that it is in no way connected with coagulation, but is due to the subsidence of the red corpuscles, leaving a layer of clear liquor sanguinis at the top. If two ligatures be applied, about an inch apart, upon a subcutaneous vein of one of the legs of a cat, care having been taken not to disturb the connections of the vessel, or inflict injury upon it, and the leg be suspended by the paw in the vertical position, the clear appearance will begin to show itself below the upper ligature within five minutes. If now the limb be left for several hours, the skin having been carefully replaced so as to prevent evaporation, the clear colourless upper layer will be found to occupy nearly two-thirds of the length of the portion of vein, and to be sharply defined from the black lower layer which contains all the red corpuscles. If now the upper part be

[1] Tension upon the blood in the vessels, resulting from the bandage, supplied, I imagine, the place of the force of the heart in squeezing out the liquor sanguinis through the walls of the capillaries, deprived of their usual power of retaining it.

punctured, the clear liquor sanguinis will flow out, and coagulate upon any object held to receive it.[1]

Some of the observations above described will have a bearing upon medico-legal inquiries, showing, as they do, that not only ecchymosis, which some have denied, but even inflammation may be developed *post mortem*, provided that the return of blood by the veins is in some way prevented.

There are other bearings, both upon pathology and practice, to which I cannot even allude on the present occasion; but I thought it best to place the facts at once before my professional brethren, confident that they will receive from them all the attention that they may deserve.

In conclusion, I have to express my thanks to my friend, Mr Craig, for the kind and able assistance which he has afforded me throughout this investigation, and also to my friends, Drs Gourlay and Hill, who have on several occasions lent me most valuable aid.

[1] *Post mortem* congestions simulating inflammation, are, I suspect, due to this gravitation of the red corpuscles of the still fluid blood into the vessels of dependent parts.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

ON THE

ACTION OF THE MUSCULAR COAT

OF THE

BRONCHIAL TUBES

IN

RESPIRATION.

BY C. RADCLYFFE HALL, M.D., M.R.C.S.E.,

Fellow of the Royal College of Physicians, Edinburgh; Member of the Royal College of Physicians, London; lately Physician to the Bristol General Hospital; Author of a Treatise on the Ganglionic System of Nerves, &c. &c.

WORCESTER:
PRINTED BY DEIGHTON & CO., JOURNAL OFFICE.
MDCCCL.
PRICE TWO SHILLINGS.

ON THE ACTION OF THE MUSCULAR COAT OF THE BRONCHIAL TUBES IN RESPIRATION,

AND ON THE

EXCITING CAUSE OF INSPIRATION AND OF EXPIRATION.

BY C. RADCLYFFE HALL, M.D., TORQUAY,

Fellow of the Royal College of Physicians, Edinburgh; late Physician to the Bristol General Hospital.

The office which the muscular coat of the bronchial tubes fulfils in respiration is not at present decided. There is little doubt that it tends to prevent over-dilatation of the tubes by reacting against any undue pressure on the part of the air they contain, precisely as the muscular coat of an artery reacts against the blood-wave when forcibly propelled. We may admit also that it reacts partially and spasmodically for the removal of irritating matter from any part of the tubes, as in bronchial cough. But has it, as its discoverer, Reisseisen, considered, any regular and rhythmical action in the movements of the lungs during respiration? Does it relax with each inspiration, and contract with each expiration? Such an opinion is discountenanced by the latest writers on physiology.* To attempt to show that it is not without arguments in its favour is the object of the present communication.

It is observed by Müller, that if the muscular coat of the bronchial tubes acted rhythmically in respiration, it would have to be under the influence of the will, which is not the case; and, moreover, that the purposes supposed to be fulfilled by such rhythmical contraction would be answered equally well by elasticity. Dr. Carpenter remarks, that "it can scarcely be imagined that the bronchial tubes should, by any power of their own, contract and

* Müller, Dr. Carpenter, Dr. John Reid, Dr. Kirkes.

dilate uniformly with the contraction and expansion of the chest, unless their muscles were equally subject with those of the thorax to the influence of the nervous system, which all experiments concur in showing not to be the case."* It is urged, again, that the only contraction to be seen in experimenting on the bronchial tubes is a tonic, regular contraction, which lasts for some time. "The muscular fibres of the bronchial tubes," said Dr. John Reid, "are endowed with that kind of contractility termed *simple contractility*, which manifests itself by more slow and prolonged contractions and relaxations than that of the voluntary muscles and the heart."†

I have, then, to show,—firstly, that it is not necessary that the bronchial muscular coat should be directly under the control of the will for the purpose of taking a regular part in the act of expiration; secondly, that elasticity would not answer the purpose here fulfilled by muscular contractility; thirdly, that it is not proved that the fibres of the bronchial muscular coat are incapable of the alternating form of contraction, *i.e.*, of quick contraction and quick relaxation; fourthly, that the bronchial muscular coat is under the influence of the nerves to an adequate extent.

1. Müller observes, "The glottis is dilated during inspiration, and contracted during expiration. The bronchial tubes are also dilated during inspiration, and contracted during expiration;"‡ and yet immediately afterwards he states, "it is probable that the bronchial tubes and trachea do not contract and dilate rhythmically during the movements of respiration. Had they this power it would be quite an isolated fact; for although the hepatic duct presents rhythmic contractions, they are quite independent of the will, while, if the bronchi contracted and dilated synchronously with the other respiratory movements, their action, like that of the other parts engaged in respiration, must also be subject to the will, and it is highly improbable that the branches of the efferent tube of an internal viscus would be thus under the influence of the will."§ To this we may reply, that the diaphragm, and other respiratory muscles, present rhythmic contractions, which are not "quite independent of the will;" the heart, rhythmic contractions

* "Principles of Human Physiology," 2nd edition, p. 456.
† Art. Respiration, "Cyclopædia of Anatomy and Physiology."
‡ "Physiology," by Baly, vol. i., p. 345. § Ibid, p. 347.

which are quite independent of the will, and therefore that the more or less subordination to the will cannot be the condition on which rhythmic action depends. Moreover, the trachea and bronchi are as much afferent as efferent ducts, and are employed with a constancy and a regularity which distinguish them from any other visceral tubes. It is probable that the muscles of the glottis are only indirectly under the control of the will, and *their* alternating action in respiratory and vocal efforts is admitted. To this extent the muscular fibres of the trachea also are certainly controlled by the will in the production of high vocal notes, and in voluntary coughing. Many involuntary muscles are indirectly influenced by the will through the medium of voluntary muscles. It is not necessary for the tracheal or bronchial muscles to be controlled by the will in any other way than this. By willing to fill the tubes with air until their fibres are on the stretch, or by drawing in particles which irritate the lining membrane, we can at pleasure excite their contraction. By suspending our breath after a moderate expiration we can probably for a short time at pleasure suspend their action.

2. Elasticity opposes any change from its condition when unexcited. It recoils equally from a maximum and from a minimum size. The lessening of the bronchial tubes below their medium size is as much opposed by the elasticity of their walls, as is their dilatation above it. Elasticity tends to maintain their medium calibre, but not either to increase or to lessen this. If, therefore, expiration be at all expedited by lessening of the bore of its tubes, as it undoubtedly must be, something more than mere elasticity is required. In viewing elasticity as equally opposing undue dilatation and undue diminution of the air passages, and as being inoperative so long as the tubes remain of their medium size, we refer to the elasticity of the *walls of the bronchial tubes only*, and to this acting towards or from the centre as regards the tube. Like arteries, the bronchial tubes during health are always kept on the stretch in their length, whatever the amount of expansion or contraction of the lung; and the elasticity of the general parenchyma is never allowed to reduce the lung to the size to which it contracts it on opening the thorax after death. After the fullest possible expiration the general elasticity would reduce the lung still more were it not opposed. Under all circumstances, therefore,

the general elasticity of the lungs, and the *longitudinal* elasticity of the tubes, oppose inspiration and favour expiration. But the bronchial tubes, though on the stretch in the longitudinal direction, are not so in their transverse diameter. The elasticity of their walls cannot lessen the calibre of the tubes any more than it can increase it. What there is of concentric elasticity in the walls of the bronchial tubes is opposed equally to a full expiration and to a full inspiration. In this mode of acting the bronchial tubes precisely resemble arteries. On exposing a small artery, it will contract to half its natural size, in virtue of its muscular coat. It could not do this by its elasticity solely.

The *longitudinal* elasticity of the bronchial tubes can assist expiration merely by drawing the air-cells towards the larger tubes, by the mutual compression of the air-cells which this entails, and by maintaining the internal surface of the tubes smooth and free; not by any such tightening of the circumference of each tube as will diminish its calibre. Yet, to render the expulsion of the breath complete and equable from every portion of lung at the same time, this latter action of uniform lessening of the bore of the tubes is what is chiefly required. Every one has noticed how much more readily an empty sponge is made to fill itself with water than a wet one to empty itself, in consequence of the difficulty of compressing at the same moment every individual portion of it. A like compression of every individual portion of the lung is required for perfect and full expiration. And here the compressing force is placed within the sponge, and lines all its tubes, in the form of a muscular coat.

The structure of the bronchial tubes teaches so much. It is true that the larger bronchi have plates of cartilage, the elasticity of which operates in the transverse direction of the tube; but these are so far from being always kept on the stretch, like the longitudinal elastic fibres, that their principal use is to maintain an open condition of the tubes under all circumstances, by resisting alike concentric and eccentric force. Nor would any considerable lessening of the larger tubes assist expiration. Unless, indeed, it occurred subsequently to progressive lessening of the smaller and more peripheral tubes, it would obstruct the egress of air from the air-cells. If muscular contractility be the agent for lessening the tubes, we find a correspondence with the requirement in the greater

development of the bronchial muscular coat in the smaller tubes; but if elasticity be the agent, this correspondence is wanting; elastic tissues exist in largest proportion in the larger tubes. In the larger tubes we find plates of cartilage, and longitudinal elastic fibres thickly and closely packed. Between these, and separating them, is placed a thin layer of unstriped muscular fibres, which, as they are not firmly attached to the plates of cartilage, can have but little influence over them; and next to the longitudinal fibres, the mucous membrane. In the smaller tubes the cartilages no longer exist; the longitudinal elastic fibres are all placed lengthwise, never across the tube; they branch at their extremities, and intertwine to an extent sufficient to ensure their keeping together, but not sufficient to enable them to either lessen or enlarge the tube in the wall of which they are situated. The circular muscular fibres form a thicker and stronger layer than in the large tubes, external to and surrounding the longitudinal elastic bands. Arrived at the commencement of a vestibular passage, or passage of entrance to several air-cells, the muscular coat ceases abruptly, whilst the longitudinal elastic fibres spread out from under it, and stretching in curved loops over each bunch of air-cells, form the frame-work upon which the thin basement membrane, with its delicate pavement epithelium, is spread to constitute the wall of an air-cell. It results from this arrangement that the openings of the air-cells can never become closed by any action of the muscular coat. Had the muscular fibres been the most internal, their energetic contraction might have closed up (like a sphincter) the door of the vestibule, and instead of dyspnœa, a fit of asthma would have caused asphyxia. As it is, the effect of the contraction of the circular muscular fibres is to press upon the longitudinal elastic bands; these, being always on the stretch, by opposing the pressure, render it uniform and equal throughout the tube; and by their expanding to embrace the air-cells at the point where the termination of the muscular coat leaves a ring of muscle with a free margin, convey the muscular compression on to the walls of the cells, and effectually prevent any ring-like constriction. Thus the channel of the tubes is kept smooth and free, and the openings into the air-cells patent, under every degree of expansion and contraction of the lung.*

* Did space permit, it would be easy to show the bearing which these considerations have on the mechanism of the production of emphysema.

We may conclude that elasticity alone is inoperative in reducing the bore of the air-passages to anything less than its medium condition; that no especial elastic tissue is so arranged as to answer such a purpose; that muscular fibre is so arranged as to answer precisely this purpose, and no other.

3. To ascertain the fact, whether the alternating form of contraction could be excited artificially in the muscular coat of the bronchial tubes or not, the following experiments were made.

Experiment 1.—The trachea and the right vagus nerve were laid bare in a full-grown rabbit. Both needles of an electro-magnetic machine, when applied to the front of the wind-pipe, occasioned general convulsive movements of the body, and spasmodic choking and gasping. Both needles applied to the trunk of the vagus caused similar effects to a still greater degree, shrieking and great pain. The heart beat violently. So far the electricity had evidently been conducted to other tissues by the moist and warm surfaces to which the needles were applied. These effects having subsided, the trachea was divided, and a flexible tube inserted into its lower portion. By directing the free end of this tube against the flame of a spirit lamp, the number, force, and length of the expirations could be readily observed. Galvanism to the vagus nerve quickened respiration, and rendered it tumultuous, disordering the action of the thoracic muscles. The number of expirations was counted for periods of ten seconds. Without galvanism, the expirations were now at the rate of 150 per minute. The right vagus was next isolated over a smooth slip of dry wood, and two slender needles stuck through the nerve into the wood, but not through it, so as to place the intervening portion of nerve in an electric circuit, without the electricity being directly conducted to other animal textures besides the nerve. The nerve was then divided above the wood, near to the base of the skull, so as to prevent any reflex excitement through the root of the vagus. No alteration in the respirations immediately followed this division of one vagus. The galvanic poles were now applied to the needles. The expirations instantly rose to 171, and became more forcible. Without galvanism, the expirations then fell to 72. The other vagus (the left) was next divided in the neck, both nerves being now separated from the brain. With galvanism applied as before, the expirations were at first short, quick, and 138; they then became slower and more

lengthened. In a few minutes they were, without galvanism, 42; with galvanism, 72. The most marked effect of the galvanism upon the expiration, now that this was slower, consisted in the more prolonged, continuous, and forcible expulsion of the air.

The thorax was now opened on the right side, so as to expose the right lung and the right side of the heart. The expirations without galvanism were, 48; with galvanism to the left vagus, 54. No movement whatever could be excited in the exposed right lung, which of course was collapsed, either by galvanism to the right vagus or to the lung itself. The heart ceased to manifest any increase of action from galvanising the nerves in the neck, but contracted with increased vigour and frequency on galvanising the ventricles. The opposite side of the thorax was now opened. Of course respiration instantly ceased, and could not be re-excited by galvanising one vagus, both vagi together, lungs by themselves, or lungs and vagi. No effect now followed the application of galvanism to the bronchial tubes, either whilst the lungs were warm and *in situ*, or after their removal from the chest.

Experiment 2.—The trachea and left vagus were laid bare in a large fowl. Expirations were now 48. On applying galvanism, as in the preceding experiment, to the trachea, vigorous convulsive movements of the whole body, and rapid quivering elevations and depressions of the wind-pipe ensued; so rapid that it could not be ascertained satisfactorily whether the individual rings of the trachea were approximated to each other or not. The trachea was then cut through, a tube inserted, and galvanism applied to the lower portion. This affected equally the upper and the lower segment of the wind-pipe, proving that the electricity was conducted by the warm, moist muscles on which both portions of wind-pipe rested. The left vagus was now isolated over a slip of wood. Expirations without galvanism, 48; with galvanism to the nerve, 72. With galvanism to the trachea only, expirations 54, and general struggling. With galvanism applied again to the left vagus, expirations 60; without galvanism, 48.

The vagus was not cut through in this experiment.

After death, galvanism was immediately applied to the heart, stomach, trachea, bronchi, and substance of lung, but produced no effect.

Experiment 3.—A large rat was held under water. It was 80 seconds in drowning. The rat was taken out of the water, and its trachea was divided. A tube was fastened into the lower portion of the trachea, and its orifice was held under water at some depth. On opening the front of the chest, the pressure of the water in the tube prevented the egress of the air from the lungs, which therefore did not collapse. Galvanism was then applied to both vagi at once, and caused immediate, sudden bubbling of air from the mouth of the tube under water. After this, no effect. The tube was then emptied of water, and the lungs were fully inflated through it, and the tube again submerged. According as the depth of the tube in the water permitted the resiliency of the lungs to operate or not, the air bubbled out spontaneously, but no further action could be elicited by galvanism applied to the vagi, to the trachea, or to the substance of the lungs.

Experiment 4.—Both lungs were exposed in a large frog. They rose and fell with each pulsation of the heart, but had no other movement. Mechanical irritation of their surface induced no contraction in the wall of their transparent honey-combed lung-sacs. When artificially over-inflated through the glottis, the lungs would return to their medium size, expelling the superabundant air, but this resulted from elastic recoil merely. Mechanical irritation of the vagi had no perceptible effect.

Examined with a half-inch power, whilst the animal was alive, the blood-globules were seen to stream through the vessels of the diaphanous walls of the lungs with great but very irregular rapidity. When the lung recoiled after its pulsational elevation, a momentary check occurred in the race of the blood-globules. In the larger vessels, this check caused the globules to oscillate; but in the capillaries it occasioned merely a more tardy rate of travel. If, however, the capillaries farther on had become impacted with globules, so as to become impervious, then the nearer capillaries leading to them also presented oscillation of the blood-globules. So long, therefore, as the whole net of capillaries is free, the course of the blood through them is regular and uniform in passing on, but not quite uniform as to rate of travel, being quickened by each contraction of the heart; whereas in the vessels larger than the capillaries the blood is not uniform and regular in passing on, but

gushes in a to-and-fro tidal stream, advancing more than it recedes with each systole of the heart. To equalize the stream and convert an intermittent into a steady and constant pressure, both the muscular and elastic properties of the coats of the arteries are doubtless called into action. This observation appears to answer in the negative a query, propounded by Dr. Charles Bell, viz., whether the actions of the capillaries in general do not receive a constantly repeated stimulus from the alternate flux and reflux of the blood-wave. It also demonstrates that the capillaries will be always equally full in a state of health, provided the circulation through them be unobstructed; but that their fulness will instantly become augmented provided any obstruction occur.

The check in the blood-wave in the smaller vessels (not capillaries) immediately preceded the pulsation of the heart, and accompanied the slight falling of the lung from its pulsational elevation. In point of time, it occupied about one part out of five. In the capillaries, the blood-globules were noticed to pass across the field in single file, each globule becoming elongated and somewhat dumb-bell shaped as it passed through a small capillary, and resuming its ovoid globular outline on entering a larger vessel. The walls of these small capillaries could not be seen at the focus employed.

The observation was continued for three hours. As the lung became dry from exposure, and the heart's contractions more languid, the lung appeared more and more congested to the naked eye. Under the microscope this congestion was found to consist in mere impaction of globules in the vessels, not in extravasation of globules or of *liquor sanguinis*, so far as could be made out. It does not follow that such would be the case in a similar state of congestion of the lungs in the living body, warm and moist.

Experiment 5.—On exposing the lungs in a frog, they burst out and expanded by successive deglutitions of air to their maximum size. No appearance of anything like muscular reaction of the lung followed each gulp of air. On the jaws being held separate, the frog made a twisting movement, and the lungs immediately collapsed. As soon as the lungs had again become filled with air, galvanism was applied to both lungs at once. It occasioned general struggling and instantaneous emptying of the lung-sacs. Allowed

to become filled with air again, galvanism was applied to one of the lungs only: no effect.

Experiment 6.—Three frogs. It was tried to induce collapse of the lungs by galvanism. This effect always ensued when both lungs were galvanised together, so that the current of electricity passed across the body of the frog, and excited convulsive movements, but never so long as the galvanism was restricted to the walls of one lung. Galvanising the vagi had no perceptible effect. As in the preceding experiment, the lungs rose and fell with each pulsation of the heart. In one particular a correspondence was noted between the state of the circulation and the amount of pulsational elevation. The circulation was freest, and the rise of lung greatest, when the distension of the lung with air was to a medium extent. If the lungs were distended to the utmost, pulsational elevation was scarcely noticeable. If almost empty, the same. Under the microscope, the difference in rate of travel and in freedom of passage of the blood-globules, in accordance with the amount of distension of the lung with air, was equally well marked. When the lung was greatly distended the circulation through the capillaries was almost at a stand. When the lung was almost empty many capillaries were obstructed by the pressure of adjoining portions of the collapsing walls, and the circulation in others was more tardy. But when the lung was moderately full of air, the circulation was most free, most equal, and most rapid. It could not be made out whether the rate of capillary circulation was influenced by the purity of the air contained in the lung, that is, whether circulation was faster immediately after the ingestion of fresh air than after the air has remained in the lung-sac for some time, or the reverse.

It will be observed, in passing, that we have here by analogy a demonstration to the eye, of what experience has long since taught, that moderate exercise of the lungs promotes freedom of circulation through their blood-vessels, whilst inactivity of the lungs, and over-distension of them with air, equally oppose freedom of circulation, and thereby favour the production of congestion, or the augmentation of any which may already exist.

Experiment 7.—Both lung-sacs were removed from a large frog.

The animal lived for two hours, making frequent inspiration-like movements of deglutition.

Experiment 8.—Galvanism was applied to the exposed surface of the heart in three frogs. At first the pulsations were increased in frequency, maintaining their proper rhythm. Presently, the ventricles contracted slowly, until they were squeezed tight and firm, without any attempt at dilatation interrupting the contraction. They remained in this state for from 8 to 15 minutes, the galvanism being taken away. They then gradually relaxed, admitted blood from the auricles, and proceeded to pulsate rhythmically again. Galvanism, again applied, induced the tonic contraction as before. In two of the frogs this was done twice, in the third three times, after which the heart remained contracted and motionless, and refused any further manifestation of activity.

In this artificially induced persistent spasm of the heart in the frog, have we not the type of the cause of death in angina pectoris, and perhaps also in certain cases of suddenly fatal epilepsy?

We learn from these experiments, that the expirations can be notably quickened and rendered more forcible in birds and mammalia by galvanising the vagi; that in the frog, galvanism applied to the lung alone has no effect, but that if conducted to the spinal cord, so as to excite general movements and opening of the glottis, it then indirectly affects the lungs by opening their entrance-passage, and allowing their elasticity to expel the air they contain; that experiment thus confirms anatomical examination in deciding that there is no muscular tissue in the lungs of Batrachian reptiles; and that a muscle whose normal duty it is to contract in the alternating manner, viz., the heart, may be made, by over-stimulation, to assume the persistent tonic form of contraction.

The capability of the bronchial muscular coat to undergo the alternating contraction, which it must if it alternately contract and relax with every expiration and inspiration, is not demonstrated to the eye, but it seems to be all but proved inferentially. The general muscles of respiration were excited by galvanism applied to the vagi, even when these were prevented by division from acting as excitors through the medulla oblongata, and when precaution was taken to prevent the electricity from being conducted away from the nerves. We must assume that the galvanised vagi acted

centrifugally, and produced an impression of some kind on the terminations of the cerebro-spinal nerves distributed in the lungs, and induced through them excito-motory action of the respiratory muscles at large. What proof, then, do the experiments 1 and 2 furnish that the bronchial muscular coat was stimulated at all, since the excitement of the general respiratory muscles is enough to account for the increased frequency of the respirations? It will not, however, explain why the effect should be manifested chiefly by expiration, rather than by *in*spiration. Under ordinary circumstances, any interference with breathing is first and principally shown by the inspiratory act, whilst in these experiments it was noticeably the expiratory part of respiration that was made more forcible by galvanising the vagi. I infer that the bronchial muscular coat was stimulated to contract, and that it did so rhythmically after its normal fashion, and that it was in consequence of the increased rapidity of its contractions thus induced that the expirations were increased in force, suddenness, and frequency. If it be objected that the bronchial muscular coat has never been seen to present any other than tonic contraction when exposed to view, we may reply that the conditions are then so opposite to the natural ones under which breathing is performed, that we have no right to decide what the muscle *cannot* do during health from what it *will* do when exposed to sight in a cooled and collapsed lung. In experiments on the stomach, Longet proved that the state of fulness or emptiness of the organ modifies to the greatest extent the kind and degree of movement excited by stimulating the par vagum. So may the emptiness of the bronchial tubes, and the removal of the general elasticity which is always operating in the normal condition, prevent the bronchial muscular coat from acting in its normal manner. In the preceding experiments, left undisturbed and unexposed, and in every respect, excepting in the application of a stimulus to its nerves, placed under its natural conditions, we have reason to expect that the bronchial muscular coat, if excited at all, would show that excitement by an increase of its normal mode of acting. Dr. C. B. Williams and other observers have established the fact of the contractility of the bronchial tubes, and Volkmann has found that this contractility is excited by galvanising the vagus. If, then, contraction were excited in the above experiments, it could not have been the tonic form of contraction. Nothing but alternate

contraction and relaxation will account for the augmented force and frequency of the expirations. Nor is the opposite view strengthened by the occurrence of tonic contraction (*i.e.*, spasm) of the bronchial tubes in nervous asthma. Here, the exciting cause is so intense that it may affect the bronchial muscular coat precisely as we have seen that over-galvanising will affect the heart in the frog, *i.e.*, it may induce spasm in a muscle which, under gentler and more normal excitement, would perhaps (like the heart) present the alternating kind of contraction. At all events, we can no more suppose that so inconvenient and distressing a condition as that of asthma represents the ordinary and healthy mode of contraction of the bronchial muscular coat, than we can infer the ordinary mode of action of voluntary muscles from the tonic rigidity of tetanus, or that of the heart from the tonic spasm of angina pectoris. And, admitting that spasmodic asthma furnishes an illustration of tonic contraction of the bronchial tubes, have we not an equally strong pathological illustration of alternating contraction, or clonic spasm, in the repeated convulsive expirations of whooping cough, and in certain varieties of hysterical cough?

4. The last objection, viz., that the bronchial muscular coat is not sufficiently under the influence of the nerves of respiration, provided my interpretation of the experiments adduced be correct, is no longer tenable.

Having endeavoured to dispose of the objections advanced against the theory of Reisseissen, I proceed to consider what may be stated in its support.

A strong argument by analogy is deducible from the fish, in which the normal movements of the respiratory organs may be watched. Respiration in the fish consists in the deglutition of water, as in the batrachian it consists in the deglutition of air. There is, therefore, no distinct inspiration and expiration. Still the process is divided into a stage of ingress, with open mouth and closed gills, and one of egress, with closed mouth and open gills; and the two acts are performed in rhythmical order, like the inspiration and expiration of lung animals. The hyoid apparatus acts as a piston to the common cavity of the mouth, pharynx, and gills. By its retraction when the gill outlet is shut, water is sucked through the open mouth. The muscles which retract the hyoid arch relaxing, the mouth is closed, the arch advances, the opercular covers press down against

the expanded gills, the branchiostegal valvular folds which closed the gill outlet are tucked up, and the water is driven out. Between the two rows of fringe-like processes which each branchial arch usually presents, a plane of well-developed muscular fibres exists. Judging from its anatomical connexions, this interbranchial muscle will move the two rows of gill processes upon each other, approximate them when separated, and compress the gill processes altogether against the inner boundary of the branchial chamber. Supposing that such muscular action were to operate during the ingress of the water, it would prevent the free access of the fluid to the surface of the vascular branchial fringes, and defeat the object intended. But supposing the gill processes to be separated to receive the influx of water, the interbranchial muscles being relaxed, the contraction of these muscles during the subsequent stage of egress of the water will expel the fluid from between the branchial fringes, and thus materially assist in emptying the recesses of the gill-chamber, and also by squeezing the water through the vascular fringes, bring the blood of the fish into closest contact with the aërating medium. May we not fairly assume that the interbranchial muscle of the fish corresponds to the bronchial muscle of the mammal; that the act of expelling the water from the gills corresponds to the act of expiration; and that the interbranchial muscle operates as an expiratory agent? It may be that there is a further analogy. Any particles of food or of other foreign matter which, having escaped the sieve-like sensitive valves placed at the pharyngeal openings of the gills, may have insinuated themselves between the vascular membranes of the branchial processes, will here excite irritation and excito-motory contraction of the interbranchial muscles to an extent sufficient to produce the ejection of the irritating particles into the sphere of the large current of water passing through the gills. If so, the process of expulsion will be the analogue of the cough of mammalia.—The expansion and closure of the gills and gill-fringes may be readily observed in the common gold-fish in a globe of water.

Granting the analogy, let us trace its consequences. The interbranchial muscle must ordinarily act in unison with the other muscles of expiration. Were it to hold the gill processes in close approximation during the act of inspiration,—so to term the stage of ingress of water,—it would prevent the application of the water

which is to purify the blood in the membrane which it bathes; on the other hand, were it to fail to contract after the water between the separated gill processes had become depraved by fulfilling its office, it would prevent a sufficient change of water at the next inspiration, and would perhaps allow the closing gill-covers to press together portions of the bronchial processes irregularly and unequally. Again, we cannot suppose the interbranchial muscles to exist merely for an occasional purpose but rarely required, and not to fulfil a constant purpose, for which their arrangement admirably adapts them. I assume, therefore, that they act rhythmically with every closure of the gill-covers, *i.e.*, with every act of expiration. In what does this rhythmical action consist? Merely in their fibres contracting every time they are stretched, ceasing their contraction as soon as they have effected it. And if we deny this we fall into a conclusion far more improbable, viz., that by elasticity and the action of the hyoid and opercular muscles, the gill processes are alternately opened and shut, separated and approximated, and yet that an extensive layer of muscle placed between each row of branchial fringes, during all this constantly recurring movement of the parts to which it is attached, remains passive. So, in mammalia, it is admitted that during every inspiration the glottis, trachea, and bronchial tubes dilate and widen, and during expiration contract and lessen. Unless, then, the tracheal and bronchial muscles do rhythmically contract with each expiration, and relax with each inspiration, we are forced to conclude that they are alike unaffected by stretching and shortening of their fibres; a conclusion opposed to the ascertained attributes of both striped and smooth muscular fibres.

The rhythmical contraction of the bronchial tubes is conformable with what we know of the physiology of respiration in general. How is it excited, directed, and controlled? The rhythm of the bronchial tubes must coincide with that of the respiratory muscles at large. We cannot voluntarily alter the rhythm of respiration, without wilfully affecting the external conditions of respiration, either by modifying the action of the muscles, the quantity and quality of the air, or in some other way the relation of the blood to the lungs. And if these external conditions be altered, respiration is altered, whether we will or not. Hence the rhythm of respiration depends more on external conditions than on the nervous

centre itself. The nervous centre is the governor of the process, but not the cause of it, and in its government it is largely dependent on the conduction of impressions from the acting organs.

We must seek, then, for the exciting causes of the rhythmical action of the respiratory muscles, not in the medulla oblongata, which is merely the controlling power, but in the external conditions which excite in succession the acts of inspiration and expiration. Respiration, as a double excito-motory act, requires an excitor for inspiration, and an excitor for expiration, so arranged that they shall alternate in their application, and consequently in their effect. This constitutes the rhythm. "What," said Dr. John Reid, "are the excitations which lead to the performance of the muscular movements of expiration? Do the same excitations that occasion the muscular movements of inspiration operate in the production of the expiration which immediately follows, so that they are to be considered two stages of the one and same muscular action? These are questions which we are not prepared to answer."* In most works on physiology, the exciting cause of expiration has not been made a subject of consideration, whilst many and various conditions have been assigned as the ordinary exciting cause of inspiration. Of these may be enumerated, the presence of carbonized blood in the vessels of the lungs; of carbonic acid in the air-cells; and of carbonized blood in the capillaries of the system at large, either superadded to one or both of the former conditions, or constituting in itself the exciting cause of the act of inspiration under ordinary circumstances.

Stating them as mere postulates, there are some grounds for considering that the ordinary exciting cause of inspiration is distension of the capillaries of the lungs with carbonized blood; and the exciting cause of expiration, the presence in the air-cells and bronchial tubes of unduly carbonized air.

That distension of the pulmonic capillaries with carbonized blood, and not the presence of carbonic acid in the air-cells, is the ordinary exciting cause of the act of inspiration, is presumable from the following considerations. In the fish, the sensation which excites the taking into the mouth of fresh water cannot depend on the presence of carbonized water in the gill-processes, since the gills have just been emptied of the used water. The presence in

* Loc. citat.

the branchial capillaries of venous blood requiring oxygenation is the only adequate condition we can find. In mammalia, the desire for inspiration immediately succeeds to the expulsion of the impure breath, at the moment when the lungs must contain the smallest proportion of carbonized air. The most urgent distress for fresh air attends disease in man, in which pulmonic engorgement prevents the access of air at all to a great portion of the lungs, or in which a sticky secretion clogs the air-cells and their vestibules. Here the desire for breath is greatest, whilst the amount of foul air in the air-cells, because of the absence of *all* air, is least; but the capillaries are gorged with venous blood, which cannot part with its pulmonic excretion. The presence of unduly carbonized blood in the capillaries of any part of the system, at all events, its presence in the medulla oblongata appears capable of exciting an inspiratory act, independently of the state of the lungs, as in the frog from which the lungs had been removed. If the like condition of the pulmonic capillaries be the ordinary excitor, we have a similarity in the conditions which act as excitors of inspiration in different parts of the system. But what proof have we, that under ordinary circumstances the state of the lungs has anything more to do in exciting the desire for breath, than that of any other part of the body? It would be strange if the instinct which points to the chest as the seat of the earliest feeling of desire for breath were altogether wrong in its indication. By division of one vagus the number of respirations is dropped about one-fourth. By division of both vagi at once the respirations are dropped one-half. I see no other mode of accounting for this than by considering that one great normal source of excitation of inspiration (viz., the lungs,) has had some of the wires of its electric telegraph to its nervous centre cut off, and that it can therefore send only an imperfect message to demand supplies. In this we see an illustration of the law that every reflex act is considerable in proportion to the sum of the excitement set up in the nervous centre, and this usually is in direct proportion to the number of nerve-fibres by which the excitation is conveyed. Cut through half of the excitor nerve-fibres, and other circumstances the same, the excitation is reduced by one-half, and the resulting movements become only half as forcible, or half as frequent. Now, did the lungs only share with the rest of the body in exciting the

sensation of want of breath, division of all their nerves (and the vagi do not constitute half of their nerves), would not produce the full effect which ensues from division of the eighth nerves. Conversely, through what other nerves can we quicken respiration in the same manner that it was quickened by galvanising the vagi in the experiments I have narrated? The vagi are the chief, though not the only excitor nerves proper to the lungs; as their irritation accelerates the frequency of the respirations, and their division greatly retards it, it seems to be proved that the lungs are greatly concerned in setting up the sense of desire for breath.

The following considerations support the opinion that the presence of carbonized air in the smaller air-passages and air-cells is the exciting cause of expiration. In the fish, the water is expelled from the gills as soon as it has received the excretion of the branchial membrane, *i.e.*, expiration occurs as soon as the water has become carbonized. In batrachian reptiles the air is expelled from the pulmonic bags after it has remained for an uncertain period undergoing admixture with the pulmonic excretion. In mammals, the desire to expire arises as soon as the fresh air inspired has had time to become depraved. The desire for expiration is intense in direct proportion to the length of time that breathing has been suspended, and consequently in direct proportion to the degree in which the air contained in the lungs has become impure, excepting only when the lungs happened to be contracted below their medium size when the suspension of breathing was commenced. Carbonic acid when inhaled induces closure of the glottis. It is a fair deduction that it would cause contraction of the tracheal and bronchial muscles if applied in strength to the membrane which covers them. If the carbonic acid of the breath so affect the bronchial muscular coat, as the contraction of that coat can only participate in the expiratory part of respiration, it follows that the presence of carbonic acid in the air-passages tends to excite one link in the chain of expiratory actions.

Is the effort of the bronchial muscular coat to contract one source of that sense of constriction in the chest which is felt on holding the breath too long after a full inspiration? If so, the sensation of muscular tension in the air-passages may assist in rendering the desire to expire air from the distended lungs more urgent. But this muscular tension is certainly not the first nor

the chief excitor of the act of expiration. Reptiles expire air without having any muscular texture in the walls of their lungs. Partly from this cause, but chiefly from their being cold-blooded animals, reptiles can hold their breath for a long and uncertain time. Having to remain under water for irregular periods, they would be only incommoded by possessing muscular lungs, which would tend to contract on the contained air as soon as this had become charged with carbonic acid. Mammals, on the other hand, require purer blood, and cannot expel carbonized air from their lungs too rapidly, or replace it too completely. Hence they possess muscular lungs.* When a rat drowns, bubbles of expired breath make their appearance in 30 or 40 seconds, whether the animal struggles or not. When a frog is submerged, bubbles of expired air may not rise for a quarter or half-an-hour, or they may rise immediately, just as the animal struggles or remains quiet. I infer that in the rat, the bronchial muscular coat contracts, and compels some air to escape; but in the frog, the lung-sacs having no muscular fibre to become excited, expel the air only when an accidental opening of the glottis permits its escape.

The non-existence of muscle in the lungs of the batrachian, where respiration is not regularly rhythmical, and its existence in those of mammalia, where respiration is regularly rhythmical, points out that the contraction of the bronchial muscle is at least not opposed to rhythmical action.

It has been ascertained that a stream of carbonic acid checks circulation in the capillaries. If the presence of carbonic acid in the air-passages thus checks circulation in the capillaries of the lungs, it of course tends to produce the condition of vascular fulness, assumed to be the exciting cause of inspiration. In this case the carbonized air will not only excite contraction of the bronchial tubes and other concomitant acts of expiration, but will also favour the occurrence of the next act of inspiration, by inducing the commencement of that fulness of the capillaries which, increasing, excites inspiration. Occasionally, indeed, it would seem as if

* Cetaceans can remain below water for a much longer period than other mammals, and yet possess muscular bronchi, but their normal frequency of respiration (in the whale) is not quicker than 5 or 6 per minute, and there is special provision made in the superficial position of the larger bronchial tubes, as well as in the extensive retiform blood-sinuses, for preventing distress.

the two excitations were in operation together, and that, either, one prevailed because it became the stronger of the two, or else because volition interfered. Thus, after a moderate expiration, if a man holds his breath as long as possible, the immediate desire will be for inspiring. Here there are carbonized air to excite expiration, and capillary distension to excite inspiration. As the lung is but imperfectly filled, the latter condition predominates in intensity. The bronchial tubes having left off with expiration, are kept below their medium size; their contractility is tired out, and their elasticity tends to enlarge them up to their medium calibre. On every account the tendency is towards inspiration. But let the breath be held after a full inspiration, and the conditions are reversed. The expanded bronchial tubes tend to return by their elasticity down to the medium calibre, their stretched and specifically excited muscular coat tends to contract, the tubes and cells filled with air, which has become impure, occasion intense desire for expiration, and expiration ensues. If the breath be held at a medium amount of expansion of the chest, so that the conditions are evenly balanced, the subsequent act is that of inspiration or of expiration indiscriminately. By an effort of will we may interfere with these tendencies, because the voluntary movements of respiration are stronger than the automatic. By a strong effort, for example, after holding a full, but not the fullest inspiration as long as is comfortable, we may draw in a little more air with momentary relief, but our feelings prove that we are thwarting nature. The automatic arrangements are tending towards expiration, and expiration must follow almost instantly.

Thus, inspiration necessarily leads to the condition which is the exciting cause of expiration; and expiration necessarily leads to that which induces inspiration.

All tubular muscles appear to respond directly, without the necessary intervention of any nervous centre to the mechanical stimulus of distension; and all are paralysed by over-distension. It is doubtful with respect to the bronchial muscular coat, whether any amount of inspiratory force can call forth this characteristic. The inward rush of air finds so ready a diffusion amongst that which fills the air-cells that these more yielding structures are the parts to suffer from excessive force of inspirations, and intervesicular emphysema, not burst, or dilated, or palsied bronchial tubes is the

result. But if the contractility of the tubes be never excited by force of inspiration, the expansion by heat of air shut in by closure of the glottis may increase the tension of the tubes as well as of the air-cells, and this mechanical distension may excite the contractility of the muscular tubes, in addition to the other exciting cause of expiration, viz., the presence of carbonized air. I find, with the spirometer, that a very slow and protracted expiration after a full inspiration does not raise the cylinder so high as a more speedy expiration. In other words, there is more breath left behind in the lungs. Are the bronchial tubes tired of keeping up their contraction until all the reserve air is expelled? Of course, the vascular fulness becoming greater in proportion to the time occupied by the experiment, may render the desire to inspire irresistible before all the reserve air has been expired.

Under whatever circumstances, if mechanical distension of the bronchial tubes do influence their contractility at all, it will always operate on the side of expiration. Still any such influence is quite subordinate to that exercised by the presence in the air-cells of carbonized air.

After a full inspiration, I can hold my breath for a period represented by 4. Here there is the least carbonic acid relatively to the amount of air in the lungs, but the greatest mechanical distension of the air-tubes. After a moderate inspiration, for $1\frac{3}{4}$; after a complete expiration, for only 1. Here is the largest relative amount of carbonic acid, but the least muscular distension —there is rather muscular contraction—of the bronchial tubes. Provided a very full inspiration immediately preceded the last trial, so as to freshen up the residual air in the lungs, the time during which suspension of breath could be maintained was increased one-half. Here the degree of dilatation or of contraction of the bronchial muscular coat was the same as in the third trial, but the purity of breath was greater, and the period of its suspension was consequently greater also.

We must conclude, that although the state of dilatation of the air-passages which immediately precedes the commencement of expiration may, by stimulating the stretched muscular fibres to contract, co-operate as a secondary and subordinate cause of the expiratory act, yet the carbonization of the air in the lungs is the essential and primary exciting cause of expiration, and with the

degree to which the breath has become carbonized the desire to expire air directly corresponds.

The residual air left in the air-cells after a complete expiration is probably always more or less carbonized. It is fit for use when *minus* carbon; unfit when *plus* carbon; but some amount of carbon it must always contain. Consequently, it must either be the condition of *plus* carbon only which excites expiration, or otherwise the exciting cause does not make its impression in the air-cells, but solely in the tubes to which common pure air does reach with every inspiration. The exciting impression is probably made in both places, but the question cannot yet be determined.

Inspiration has many other exciting causes besides the state of fulness of the pulmonic capillaries with venous blood, which is here assigned as its ordinary cause; such are impressions suddenly made on any part of the skin or of the mucous surfaces, acute sensations, and mental emotion. But if the view here espoused be correct, expiration has no other primary exciting cause besides the presence of air in the lungs charged with the pulmonic excretion. A frog, with its lungs removed, makes *in*spiratory efforts of breathing only. When the will does not interfere, the spontaneous effort of breathing made by a person struck, or surprised, is one of inspiration. As inspiration is a necessary preliminary to expiration, —as expiration is necessarily excited by air when inspired and used in the lungs, to render the act of inspiration liable to several different modes of excitation, is indirectly to confer the same privilege on expiration, although the proximate exciting cause of the two acts differs.

It is an advantage resulting from this ready and instinctive excitability of inspiration, that the body is instantly fitted for powerful muscular action by the very stimulus which renders that action necessary. For all active efforts a tolerably full and fixed chest is required as a fulcrum from which the muscles may act. Were the effort instantly excited by sudden sense of danger to be one of expiration, the body would be rendered mechanically as powerless by the emptying of the chest as it often is mentally by fear.

To recapitulate,—There seems reason to admit,

1. That, by their general elasticity, the lungs are always kept on the stretch, and tend to contract to a smaller size than is ever permitted during life. This, under all circumstances, is favourable to expiration.

2. That by the elasticity, which acts transversely in the walls of the air-passages, the tubes are accommodated to the amount of air admitted into, or contained in them; but that, for enlarging the tubes for inspiration, and for lessening the tubes below their medium size for complete expiration, this elasticity is useless.

3. That the muscular contractility of the bronchial tubes, whilst, like the transverse elasticity, it resists over-dilatation during full inspiration, *can* reduce the tubes to less than the medium size, as is required in forced expiration.

4. That, inasmuch as forced expirations always alternate with inspirations, the contraction of the bronchial muscular coat must here be of the alternating kind, and not of the tonic persistent kind noticed in certain experiments.

5. That, if the bronchial muscular coat can thus contract rhythmically in forced expiration, there is no reason for denying that it so acts, to a less extent, in ordinary breathing. Considering, indeed, the constant interruption to quiet breathing in the daily occupations of every man, from exercise of the voluntary muscles generally, and of the voice, from variations of temperature, and from mental causes, it is little probable that any part of the machinery required for forced respiration is quite unemployed during ordinary breathing.

6. That, as far as our knowledge will permit, the objections to such a view have been shown to be without sufficient foundation.

7. That the exciting cause of inspiration in ordinary breathing is fulness of the capillaries of the lungs with carbonised blood, but that there are many other auxiliary and occasional exciting causes of inspiration.

8. That the sole and exclusive exciting cause of expiration is the presence of unduly carbonized air in the lungs.

In conclusion, these views, if correct, are not devoid of practical interest. For an application of a portion of them, I must refer to the ingenious remarks of Dr. C. B. Williams, under the head of "Asthma, Paralytic Dyspnœa, and Dilatation of the Bronchi."*

* "Library of Medicine," vol. iii., from which I must quote the following, as more explicitly disclosing Dr. Williams's own view than any other passage I have met with. "It is probable that the contraction of the circular fibres of the bronchi, excited by a certain degree of foulness of the air that is within them, is an essential part of natural expiration."—p. 90.

The mere existence of the muscular elastic and cartilaginous textures which enter into the structure of the bronchial tubes, will dispose us to admit the influence of Gout and Rheumatism on bronchial affections. And the admission, that the bronchial muscular coat co-operates in every expiration, supplies us with an additional reason, were any wanting, for endeavouring to render bronchitic attacks as short as their naturally tedious course will permit, and for taking every care to prevent relapses in a very relapsing disorder. Seeing that the effect of inflammation is, first to paralyze, then to soften and destroy muscular fibre, and to spoil elastic tissue, the effect of bronchitis in producing defective expiratory power, local dilatations of the bronchial tubes, or vesicular emphysema, is susceptible of explanation on merely mechanical principles. So also may fatty degeneration of any part of the bronchial muscular coat give rise to local dilatation, by furnishing a weak and yielding spot in an elsewhere contracting tube.*

* As Mr. Rainey has proved with respect to fatty degeneration of the walls of the air-cells in (one form of) vesicular emphysema.

OBSERVATIONS

WITH

HUTCHINSON'S SPIROMETER.

BY

C. RADCLYFFE HALL, M.D., TORQUAY;

Fellow of the Royal College of Physicians, Edinburgh; late Physician to the Bristol General Hospital.

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 $\frac{1}{100}$ for every degree Fah.

* Cyclopædia of Anatomy and Physiology, Art., "Thorax;" also, an Epitome of Dr. Hutchinson's Paper, from Medico-Chirurgical Transactions for 1845.

† Table V., Art. "Thorax;" Cyclopædia 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$\frac{3}{10}$ 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$\frac{7}{10}$ 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$\frac{3}{10}$

OBSERVATIONS

WITH

HUTCHINSON'S SPIROMETER.

BY

C. RADCLYFFE HALL, M.D., TORQUAY;

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, EDINBURGH; LATE PHYSICIAN TO THE BRISTOL GENERAL HOSPITAL.

Reprinted from the "Transactions" of the Provincial Medical and Surgical Association.

WORCESTER:
PRINTED BY DEIGHTON AND CO., JOURNAL OFFICE.

MDCCCLI.

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 $32\frac{3}{10}$ 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 less than standard vital capacity at same stature	Deficiency of chest in vital capacity	Remarks
				Minimum	Medium	Maxm.			
Ft.	In.	Cub. in.	In.	Inches	Inches	Inches	Inches	Cub. in.	
5	6	6	34	32½	37½	40½	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	29	37	33½	38½	42½	39	45	Ditto.
,,	,,	10	35	...	...	...	...	...	Ditto.
5	9	4	37	34½	39½	42½	42	38	Ditto.
,,	,,	12	36	...	...	...	...	...	Ditto.
,,	,,	2	38	...	...	...	...	...	Ditto.
5	10	12	38	34½	39½	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	35½	40½	44½	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 Cyclopœdia 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 $2\frac{5}{10}$ 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 $2\frac{5}{10}$ to eight. For example, a healthy boy, four feet three inches in height at nine years of age, having a thoracic mobility of $2\frac{1}{4}$ 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. Paternal 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. Over-drugging leads to homœopathy; neglect of the use of water to hydropathy; intemperance to teetotalism. 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	Houseplasterer	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	39	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	4		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 6	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.

Royal Institution of Great Britain.

WEEKLY EVENING MEETING,

Friday, April 8, 1859.

Sir Henry Holland, Bart. M.D. F.R.S. in the Chair.

James Paget, Esq. F.R.S.

On the Chronometry of Life.

The design of the discourse was, to illustrate the law that the processes of organic life are regulated with a regard to time, as exact as that which is observed by them in respect of size and weight and quantity of material employed in them; and to show that such an observance of time is characteristic of life, depending essentially on properties inherent in the living bodies themselves, and not on conditions external to them.

Laws indicating the limitation of the organic formative processes, in respect of quantity, are evident in the facts that, in the ordinary conditions in which each living being is found, it and all its parts have appropriate size and weight and mutual proportion. These may, indeed, be modified by the variations of external conditions, or by events that are of the nature of accidents: but the range of possible variations is, in nearly all cases, comparatively narrow; and the boundaries are soon reached, in which changes of external conditions become incompatible with life.

An instance of a corresponding limitation of the organic processes in regard to time might be noted in the natural duration of each creature's life. It is, indeed, not possible to assign any exact number of hours, days, or years, as the constant limit of life in any species; but it is enough to prove a law of time, as limiting the total duration of the organic processes in each, when we see that, in man, and in other species, the length of life, when not diminished by disease or violence, is as fixed as the natural weight or stature is, and that the term of life is marked by changes whose source is inherent in the living body. Watching these changes in the senile degenerations of the human body, it is evident that life does not cease, naturally, because of any change in the external conditions of living; and that the body is not, with advancing years, gradually worn out, as if there were a gradual consumption of a store of material or of force; but that, as, at a set time, the development of the body ceases and growth goes on,

and then growth ceases and the body is only maintained in its perfection, so, after a time of such maintenance, the method of the formative processes in the body changes, it slowly degenerates, and through degeneracy dies. And all these stages are, alike, natural, constant, timely; all, too, are together, characteristic of life; there is no such succession of events to be traced in any form of dead matter.

Observance of time may, again, be noted in the formative processes concerned in any of the organs whose changes mark the divisions of a life into its chief periods; *e.g.*, in the teeth. Considering merely the conditions in which the teeth of the first set are placed before they project from the gums, there appears no reason why one should be cut before the other, or why they should not all grow with equal speed. Yet while they all grow alike in regard of structure and composition, they have very different rules in regard to the time-rate of their formation. And a yet more marked instance of time-regulation is in the contrast of the teeth of the first set with those of the second. In all essential characters, except those of strength and size, the two sets are much alike; yet there is the widest difference in the rates at which they are formed, and in their duration. The second teeth require as many years for their formation as the first require months; the first live but a few years, the second should live as long as the rest of the body, and sometimes do so. Now there appears nothing to which, as to an efficient cause, this difference can be referred. Its utility and final cause can be discerned; but, as to that which verily determines the rates of growth, and the durations of the teeth, it can only be referred to a First Cause; or it may be said, as of other things subordinate to a First cause, that it depends on some of those properties which each living being inherits from its parents, and through which it results that, in respect of time, as well as of method and quantity, the formative processes in the offspring are a repetition of those of the parent.

The observation of the development and changes of the teeth affords, moreover, an excellent instance of the punctuality with which time-work is regulated in the organic processes, and of the manner in which several different, and really independent, processes, being set to the same time-rate, are made to co-operate to the end of utility in the economy. This is evident in the coincidence of the development of the teeth of the second set, with the removal of those of the first; and in the coincident growth of the jaw, and all its muscles and other apparatus for mastication. In all of these (and the same might be said of any other system of organs in any species) the formation of every part is achieved with an admeasurement of time as precise, and as perfectly designed, as that of its shape, or size, or structure.

For examples of organic processes, adjusted to be complete in definite periods of time, the germination of seeds, and the hatching of eggs, could be cited. In plants, and in cold-blooded animals, the time varies according to temperature, yet not without evidence of a proper time-rate; but among birds, each species has its own time for incubation, as fixed as its other specific characters. In other words, the development of the structures of an egg into those of a young bird,

appropriately fitted for life in the open air, is timed to a certain rate of progress; so much work is to be done in so many days, neither more nor less; and on each day its appropriate and special portion of the work. And it is evident that the time occupied in the process is determined by the inherent properties of the egg itself. For if the eggs of any number of species be exposed to the same heat and other conditions, in a hatching machine, then, as surely as the bird produced from each will be like its parents, so surely will it be hatched in the same time as its parents were; in other words, the observance of a specific time-rate in the process of development is as exact as that of any other specific character.

With this observance of time in the development of the young might be noticed that which is, commonly, coincident in the parent. Not to cite the example of all the mammalia, that of pigeons might be taken, in which, during the incubation of their eggs, the crops of the parents are remarkably developed, so that they may be fitted for the secretion of a fluid destined to make the food of their young offspring more suited for their sustenance. The correspondence of these time-rates, observed, at once, in the development of the young pigeons, and in that of the crops of the parents, demonstrates, in both, a provision for chronometry in their organic processes, as clearly as the faces of two clocks, constantly keeping time together, would prove that they both have some apparatus for chronometry within.

Further, the provisions made by parents for their future young afford evidence of the time-regulation of organic processes, in so far as those provisions seem to indicate a reckoning of the time necessary for their completion. For example, certain turtles lay their eggs in hollows made in the sand, leave them there to be hatched, and at the time of hatching return to them for the sake of their young. It might be asked, how can these creatures, and many others in similar cases, reckon the passage of time? Most probably, they do not reckon it at all; but just as the timely attained fitness of their organization for preparing and filling their nests impelled them to those acts, so some time-regulated organic processes, taking place in them after the laying of their eggs, bring about at length a new condition, of which a dim consciousness becomes an impulse to them to return to their nests. Such an explanation would involve little guess-work; for changed organization is, manifestly, often the source of impulse to instinctive actions, and the parental organization does commonly change at a rate commensurate with that of the development of the offspring. And a similar reference to chronometric processes in the body, might explain many, though probably not all, other instances in which animals seem to have a power of reckoning the passage of time.

The phenomena of disease, especially in fevers, agues, the consequences of injuries, and many cutaneous eruptions, would afford abundant instances of the observance of time in the organic processes. The vaccine disease might be generally watched as an illustration, being characterized by a vesicle at each place of insertion of the virus, which vesicle begins to appear on the third day, and on the following days

passes through changes which are as exactly regulated in time as they are in visible characters. The changes in this vesicle are, moreover, indicative of a coincident succession of events in, or produced by, the virus inserted, which, in the blood of the vaccinated person, increases, and, incorporating itself in the vesicle, reaches its highest development and greatest inoculating power on the eighth day, and then degenerates.

The vaccine might, in most essential points, be regarded as a type of morbid poisons, *i.e.*, of such as are the products of disease. Whether inserted in the blood by inoculation, or bred therein, they commonly occupy definite periods of time in their development, and increase, and decline; as with a life which is chronometric in all its phases and in its total length.

The instances of morbid poisons would supply examples of organic processes timed to various numbers of days; and many that are completed in a day, or in given portions of a day, are traceable in the events of sleep and waking in animals (and, perhaps, also in plants), in the daily variations of the pulse, and of breathing, the returns of hunger and thirst, the regulated times of the digestive functions, &c. In man, indeed, consciousness and will are so concerned in some of these functions, that they may seem to lack that regularity which belongs to merely organic processes; but, if studied generally, and in other species as well as man, they all tell of such processes accomplished with regular measurement of time, and not determined by the external events or conditions of the day or night. Thus, for sleep and waking, and the times of hunger and thirst, man's independence in regard to day and night, or light and darkness, and the habits of different species whose times of activity are, severally, in the early or later day, in twilight or at night, may prove that the earth's diurnal changes are not the causes of these diurnal peculiarities of animal life. The very cause of sleep, and of that which is yet more mysterious, waking, may be unknown; but they are evidently connected and correlated with those alternating conditions of the structures, of which men, and probably all animals that sleep and wake, are conscious in the sensations of fatigue and of refreshment. The ordinary activities of one portion of the twenty-four hours, the activities, especially, of the muscles and nervous centres and the senses, produce an amount of structural, or chemical, change which is exactly repaired in rest during sleep. In other words, the organic processes for the repair of structures changed (as all structures are) by exercise, are adjusted to such a rate, that, in general, and on an average, in the time of sleep, they may completely restore the parts that are impaired in the activity of waking time. And so, of that replacement of substances in the several structures and in the blood, which is the purpose of feeding; the processes of digestion and of the several stages of assimilation are so timed, as to accord exactly with the times of daily taking food.

The most minute observances of time in organic processes might be noted in organs that have rhythmic motions, as in hearts and breathing muscles, ciliæ, the vacuoles of certain zoospores, as Volvox and

Gonium. In the Croonian Lecture at the Royal Society, in 1857, the speaker had endeavoured to prove that these and other rhythmic movements in plants, as well as animals, are due to corresponding time-regulated nutrition. He had expressed his belief that "rhythmic motion is an issue of rhythmic nutrition, *i.e.*, of a method of nutrition, in which the acting parts are, at certain periods, raised, with time-regulated progress, to a state of instability of composition, from which they then decline, and in their decline may change their shape and move with a definite velocity, or (as nervous centres) may discharge nerve-force." And this would be still maintained; but whether it were true or not, the rhythmical nutrition of rhythmically acting muscles would be certain. If not a cause, it must be a consequence of such acting; for it is inconceivable that the heart (for example) or the diaphragm, or any other rhythmic muscle, should be free from waste or impairment in its action, or from the necessity of being renovated in its rest. Difference of mode of action could not determine a difference in the immediate effect of action. With long exercise, muscles become so changed that their changed state can be felt in the sensation of weariness, and proved by chemical analysis. But the change thus proved is only the accumulation of the changes wrought in many muscular actions, each of which has contributed a share to the whole amount, just as each revolution of a wheel contributes to the final wearing out. Similarly, every action of the heart, or of the breathing muscles, is attended with change or impairment of composition; but, the impairment is repaired in the next following period of rest or relaxation. In other words, the alternating actions in shortening, and rests in lengthening, of the muscular fibres are correlative and synchronous with their alternating impairments and repairs of composition. The chronometry of such organic processes seems perfect; nutrition is in them divided, as it were, into units; and for each unit, there might be reckoned a unit of time.

Two results of this constant maintenance of rhythmic muscles are remarkable; namely, the enormous power they are capable of exerting, and their freedom from fatigue when only naturally acting. The latter result is proved to depend on the constant maintenance of the muscles, in their timely intervals of rest, by the weariness which is produced in the same muscles when they act otherwise than rhythmically, as in the muscles of respiration when employed in any voluntary movements, or in coughing or other violent respiratory acts.

The instances adduced thus far might supply examples of organic processes adjusted to periods of time varying from the length of human life to less than a second. They were all examples of large classes of facts, from which might be filled up the instances of observance of other and very diverse periods of time; and in all of them, the time-rate is essentially determined, not by external conditions (though these may, in some measure, modify it) but by the inherent properties of the organic bodies themselves.

In another large group of instances, those, namely, in which vital processes are completed, or attain some climax, in a year or in a set

portion or season of a year, an independence of external conditions appears less evident. The higher organisms, chiefly by reason of their having in themselves the power of generating heat, may manifest their own time-laws with comparatively little disturbance from without. But in the vegetable world, and in the lower animals, the organic processes are, for the most part, suspended during part of the year, for want, chiefly, of the heat which is a necessary condition of their activity, and the variations of which, for the rest of the year, very greatly affect their rate. Yet even in these, there appear sufficient indications that the times in which the processes of organic life are accomplished depend, essentially, on the specific properties of the several organisms themselves.

Thus, under the same external conditions, each species observes a proper rate of its own. All the plants, for example, of a given locality are subject to the same temperature, and other seasonal conditions; but their rates of living, like those of various eggs placed in the same heat, are different; each reaches the chief events of its life at a certain period of the year. Variations of the seasons may affect all of them; but their method of succession is not thereby changed; they observe the same proportions in the times severally required for their organic processes; and this unaltering proportion indicates a time-rate specific for each, though equally variable in all.

Moreover, among plants, there are numerous examples of varieties, which differ from the general characters of their species only, or chiefly, in regard to the times at which their vital processes are accomplished. Such are the variations that are known as "late," and "early," among flowers or fruits; of which some may be propagated by seeds. [Specimens were shown from two horse-chesnuts growing opposite to one another by the great gate of the Kew Gardens, of which one is, every year, three weeks earlier than the other, in all the processes of its life; and of varieties of Erythronium Dens Canis, from the same gardens, the plants of which, growing side by side in the same bed, always present a similar difference in their times of flowering, &c., though in all other respects alike.] It would be difficult to imagine a variety thus marked only by a peculiarity in rate of living, if temperature, or the influence of the seasons, alone determined the rate of life in the species. The simplest explanation seemed to be that, as there may be varieties in size and number of organs, and almost all the other properties of a species, which together make up its specific character, so there may be also varieties in regard to that time-rate of the processes of organic life which, even by this variability, is indicated as essentially dependent on the properties of the organism itself.

Again, there are some species in which there seems to exist a singular independence of external conditions. Instances of this are found in the Eriogaster lanestris, and the other moths mentioned by Kirby and Spence. If pupæ, formed in June or July, be "selected of the same size, and exposed to the same temperature, the greater number of them will disclose the perfect insect in the February following; some not till the February of the year ensuing, and the remainder not before the same month in the third year." (Vol. iii. p. 264.) The design of so singular an arrangement is, as they observe, to secure that insects, coming into active life in February or March, may not be utterly exterminated by the ungenial weather of a single season, or of two such seasons in succession: but the very cause of the differences among the pupæ, in their relations to the same external conditions, must be in their own properties.

A somewhat similar instance of apparent complete likeness among seeds in all respects except that of time, is in those of a Begonia, which, if taken from the same pod, and all planted together, and all kept in the same conditions, will germinate, some in a day, some at the end of a year, and some at various intermediate times.

To these indications of self-dependent time-rates in the lower organisms, might be added all the facts of another class, which show punctuality in the adjustment of several distinct processes. Scarcely an event of life could be watched which would not show it.

[The instance by which it was illustrated, was that of a Saxifraga, whose stamens, like those of Parnassia, arrive at their very maturity, not all together, but in pairs, and in pairs bend upon the pistil, each pair rising again before another pair bends down.]

And, lastly, the influence of temperature on the rate of the formative processes in the lower organisms is scarcely, or not always, greater than that of nutriment and other external conditions, is on their quantity. The occurrence of "good" and "bad" seasons indicates the latter influence, as that of "early" and "late" seasons does the former. Plants of the same species growing, some in an arid, others in a rich soil, differ exceedingly in size; the one are stunted, the others exuberant; here nutriment modifies the quantity of formation as, in other instances, varieties of heat will modify its rate. But this being so, it may be held that as a certain average size or quantity of growth is a characteristic of each species, and an issue of its very nature, so is a certain average time or rate of growth. Quantity and rate may alike be varied by external circumstances, but the standard or medium of both, as well as the limits of variation compatible with life, are determined by the natural and inherent properties of the species.

Whatever evidence these and the like facts might supply, that, in connexion with the seasons, the time-rates of the organic processes in the lower organisms are essentially dependent on the inherent properties of each organism, similar evidence might be adduced for the case of the higher, and especially the warm-blooded animals. In these the varieties of seasons have less influence in modifying the rate, as well as all the other measures, of life; and the less influence, the higher the species, or the degree of development of the individual. Moreover, there are in birds some instances in which organic processes have a tendency to observe certain times of the year even when the seasons are changed. Thus among those brought from Australia to this country, some of the parakeets breed here in December; the black swan sometimes breeds in November as well as in May; the New

portion or season of a year, an independence of external conditions appears less evident. The higher organisms, chiefly by reason of their having in themselves the power of generating heat, may manifest their own time-laws with comparatively little disturbance from without. But in the vegetable world, and in the lower animals, the organic processes are, for the most part, suspended during part of the year, for want, chiefly, of the heat which is a necessary condition of their activity, and the variations of which, for the rest of the year, very greatly affect their rate. Yet even in these, there appear sufficient indications that the times in which the processes of organic life are accomplished depend, essentially, on the specific properties of the several organisms themselves.

Thus, under the same external conditions, each species observes a proper rate of its own. All the plants, for example, of a given locality are subject to the same temperature, and other seasonal conditions; but their rates of living, like those of various eggs placed in the same heat, are different; each reaches the chief events of its life at a certain period of the year. Variations of the seasons may affect all of them; but their method of succession is not thereby changed; they observe the same proportions in the times severally required for their organic processes; and this unaltering proportion indicates a time-rate specific for each, though equally variable in all.

Moreover, among plants, there are numerous examples of varieties, which differ from the general characters of their species only, or chiefly, in regard to the times at which their vital processes are accomplished. Such are the variations that are known as "late," and "early," among flowers or fruits; of which some may be propagated by seeds. [Specimens were shown from two horse-chesnuts growing opposite to one another by the great gate of the Kew Gardens, of which one is, every year, three weeks earlier than the other, in all the processes of its life; and of varieties of Erythronium Dens Canis, from the same gardens, the plants of which, growing side by side in the same bed, always present a similar difference in their times of flowering, &c., though in all other respects alike.] It would be difficult to imagine a variety thus marked only by a peculiarity in rate of living, if temperature, or the influence of the seasons, alone determined the rate of life in the species. The simplest explanation seemed to be that, as there may be varieties in size and number of organs, and almost all the other properties of a species, which together make up its specific character, so there may be also varieties in regard to that time-rate of the processes of organic life which, even by this variability, is indicated as essentially dependent on the properties of the organism itself.

Again, there are some species in which there seems to exist a singular independence of external conditions. Instances of this are found in the Eriogaster lanestris, and the other moths mentioned by Kirby and Spence. If pupæ, formed in June or July, be "selected of the same size, and exposed to the same temperature, the greater number of them will disclose the perfect insect in the February following; some not till the February of the year ensuing, and the remainder not before the same month in the third year." (Vol. iii. p. 264.) The design of so singular an arrangement is, as they observe, to secure that insects, coming into active life in February or March, may not be utterly exterminated by the ungenial weather of a single season, or of two such seasons in succession: but the very cause of the differences among the pupæ, in their relations to the same external conditions, must be in their own properties.

A somewhat similar instance of apparent complete likeness among seeds in all respects except that of time, is in those of a Begonia, which, if taken from the same pod, and all planted together, and all kept in the same conditions, will germinate, some in a day, some at the end of a year, and some at various intermediate times.

To these indications of self-dependent time-rates in the lower organisms, might be added all the facts of another class, which show punctuality in the adjustment of several distinct processes. Scarcely an event of life could be watched which would not show it.

[The instance by which it was illustrated, was that of a Saxifraga, whose stamens, like those of Parnassia, arrive at their very maturity, not all together, but in pairs, and in pairs bend upon the pistil, each pair rising again before another pair bends down.]

And, lastly, the influence of temperature on the rate of the formative processes in the lower organisms is scarcely, or not always, greater than that of nutriment and other external conditions, is on their quantity. The occurrence of "good" and "bad" seasons indicates the latter influence, as that of "early" and "late" seasons does the former. Plants of the same species growing, some in an arid, others in a rich soil, differ exceedingly in size; the one are stunted, the others exuberant; here nutriment modifies the quantity of formation as, in other instances, varieties of heat will modify its rate. But this being so, it may be held that as a certain average size or quantity of growth is a characteristic of each species, and an issue of its very nature, so is a certain average time or rate of growth. Quantity and rate may alike be varied by external circumstances, but the standard or medium of both, as well as the limits of variation compatible with life, are determined by the natural and inherent properties of the species.

Whatever evidence these and the like facts might supply, that, in connexion with the seasons, the time-rates of the organic processes in the lower organisms are essentially dependent on the inherent properties of each organism, similar evidence might be adduced for the case of the higher, and especially the warm-blooded animals. In these the varieties of seasons have less influence in modifying the rate, as well as all the other measures, of life; and the less influence, the higher the species, or the degree of development of the individual. Moreover, there are in birds some instances in which organic processes have a tendency to observe certain times of the year even when the seasons are changed. Thus among those brought from Australia to this country, some of the parakeets breed here in December; the black swan sometimes breeds in November as well as in May; the New

Holland Cereopsis-goose has bred at the Zoological Gardens every February for five or six years.* Among migratory birds, also, it has been observed that when they are kept in confinement, and removed from all the circumstances that might be supposed to induce or necessitate their journeys, they yet become restless at the return of the season for their migration.

In these and the like facts there appear indications of a chronometry in the organic processes of warm-blooded animals, which corresponds with that of the seasons, but is essentially independent. And, if it be so, these might form a group of facts, in addition to those of the diurnal variations of the organic processes, in which vital changes are set to the same rules of time as changes of the surface of the earth, yet have their own proper laws; and concerning which it might be said, that the cycles of life, and of the earth, do, indeed, correspond, but only as concentric circles do, which are drawn round one centre, but are not connected, except in design and mutual fitness.

But, however this might be, all the instances of time-regulation cited in the discourse (all being examples of large groups of facts), would seem sufficient to prove, that the observance of time in organic processes is as exact and as universal as that of any other measure; that each species has a certain time-rate for the processes of its life, variable, but not determined, by external conditions; and that the several phenomena commonly studied as the periodicities of organic life, are only prominent instances of the law which it was the object of the discourse to illustrate.

[J. P.]

* Mr. Sclater, to whom the speaker was indebted for this fact, supplied also dates which tend to prove that the Australian parakeets, in this country, breed less often in December than in the months from May to September, inclusive; but even a minority of instances of the observance of times, and a general tendency towards it, when the force of such external conditions as those of the seasons is strong against it, is good evidence that inherent properties are the mainsprings determining the rates of life.

CASE

OF

COMMUNICATION WITH THE STOMACH,

THROUGH THE

ABDOMINAL PARIETES,

PRODUCED BY ULCERATION FROM EXTERNAL PRESSURE;

WITH OBSERVATIONS ON THE CASES OF GASTRO-CUTANEOUS FISTULÆ ALREADY RECORDED.

BY

CHARLES MURCHISON, M.D. Edin., L.R.C.P.,

ASSISTANT-PHYSICIAN TO KING'S COLLEGE HOSPITAL, AND TO THE LONDON FEVER HOSPITAL.

[*From Volume XLI of the 'Medico-Chirurgical Transactions,' published by the Royal Medical and Chirurgical Society of London.*]

LONDON:

PRINTED BY

J. E. ADLARD, BARTHOLOMEW CLOSE.

1858.

CASE

OF

COMMUNICATION WITH THE STOMACH,

THROUGH THE

ABDOMINAL PARIETES,

PRODUCED BY ULCERATION FROM EXTERNAL PRESSURE;

WITH OBSERVATIONS ON THE CASES OF GASTRO-CUTANEOUS FISTULÆ ALREADY RECORDED.

BY

CHARLES MURCHISON, M.D. Edin., L.R.C.P.,

ASSISTANT-PHYSICIAN TO KING'S COLLEGE HOSPITAL, AND TO THE LONDON FEVER HOSPITAL.

Received 9th November.—Read 24th November, 1857.

In May of the present year, when on a visit to Aberdeen, I had frequent opportunities of seeing, and making observations on, the patient whose extraordinary case is detailed in this paper. She was then in the Aberdeen Royal Infirmary, under the care of my friend, Dr. W. Keith, the senior surgeon. I am indebted for the following details as to the patient's history to Dr. Keith, who has also kindly permitted me to bring the case before the notice of the Royal Medical and Chirurgical Society.

I have also appended to the paper a collection, arranged in a tabular form, of twenty-five cases of fistulous communications between the stomach and external surface, with some general observations deducible from these cases.

A. *Case of Catherine Ross.*

Catherine Ross, æt. 34 (born June 16th, 1822), a native of Aberdeen, was admitted into the Aberdeen Royal Infirmary, under the care of Dr. Keith, February 19th, 1857, with a large fistulous opening in the walls of the abdomen, situated between the scrobiculus cordis and umbilicus, and communicating in the freest possible manner with the interior of the stomach.

1. *Family history.*—Her father and mother are both alive. They have had five sons and five daughters. Two of the sons and two of the daughters died in infancy. She has a married sister subject to some convulsive affection, probably hysterical. She has also a brother afflicted with epilepsy, and an unmarried sister little better than an idiot. The parents themselves are in good health of mind and body—always sober and industrious.

2. *Previous individual history.*—This woman has on more than one occasion made herself noted by feigning diseases, so as to deceive her friends and medical attendants. In 1840, she was, for a period of three months, under medical treatment for hysteria in its most exaggerated form, being often convulsed and frequently cataleptic. She would pass eight, ten, or even fourteen hours in a seemingly unconscious state of mind, frequently conjoined with a constrained and unusual position of body. In 1844, after various attempts on her lower limbs, she succeeded in producing such a solid œdematous enlargement of the left upper extremity, as to deceive her friends, and mislead for two years more than one medical attendant, into the belief that she laboured under elephantiasis. During that period, severe and long-continued blistering was employed, and every conceivable mode of treatment had recourse to, with no perceptible alteration in the size of the limb. The cicatrices resulting from the blisters, becoming distended with effused fluid, and consequently rugged looking and elevated above the level of the surrounding skin, did more than anything else to complete the *vraisemblance* of actual elephantiasis. Wearied

out by a fruitless attendance, her medical advisers readily acquiesced in a proposal, emanating from herself, that the limb should be amputated at the shoulder-joint; and Dr. Keith was requested to see the case, with a view to her removal to the hospital. He pointed at once to the abrupt termination of the swelling at the insertion of the deltoid muscle, as demonstrative of an artificial enlargement; but as it would scarcely have been credited that the patient could, for two whole years, have kept up such a deception in the very midst of her family, no suspicion was betrayed in their presence, and she was admitted into a small ward of the Aberdeen Infirmary, on the 25th of August, 1846, the entry of her disease made in the Case Book being "Feigned elephantiasis." Being in the hospital at seven next morning, Dr. Keith walked suddenly into her ward, and found her hurriedly concealing a garter under her pillow. The fact of her having been in bed for two years, with no stockings on all that time, left no room to doubt that the garter had been tightly applied, at the insertion of the deltoid, on her left arm, by her teeth and right hand; and, accordingly, on exposing the arm, there was the deep sulcus, marked by the very ribbing of the worsted band. Dr. Keith gave her one chance, and promised not to expose the cheat, provided she would now consent to be cured: but in vain. She found means to strangulate the arm every night; so that, after a trial of three days, both her hands and arms were restrained in the sleeves of a strait waistcoat. After this, the cure advanced with great rapidity, and in five days the skin hung quite loosely upon the arm. The waistcoat was not removed for three weeks; indeed, not until her own entreaties, coupled with a promise of better behaviour, gave hope that she was wearied of deceit. On the 26th of October, 1846, after sixty-two days' treatment, she was dismissed cured; both arms being much alike; her spirits good; her mind active and intelligent; and she herself professing great gratitude for what she was pleased to designate "the wonderful cure!"

For two years after this she continued well, keeping

house for an uncle; when at length she took a fancy that she had heart disease, for which a surgeon inserted a seton in the vicinity of the scrobiculus cordis, and kept it open for some years. It was in November, 1853, that Dr. Keith again, and only by accident, saw her. The seton had at length ulcerated out; but he was told that the wound would not heal, and his opinion was asked. He examined a deep, round, fiery-looking ulcer, exactly midway between the umbilicus and the ensiform cartilage, of the exact shape and size of an old copper penny of the reign of George III, which she had very nimbly nipped out with her nail, along with the dressings. Dr. Keith warned her of her folly, and explained to her the risk she ran of ulcerating a passage into her belly, which might prove fatal. She evidently paid little attention to what was said, and Dr. Keith refused to take charge of the case. At length, after fully three years' pressure by a belt, of a copper coin over the ulcerating surface, she was seized with irritability of the stomach, bringing up bile and blood. Along with these symptoms, she had much fever for five days; and on one of these days, viz., March 2d, 1854, she discovered, on removing the dressings from the wound, that a piece of biscuit and a portion of orange peel, along with a quantity of fluid, escaped from the opening. For many days after, the whole food swallowed was discharged by the opening, and she became greatly exhausted. By the 23d of March, however, her own shrewdness taught her to fit a plug of gutta percha into the wound, so as actually to cork it up. This plug, somewhat enlarged on two subsequent occasions, as the opening has become a little larger, and kept *in situ* by a belt round the waist, has ever since sufficed to retain the great bulk of her food within the stomach, where it would appear to be well and quickly digested; her flesh, for a naturally spare person, being well kept up. Her general health has improved, and her hysterical symptoms have, for the most part, disappeared, from the period that the opening was established; she having now something special with which to occupy her mind and take up her

attention. After a delay of nearly three years, she sought admission into the hospital, as already stated, on February 19th, 1857, her professed wish being to have the opening closed.

3. *Observations on her condition while in hospital.*—She has jet-black hair and dark eyes, pale skin, prominent lips, melancholic countenance, and although spare, is not much emaciated. She has kept her bed for nine years; and consequently she is quite unable to walk or even to stand, and even feels faint and weak when she sits up in bed. There is no great stiffness of the joints, however, and the limbs are wonderfully well nourished; so that the chief reasons for her keeping her bed appear to be habit and her own dogged obstinacy.

In the epigastric, and upper part of the umbilical region of the abdominal parietes, is an oval opening communicating with the interior of the stomach. The long diameter is transverse, and measures four inches, the vertical diameter being about three inches. When the patient sits up or stands, or, even when reclining, if she coughs, or makes an effort as if to vomit, the walls of the stomach fall out through the opening, the whole organ becoming everted. She can take the stomach out, and push it in again, without feeling the slightest pain, even on free manipulation. Three fingers can be introduced into the cavity of the stomach with ease, and one of them passed down into the pylorus, or upwards into the cardiac orifice. She commonly keeps the opening closed by a gutta percha plug, covered with chamois leather and oil silk, and retained by a circular bandage. When this plug is removed, everything she swallows is almost immediately ejected through the opening, and even when the plug is *in situ*, some of the liquid portions of the ingesta ooze away by its sides, and, in a slight degree, irritate the surrounding skin. The margins of the opening, for some inches, are red, smooth, glistening, and tender. The mucous membrane of the stomach is of a bright vermilion-red colour, and occasionally bleeds a little, at isolated spots, when irritated. Although handling of the mucous mem-

brane and other irritating causes seem to produce no pain, they are always followed by a feeling of sinking and great faintness for a few minutes. A piece of blue litmus paper applied to the moist surface of the empty stomach, remains unaltered; but after food has been swallowed, or if the litmus paper is introduced into the pulpy food undergoing digestion, it is immediately turned red. The mucous membrane is disposed in rugæ, arranged longitudinally as regards the long axis of the stomach, but assuming a circular direction around the everted organ. Undulating movements may often be seen passing along the surface, which are increased by the contact of food. The edges of the opening are fully three quarters of an inch thick, red, and glazed; not at all ulcerated, but firmly cicatrized. The mucous membrane of the stomach appears gradually to lose itself in the surrounding skin, so that the margins of the opening are not unlike those of the human lip; and, indeed, the whole opening bears a coarse resemblance to a large mouth, half open.

Her tongue is clean and soft all day, but dry and parched towards night. She is usually very thirsty, and drinks a great deal of water. Her appetite is seldom at all impaired; and, at times, is so keen as to amount to pain. She often requires to swallow a piece of bread on the instant, to remove an unbearable feeling of sinking. She can eat and digest any kind of food, but she complains of pain at the pit of the stomach after eating solid food. Her preference is for eggs, and boiled fish, tea and bread, and vegetable soups. No pain follows the use of any of these; but she always feels great annoyance from the increase of irritation around the opening, owing to the escape of the fluid contents of the stomach, by the side of the plug, after each meal. She has only about one stool in the twelve days, consisting of numerous, hard, rounded scybala, like, but larger than, the droppings of sheep.

Her pulse is 80, regular, soft, and of fair volume. On auscultation there is nothing abnormal heard about the heart's sounds. The impulse of the heart cannot be distinctly felt from the stomach, on introducing the fingers and pressing them upwards, but the attempt to do this occasions considerable pain. It is very noticeable, however, how very close to the posterior wall of the stomach is the abdominal aorta, and how very immediately the tip of the finger meets its pulsation, when inserted into the opening.

Her respirations do not exceed twelve in a minute. She has no cough, nor any symptom of chest disease.

Her urine presents normal characters, but is rather scanty.

She has never been married, nor had any family.

She began to menstruate at the age of 14; and continued regular up to the time at which the opening in her belly took place. Since then the menses have entirely ceased. She has no leucorrhœa.

Her temper is placid, and she is affectionate to her parents. She at times has fits of crying, but not of laughing, as she once had; but, occasionally, "she flies off into a rapture," as her mother calls it. Her sleep is disturbed, but little seems to suffice: for a long time she has been in the habit of taking the equivalent of fully an ounce of laudanum a day. She is intelligent, and well informed for one in her station of life; she speaks rationally on every subject, but is silent when her own misdeeds are alluded to. She has never attempted self-destruction, but is calm in the prospect of death, and says she prays for it. Had she succeeded in her original wish of getting an arm amputated, so as to make herself an object of commiseration, and afford a sufficient excuse for living on her friends in perfect idleness, the probability is that she never would have fancied heart-disease and never have commenced the process of continuous pressure, by which, after a period of three years, she succeeded in safely effecting an entrance into her stomach. Three years of the consequences may have somewhat sickened her of the experiment, and when she sought admission into the hospital, it was, as already stated, with the professed object of having the opening closed. Dr. Keith was inclined to think that this

might be effected by bringing the edges together after effectual paring, provided that the patient was really wishful to have it so. But, after some delay to weigh the question, he came to the conclusion that she would render every such attempt fruitless. She has gained her point; lives an idle life; eats, drinks, reads a book, and is satisfied. She feels herself to be an object of great interest; and one has only to witness the perfect complacency with which she uncorks her stomach, allows her dinner to gush out, and invites a visitor to search with his finger for the cardiac or pyloric orifice, to be convinced, that however much she might relish the *fuss* of an active attempt to close the opening, she has no wish nor intention that it ever should be closed.

Some weeks ago I wrote to Dr. Keith, to inquire if Catherine Ross ever vomited, and if any observations had been made of the stomach during the act of vomiting. The replies which I received, with an account of a very interesting experiment undertaken by Dr. Keith, were to the following effect: at intervals of a month or two she has a feeling of nausea, accompanied by a burning sensation at the scrobiculus cordis; but this ends only in an escape of bilious fluid at the side of the plug. Prior to the formation of the opening she vomited very frequently; but, in a period of three years and seven months, *i. e.*, since the 2d of March, 1854, she has only about ten or twelve times brought up one or two mouthfuls of yellow-tinged mucus, and that only when the plug was in the opening. On the 30th of last October, Dr. Keith made the following experiment: the plug was removed, and as much of the contents of the stomach (consisting of vegetable soup recently swallowed) as the ordinary protrusion of the stomach would eject, were allowed to escape. The fauces were then tickled with a feather held in the patient's own hand. Immediately the whole remaining contents of the stomach were projected from the opening. This appeared to depend, in the first place, on a strong spasmodic contraction of the stomach itself, and principally of its pyloric extremity, for

there was squirted from the pylorus, as if from a syringe, about a teaspoonful of light yellow bile. Simultaneously, however, with this, the abdominal walls were observed to become rigid, and this became more apparent on continuing the irritation of the fauces; for the next effect was seen to be violent contraction of the recti and other abdominal muscles, producing complete and almost instant eversion of the stomach. This organ was turned fairly inside out, evidently owing to the downward pressure of the contracting diaphragm meeting the upward pressure of the contracting muscles of the abdominal parietes. The upper wall of the stomach, indeed, could be seen and felt to be pressed downwards by the diaphragm. After a few minutes the patient herself restored the stomach to its place, pushing it in with the gutta-percha plug; and within ten minutes she partook of another plate of soup, as if nothing had happened, feeling neither nausea nor pain from the recent experiment.

Of the numerous points of interest connected with this case, the most important is undoubtedly the circumstance of the existence of a fistulous communication with the stomach, existing for a lengthened period during life.[1] Every member of the profession is acquainted with the case of the Canadian, Alexis St. Martin; but few, I believe, are aware that the records of medicine contain accounts of many other such cases, resulting either from injury or disease; and that, in not a few instances, the patients have lived for many years with the open fistula. I have, therefore, been induced to make the following collection of cases of gastro-cutaneous fistulæ.

[1] I have had another opportunity (June, 1858) of seeing Catherine Ross, who is now living with her parents in Aberdeen. She is, on the whole, in better health than last year, having gained somewhat in flesh. Nothing will persuade her to leave her bed; but there appears little to prevent her, except her own perverse determination. The dimensions of the opening in the abdominal parietes have slightly increased.—C. M.

B. *A Collection of Cases of Gastro-cutaneous Fistulæ.*

The cases of fistulous communications with the stomach through the abdominal parietes, which I have been able to collect from the records of medicine and surgery during the last 300 years, amount to twenty-five. For the sake of brevity, I have arranged them in a tabular form, so as to illustrate the following points:

1. The date at which the opening occurred.
2. The patient's name and residence.
3. The patient's age.
4. The cause which gave rise to the fistula.
5. The situation and characters of the external opening.
6. The size of the external opening.
7. Situation of the opening as regards the walls of the stomach, and any other morbid appearances of this organ.
8. Observations made as to the escape of food from the opening.
9. Duration of the fistula, and its consequences — whether the patient died or was cured—and the effects on the general health.
10. A brief notice of any physiological observations made as to the appearance of the mucous membrane of the stomach, the movements of its walls, and the nature and properties of the gastric juice.
11. The names of the observers of the cases, &c.
12. References to published accounts of them.

A COLLECTION OF CASES OF GASTRO-CUTANEOUS FISTULÆ, ARRANGED CHRONOLOGICALLY.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
I.	1560?	Puggebrolt, a Bohemian peasant.	Adult.	Wound from cutlass.	Not recorded.	Admitted finger.	"Below cardiac orifice."	All food and drink escaped, immediately on removing linen plug.	Many years. Enjoyed good health.	None.	Matthias Cornax.	Journ. de Méd. et de Chir. par Roux et Corvisart, tom. iii, p. 508, 1802.
II.	1650?	A beggar at Castres.	Adult.	No mention made of any injury; and probably from simple ulcer of stomach.	Not recorded.	Admitted finger.	?	Chyme flowed out immediately on removing linen plug.	Many years. Enjoyed good health.	None.	Petrus Borellus.	Petri Borelli, Hist. et Obs. Med. Phys., obs. lxvi. p. 69, 1676. Journ. de Méd. et de Chir. par Roux et Corvissrt, tom. iii, p. 508, 1802.
III.	16—?	A boy.	13	Simple ulcer of stomach? Abscess in liver opening into stomach, and through abdominal parietes.	Near umbilicus. "Fistula umbilici funesta."	"Foramen angustum"	Perforation part of stomach which was adherent to liver.	Food escaped soon after being swallowed.	Eighteen months. Increased desire for food. Died with numerous abscesses in liver.	None.	Ludolph. G. Klein.	Ephemerides Med. Phys. Germ., Ed. Norib., tom. x, obs. lxxii, p. 248, 1754. Gérard, "Perf. Spont. de l'Estomac," 1803.
IV.	16—?	Female Gardener.	Adult.	Simple ulcer of stomach? "Huic vermes fundum ventriculi perforárunt."	Left hypochondrium. "Circa latus sinistrum." Indurated margins.	"Ulcus apertum circulo, durisque labiis."	At fundus.	Beans, and all food difficult of digestion, escaped by opening.	Twelve years. Went about her work, sold vegetables, &c.	None.	?	Ephemerides Med. Phys. Germ., Ed. Lips., ann. iv et v, obs. xxxviii, p. 36, 1676.
V.	16—?	?	Adult.	An incised (?) wound.	?	?	?	Some of aliment always escaped by it.	Eleven years.	None.	?	Ephem. Med. Phys. Germ., quoted by Gooch in Pract. Treat. on Wounds, vol. i, p. 398, 1767.
VI.	17—?	?	Adult.	External wound from a knife.	Middle of Epigastrium.	Admitted finger.	?	Food escaped from it, immediately after it was swallowed.	Two months in fistulous condition—then closed up.	None.	M. Lasséré.	M. Hévin, in Mém. de l'Acad. Roy. de Chir., tom. i, p. 594, 1743.
VII.	17—?	A female.	Aged.	Cancer of stomach.	Left hypochondrium.	? Apparently small.	Probably at fundus? At "scirrhus pylorus," and contracted stomach.	Liquid ingesta escaped by it, if she turned on left side.	?	None.	Souyer Dulac.	Lieutaud, Hist. Anat. Med., vol. ii, obs. cxlv, p. 327, 1767. Gérard, "Perf. Sp. de l'Estomac," Paris, 1803, p. 71.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
VIII.	1712 to 1739.	Marguerite Egwerin, of Norlingen.	19 to 46	Necrosis of ribs with abscess, which at first only opened externally, and healed. Four years after, a second abscess, from which, on opening, food escaped. Was there originally simple ulcer of stomach?	Between eighth and ninth ribs, four inches from xyphoid cartilage, and two from left nipple. Ninth costal cartilage and portion of tenth rib necrosed.	Two inches in diameter. Easily admitted thumb. Cartilaginous margins. Skin excoriated.	Opening at middle of great curvature; between this and pylorus, stomach not contracted.	Most of food would escape by opening when not prevented.	Twenty-seven yrs. "Embonpoint." Could do light work. Intense hunger. Obstinate constipation; no stool at first for seventeen weeks! Urine scanty. Pure blood from stomach at every menstrual period. Ultimately died with numerous abscesses in spleen and liver.	When food swallowed, coats of stomach often seen to contract violently, with an antiperistaltic movement—almost convulsively, until all food ejaculated with force.	Augustus and Christopher Wencher. Haller was present at the *post mortem*.	C. Wencher, "Virginis per 27 annos ventriculum perforatum alentis historia et sectio," Strasbourg, 1743. Lieutaud, Hist. Anat. Med., lib. ii, obs. cxiv, p. 327, 1767. Haller, Disp. Chirurg., tom. v, No. 125, p. 18, 1756. Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 513, 1802. Gérard, Perf. Spont. de l'Estomac, Paris, 1803, p. 70. Cruveilhier, Traité d'Anat. Path., tom. ii, p. 566, 1852.
IX.	1715 May 16.	A widow.	36	Cancer. A scirrhous mass, 5 inches by 2½, involved stomach and arch of colon; intimately uniting these with abdominal parietes and one another. Opening through skin for 11 days before food came from it.	In umbilical region.	Easily admitted finger.	A cancerous ulcer in middle of great curvature, two inches in diameter, at centre of which a perforation the size of a shilling.	Greater part of food escaped by it, quite unaltered.	Three months. Extreme emaciation and exhaustion.	None.	M. Petit.	Petit, Mém. de l'Acad. Roy. des Sc., 1716, p. 312. Haller, Disp. Chirurg., tom. v, No. 125, obs. v, p. 31, 1756. Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 524, 1802.
X.	1719.	A virgin at Leipsic.	20 to 30	Blow in epigastrium from a carriage shaft—followed by abscess, which opened in six months.	Left of epigastrium.	Size of a large pea or haricot	?	Meat and drink escaped immediately on being swallowed.	Ten years. Went about usual occupation, to market, &c. Ultimately *cured* by a few weeks of repose.	None.	M. E. Ettmuller.	Ettmuller, "De vulnere ventriculi," Leipsic, 1730. Haller, Disp. Chirurg., tom. v, No. 163, 1756. Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 525, 1802. Gérard, Perf. Sp. de l'Estomac, Paris, 1803, p. 69.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prognosis of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
XI.	1740.?	A female.	Adult.	Simple ulcer of stomach. First a tumour at epigastrium — this disappeared—skin became drawn in and like a cicatrix. More than a year after, fluid first seen to escape from this.	In epigastrium.	? Apparently small.	?	Red wine and pulpy food seen to escape by it, just after being swallowed.	But a short time, and closed spontaneously.	None.	Morgagni.	Morgagni, De Sedibus et Causis Morborum, 1761, lib. iii, epist. 36, Ninth Edit., 1821, vol. iv, p. 410. Gérard, Perf. Spont. de l'Estomac, 1803, p. 69.
XII.	1742. July.	A widow.	37	Cancer of pyloric end of stomach.	Near umbilicus.	Admitted finger.	Pylorus adherent to umbilicus, and to left lobe of liver. Ileum "omnia erant putrida."	Everything swallowed escaped by it.	Three weeks. Died. Appetite good until death.	None.	Communicated to Haller.	Haller, Disp. Chirurg., tom. v, No. 125, obs. iv, p. 30, 1756.
XIII.	1768. May.	Mrs. Tovell, of Wenhaston, near Norwich.	60	Simple ulcer of stomach. Twenty years before had a slight injury over stomach—after this liable to pains in left side. In ten years a tumour at epigastrium, with fever, &c.; and in ten years more this opened.	In epigastrium. "No ill appearance about ulcer."	Of some size, as solid food escaped.	Near pylorus. Firm adhesion of stomach and liver round about orifice.	When dressings removed, everything swallowed would escape at orifice.	Three and a half months. Did not lose flesh, and went about usual occupations. No sickness, and one stool every day. Died from effects of cold.	None.	Benjamin Gooch and Mr. Aldrich.	Gooch, Pract. Treat. on Wounds, vol. iii, Appendix, p. 140, 1774. Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 503, 1802.
XIV.	1794. October.	M. Maillot, Lieut. of French Infantry at battle of Kayserlautern.	Adult.	Gun-shot wound. Ball extracted—followed by inflammation and sloughing of adherent wall of stomach.	Close to ensiform cartilage, rounded	Half an inch. "15-sous piece."	?	When plug removed after meals, food immediately escaped by it.	Eight and a half years at least, as he was not dead when case reported. Enjoyed good health.	Interior of stomach seen to be "d'un rouge très vif, et plissé dans tous les sens." Undulations of its surface occasionally observed, increased by contact of air or food. On making an effort to swallow, a lighted candle held before opening was agitated.	M. Percy.	Roux, Journ. de Méd. de Roux et Corv., tom. iii, p. 510, 1802. Gérard, Perf. Spont. de l'Estomac, Paris, 1803, p. 70.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
XV.	1793.	Magdelaine Goré, of Roussigny, Dept. of La Manche, a patient in "La Charité," Paris.	39 to 48	Simple ulcer of stomach. At age of 20, received an injury on epigastrium from fall on door-step. After this, liable to attacks of severe pain in stomach. Eighteen years after, an abscess, which opened on twenty-first day.	Opening oval, situated at upper and left part of epigastrium, one-third in epigastrium, and two-thirds in thoracic walls. Ninth, tenth, and part of eighth left costal cartilages wanting, and seventh sloped away. Surrounding integuments red and scaly.	1½ inch by 1¼.	Opening in anterior wall, eight inches from fundus, and four from pylorus. Extended from upper to lower margin.	Every day for eight years, three or four hours after food, was obliged, from "irresistible desire," to remove plug, and evacuate stomach. On removing plug, stomach would protrude several lines, forming a pad across opening.	Eight and a half years. Health good. Could walk about. Ate anything, and as much as three other women. One stool in four days. Rounded scybala. One pint urine in twenty-four hours. Died of marasmus after an attack of diarrhœa.	Interior of stomach, when empty in morning, seen to be "d'un rouge vermeil, enduit d'un mucus luisant." Longitudinal plicæ. Two sets of undulating movements, one towards pylorus, another towards fistula. Much gas escaped with food. Never could sleep, unless stomach was washed out with infusion of chamomile. Gastric juice described and analysed. Fluid collected in morning before meals "neither acid nor alkaline."	Roux and Corvisart; Dupuytren, Bichat, &c. Chemical Experiments by M. Clarion.	Roux and Corvisart, Journ. de Méd., tom. iii, p. 407. Gérard, Perf. Spont. de l'Estomac, 1803, p. 71. Richerand's Physiology, Fifth Ed., 1811, Dr. Copland's Eng. Ed., 1824, p. 112. Cruveilhier, Traité d'Anat. Path., tom. ii, p. 564, 1852. Preparation and wax models in Musée Dupuytren, at Paris. Appareil Digest., Nos. 60, 61, 62.
XVI.	June 6, 1822, till present time, 1857.	Alexis St. Martin, a Canadian, a "voyageur" in Hudson's Bay Compy., wounded at Michillimackinac, in Michigan.	18 to 53	Gun-shot wound. Charge of powder and duck-shot, at distance of only one yard. Blew off integuments and muscles over space, size of man's hand, carried away anterior half of sixth rib, perforated stomach, and exposed left lung.	Opening situated two inches below left nipple, in a line drawn from this to spine of left ilium. One year after injury, parts were all sound, and firmly cicatrized, except opening into stomach.	2½ inches in circumference.	Opening three inches to left of cardia, near left superior termination of great curvature.	At first, all food and drink discharged by it, on withdrawal of artificial plug; but one year and a half after injury, an accurate natural plug was formed by a protrusion of the mucous membrane, and food only escapes on the depression of this.	Thirty-five years, and is still alive (1857). Has enjoyed perfect health; has married, become the father of a family, and earned a livelihood for them by a laborious occupation.	Mucous membrane of a pale pink, varying in hue according to full or empty state of stomach. Temperature, 100° F. Rugæ. Vermicular movements seen to be increased by food. Irritation of stomach produced faintness, not pain. Physical characters of gastric juice described by Dr. Beaumont; and more recently investigated by Professor Dunglison and Dr. F. S. Smith. Fluid from empty stomach always of neutral reaction. Numerous observations on digestibility of different articles of diet, &c. Acidity of gastric juice due to hydrochloric acid (Dunglison), to lactic acid (Smith).	Dr. Beaumont, 1822 to 1833. Professor Dunglison, 1833. Dr. F. S. Smith, of Penusylvania, 1856.	Beaumont, Experiments and Observations on Gastric Juice, Plattsburgh, U. S., 1833. Dr. Combe's Edin. Ed. of above, 1838. References in most modern works on Physiology and Practical Medicine. Dr. F. S. Smith, Philadelphia Medical Examiner, July and Sep., 1856, and Brit. and For Med.-Chir. Rev., Jan., 1857, p. 241. Dr. Bunting, Lancet, July 19th, 1856. Dunglison's 'Human Physiology,' Seventh Ed., vol. i, pp. 585-6.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
XVII.	July 1, 1828.	M. A. W., a widow, Worcester Infirmary.	39	Simple ulcer, producing abscess, adhesions, and external opening.	Between seventh and eighth ribs, below right mamma. Lower margin of seventh rib, and upper of eighth, carious.	"A slit" 1 inch in length.	Immediately *beyond* pylorus, at very commencement of duodenum, two and a half inches from external opening.	Coffee and milk (curdled), &c., escaped immediately on being swallowed.	Twenty-six days. Died. Tubercles in lungs. Inflammation of diaphragm and surface of liver.	None.	Dr. Streeton.	Streeton, Lond. Med. Gazette, 1829, vol. iii, p. 43.
XVIII.	June, 1832, and Jany., 1835.	Madame G.	77 and 80	Simple ulcer of stomach. Symptoms for thirty years. At age of 77, an opening over stomach, from which "clear water" escaped for three days, and which then closed. At age of 80, this reopened, discharging water and food.	In a transverse furrow of the skin in left hypochondrium, two inches below margin of ribs. Margins of opening red. Surrounding integuments drawn in like a funnel.	"Size of a pea." "4 or 5 lines in diameter."	In middle of anterior surface a rounded opening, one and a half inch in diameter, forming large end of a funnel, of which small at external opening. Thickening of pylorus.	Red wine, and vermicelli soup, &c., seen to escape from opening (along with air-bubbles) just after being swallowed.	First time for three days. Second for four. Died of old age, &c. During last four days of life, tongue red and dry—burning thirst—no stools nor urine.	None.	M. Bineaux de Saumur.	Bineaux, Archiv. Gén. de Méd., II. Ser., tom. viii, p. 214, 1835. Cruveilhier, Traité d'Anat. Path., tom. ii, p. 566, 1852.
XIX.	Sept. 3, 1832.	Mary O'Neill, Meath Hospital, Dublin.	50	Cancer of pyloric end of stomach.	To left of umbilicus.	Size of a large pea. A gum bougee passed readily.	?	Gruel, &c., escaped immediately after being swallowed. For two days the opening was closed.	Three weeks. Died exhausted.	None.	Dr. Stokes and Mr. Hamilton.	Hamilton, Lancet, 1832, vol. i, p. 612; Archiv. Gén. de Méd., 1st Ser., tom. xxviii, p. 264, 1832.
XX.	1833.	A Widow, U. States.	39	Simple ulcer of stomach.	Opening at side of umbilicus. Surrounding skin red, excoriated, and tender.	Admitted a musket bullet.	Opening supposed to be near pylorus (from probing).	Water, &c., escaped from opening twenty seconds after being swallowed.	Six and a half months. Had great constipation. No motion for ten days. *Cured* by mechanical appliances to protect abdomen from irritating fluids, compression together of lips of wound, &c.	Does not appear that stomach was visible. A catheter introduced thirteen inches from opening excited vomiting.	Dr. J. H. Cooke.	Cooke, Western Journ. of Med. and Phys. Sc., Jan., 1834. American Journ. of Med. Sc., vol. xiv, p. 271, 1834. Dub. Journ. of Med., vol. vi, No. 16, p. 148. Lond. Med. Gaz., vol. xiv, p. 541. 1834. Archiv. Gén. de Méd., II. Ser., tom. vi, p. 430. Schmidt's Jahrbucher, 1836, vol. ix, p. 61. Cœlius's Surgery, South's Ed., vol. i, p. 477.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
XXI.	Oct. 22, 1844, to Aug. 11, 1851.	Isabella Davidson, Kelso, Scotland, and Edinburgh Royal Infirmary.	30 to 37	Simple ulcer of stomach (?), inducing an abscess, which first communicated with stomach; then extension of inflammation and perforation of abdominal parietes.	Opening close to cartilage of eighth left rib, 2½ inches from median line, and 3¼ from umbilicus. Oval. Margin rounded, depressed, hard, and red; with specks of ulceration. Surrounding integuments red, indurated, and excoriated.	Admitted thumb. Shortly before death, as large as a crown piece.	In anterior wall of stomach, about four inches from cardia.	All food escaped freely by it, unless prevented by mechanical appliances. At first, the opening was closed by a valve of mucous membrane.	Nearly seven years; but during this time opening was twice closed, viz., from Jan., 1845, to Jan., 1846; and from March, 1846, to June, 1847. Was in bed for last three or four years of life. Tongue clean. Appetite good. No thirst. During digestion, pain at orifice. Urine scanty. Bowels confined. Menstruation regular.	A committee was appointed to make observations; but in consequence of patient's state of health, it did nothing. Mucous membrane of stomach was visible, but is not described. Milk swallowed could be seen flowing over it. Contents always highly acid, during digestion. Introduction of a catheter caused faintness, but not pain.	Dr. W. Robertson, of Edinburgh, and Dr. T. Hamilton, of Kelso, N. B.	Robertson, Edin. Monthly Journ. of Med. Sc., Jan., 1851, p. 1. Archiv. Gén. de Méd., IV Ser., tom. xxv, p. 339. Schmidt's Jahrbücher, vol. lxxxii, p. 35. Private letter from Dr. Hamilton, Nov., 1857. A plate showing the external opening accompanies Dr Robertson's paper.
XXII.	1850.	A married female, at Dorpat.	35	Simple ulcer of stomach.	Opening below left nipple, between ninth and tenth ribs, 2½ inches in an oblique line from the xyphoid cartilage. Margin red, and indurated. Surrounding skin erythematous, from trickling over it of gastric juice.	Half an inch in diameter.	?	Contents of stomach freely escaped by it.	Three years; and patient still alive in 1853. Is otherwise healthy and strong; suckling an equally healthy infant. Has a ravenous appetite.	Numerous observations made as to the quantity, physical characters, and chemical composition of the gastric juice; also as to its digestive powers over different nutritive principles. Acidity of gastric juice due to butyric, and probably lactic acid; it contained no free hydrochloric acid. The secretion from the empty stomach was always neutral.	Professor Schmidt, Otto v. Grünewaldt, and Ernst v. Schröder.	O. v. Grünewaldt, "Succus gastrici humani indoles physica et chemica, et vis digestiva," Inaug. Dissert. Dorpat, 1853. Schmidt's Jahrbücher, vol. lxxxiv, p. 1, 1854.
XXIII.	Sept. 7, to Oct 8, 1851.	Mrs. R., Braemar, Scotland.	50	Cancer of stomach.	Left part of epigastrium.	At first size of a shilling; latterly increased to that of a half-crown piece.	No *post mortem* allowed; but, on introducing finger during life, distinct carcinomatous hardness felt around opening.	Everything taken by mouth passed out at once by opening, unless prevented by gutta percha plug.	Thirty-one days. Suffered great pain. Bowels confined. Died.	Introduction of laxatives through opening into stomach induced great pain and tendency to vomit.	Dr. James Cameron, of Braemar, N. B.	Private communication from Dr. Cameron through Sir James Clark.

No. of Case.	Date of occurrence of Fistula.	Patient's Name and Residence.	Age.	Cause.	Characters, Situation, &c., of External Opening.	Size of External Opening.	Situation of Opening in Stomach, and other morbid appearances of that organ.	Observations on Escape of Food, Prolapsus of Stomach, &c.	Duration of Fistula and Results. General Health of Patient.	Physiological Observations.	Observers.	References.
XXIV.	Oct., 1854.	A female. France.	52	Cancer of stomach	Near umbilicus. Tumour, formany months before ulcerating, had been in epigastrium, but then descended to umbilicus. Margin of opening gangrenous.	Enlarged rapidly by gangrene of integuments.	Cancer at pylorus, in all probability.	From date of opening everything swallowed escaped by it.	Five weeks. Died.	None.	Dr. Balluff.	Dr. Balluff, Gaz. Méd. de Paris, 1855, p. 281. Schmidt's Jahrbücher, vol. lxxxiii, p. 49, 1854.
XXV.	March 2, 1854. to present time, June, 1858.	Catherine Ross, Aberdeen, Scotland.	32 to 36	Ulceration, produced by pressure from without of a copper penny piece, applied voluntarily by patient.	Opening in epigastric and upper part of umbilical region. Transversely oval. Edges of opening ¼ inch thick, red, glazed, not ulcerated, showing a gradual transition from skin to mucous membrane. Integuments, for some inches around, red, smooth, glistening and tender	Transverse diameter, 4 inches; vertical, 3 inches. Three fingers can be passed into stomach with ease; and one into pylorus or cardia.	Opening about middle of anterior surface.	When gutta percha plug removed, everything she swallows is immediately ejected from opening; and even when that is *in situ* liquids ooze out. On sitting up, coughing, or making an effort to vomit, whole stomach becomes everted.	More than four years, and still alive and well. Tongue clean, parched at night. Great thirst. Appetite at times so keen, as to amount to pain. One stool in twelve days. Amenorrhœa.	Mucous membrane of stomach of a bright vermilion colour—will bleed at isolated points when irritated. Handling stomach causes no pain; indeed, she can take it out and put it in again with ease; but such manipulations generally produce a feeling of sinking, and great prostration. When stomach empty, blue litmus paper applied to mucous membrane is not turned red. Waves of contraction seen passing along rugæ, increased by contact of food. Observations on mechanism of vomiting.	Dr. W. Keith, Aberdeen, and Dr. Murchison.	Account of case read before Royal Medical and Chirurgical Society of London, November 24th, 1857.

C. *The Causes of Gastro-cutaneous Fistulæ.*

Gastro-cutaneous fistulæ may be divided into those which are the consequences of mechanical injuries, and those which result from disease.

I. *Mechanical Injuries.*—Seven of the cases enumerated appear to have resulted from mechanical injury of some sort, and no doubt a careful search of the records of military surgery might increase this number. Wounds of the abdominal parietes involving the stomach are in most cases mortal. There are several instances, however, on record, in which they have healed up, and a speedy recovery has resulted.[1] In other cases, again, the margin of the wound in the stomach has contracted adhesions to that in the abdominal parietes, so as to constitute a permanent fistula.

Wounds or mechanical injuries, which may give rise to such fistulæ, appear to be of four sorts:

1. Incised wounds of the abdomen penetrating the stomach. Three of the seven cases were of this nature (1, 5, 6).

2. Gun-shot wounds (two cases, 14 and 16); the discharge either directly penetrating the stomach, as in the celebrated case of Alexis St. Martin, or inducing inflammation and subsequent sloughing of a portion of the wall of the stomach bordering on the original wound, as in the case of Maillot, a French lieutenant of infantry.

3. A blow over the stomach, without any laceration of the surface, may give rise to an abscess, which may open both into the stomach and also externally. This appears to have been the cause in Case 10. This patient received a blow in the epigastrium from a carriage shaft; an abscess followed, which in six months opened externally, and food swallowed was observed to escape from the opening. Case

[1] See, for instance, several remarkable instances mentioned by M. Hévin in the 'Mém. de l'Acad. Roy. de Chirurgie,' tom. i, part ii, p. 349, 1743; Gooch's 'Treatise on Wounds,' vol. i, p. 397; and Chelius's 'Surgery,' South's ed., vol i, p. 475.

15, also, was referred to an injury of this nature; but, inasmuch as eighteen years elapsed between its infliction and the appearance of the abscess, this explanation is doubtful.

4. Ulcerations from external pressure. The case of Catherine Ross is the only one recorded resulting from such a cause. The means adopted were not novel. Soldiers and sailors have been known to tie copper coins over open ulcers in their legs, to prevent the healing of these, and so elude their duty, or obtain a discharge from the service.[1] But that a female should have recourse to such a proceeding, at such a part of the body, and when fully warned of its danger, is certainly a unique mode of courting notoriety. Yet if one really desired to obtain such a fistula, a safer process could hardly be adopted, than that followed by Catherine Ross. Its slowness gives ample time for the stomach to contract adhesions to the abdominal parietes, and thus the escape of its contents into the peritoneal cavity is prevented.

5. It seems possible that an injury of the stomach *from within*, might end in the formation of an external fistula. There are many instances on record of persons having swallowed large foreign bodies, such as knives and other cutting instruments, and surviving; the foreign bodies, after the lapse of a considerable period, escaping by abscesses through the abdominal parietes.[2] I have not been able, however, to find any case in which food is stated to have escaped from the external opening; so that probably the wound in the gut in most cases closes before the abscess opens externally.

II. *Disease.*—The morbid conditions, of which we can speak with certainty as capable of giving rise to external fistulæ of the stomach, are only two, viz., cancer and simple perforating ulcer of the stomach. In both of these affections the escape of the contents of the stomach into

[1] Gavin, on 'Feigned and Fictitious Diseases,' p. 337.

[2] See M. Hévin's 'Memoir,' loc. cit.; also the notice 'De Prusse Cultrivore' in the 'Trans. Roy. Soc. Lond.,' 1696, vol. xix, p. 179.

the peritoneal cavity, on the occurrence of perforation, may be prevented by the previous contraction of adhesions to the abdominal walls, which last may also be perforated by a continuation of the original disease.

Opinions seem divided as to the relative frequency of these causes. Thus, Cruveilhier brings forward several cases depending on simple ulcer, but says he is not acquainted with an instance resulting from cancer, although he observes, it is not rare to meet with a cancerous pylorus adherent to the adominal wall;[1] and Dr. Brinton states, as the result of his researches, that he has found about six cases resulting from simple ulcer, but only one from cancer.[2] Rokitansky, on the other hand, says a cancerous ulcer may force its way through the abdominal parietes, but makes no mention of such an occurrence when speaking of the simple perforating ulcer.[3] Of the cases collected by myself:[4]

Six appear to have resulted from cancer (7? 9, 12, 19, 23, and 24); and 12 from simple perforating ulcer (2? 3, 4, 8, 11, 13, 15, 17, 18, 20? 21, 22).

Although, with regard to one or two of the older cases, the distinction between cancer and simple ulcer may be a matter of question, yet from a careful reference to the history, symptoms, and *post-mortem* appearances of these cases, I believe the statements made in the preceding table to be correct. Gastro-cutaneous fistula would thus appear to be twice as frequently the result of simple ulcer as of cancer. Now this is exactly the reverse of what has been found to hold good in the case of gastro-colic fistula.[5] This is probably owing partly to the difference in the relative situation of the two diseases. Simple ulcer, as shown by Dr. Brinton, is very rare in that part of the stomach

[1] 'Traité d'Anat. Path.,' ii, 566.

[2] 'Brit. and For. Med. Chir. Rev.,' January, 1856, p. 175; and April, 1857, p. 479.

[3] 'Path. Anat.' Syd. Soc. Ed., vol. ii, pp. 33—43.

[4] The figures refer to the number of the cases in the table, and will be repeated in the course of the paper, in order to facilitate comparison.

[5] See memoir by author on 'Gastro-Colic Fistula,' 'Edin. Med. Journ.,' August, 1857, p. 122.

nearest the colon, whereas of simple ulcers ending in perforation a very large proportion are found upon the anterior surface.[1] Again, as the length of time necessary to perforate the abdominal muscles and integuments must be considerable, in many cases of adherent cancerous stomach the patient no doubt sinks under the disease before the perforation is complete.

Of the six cases of cancer, in three the disease was situated at the pylorus (12, 19, and 24); in two (7 and 23) it was probably near the great curvature, but in one of these (7) there was also "scirrhus of the pylorus." In the sixth case (9) there was a large cancerous mass between the stomach and the arch of the colon, which had almost established a communication between these two portions of the digestive canal, as well as between the stomach and external surface.

In some of the cases of simple ulcer the fistula appears to have resulted from a gradual extension of the ulcerative process through the adherent abdominal parietes; but in many, perhaps most, the perforation of the stomach would seem to have excited a limited inflammation of the peritoneum, ending in chronic abscess, which has ultimately discharged its contents through the abdominal wall. Several cases have been recorded illustrative of the various stages of this mode of formation. Amongst these there is one remarkable instance which was brought before the notice of the Royal Medical and Chirurgical Society, by Mr. Lloyd, on November 14th, 1843, of an abscess pointing in the abdomen, which it had been resolved to open. The patient, however, died suddenly before this was done, and the abscess was found to communicate with an ulcer of the stomach.[2]

In some of the cases it may be questioned whether this abscess has not originated external to, and quite independently of, the stomach, ultimately bursting into this viscus, and also opening externally. Thus, in Case 3 the

[1] 'Brit. and For. Med. Chir. Rev.,' January, 1856, pp. 162, 172, and 173.

[2] 'Lancet,' 1843-4, vol. i, p. 273. See also Cruveilhier, op. cit., ii, 566.

abscess may possibly have originated in the liver; and in Case 8, in the abdominal parietes, from necrosis of the ribs. Hepatic and other abdominal abscesses, we know, may burst into the stomach,[1] and there seems no reason why they should not at the same time open externally. Should this explanation apply to any of the above cases, of course the number referred to simple ulcer of the stomach would be somewhat diminished.

Case 4 was ascribed to the erosion of a worm, but was probably a case of simple ulcer, in which a round worm may have come away through the opening.

As regards the situation of the opening in the stomach in the cases of simple ulcer, in

2 cases this was doubtful (2, 11);

3 cases it was near the fundus (4? 21, 22);

3 cases it was near middle of anterior surface (8, 15, 18);

4 cases it was at or near the pylorus (3, 12, 17, 20?).

In one of these last cases the opening was situated rather on the duodenal side of the pylorus; and perhaps it should have been excluded from this list. The ingesta, however, were observed to escape as readily from the opening in this case, as if it had been in the stomach itself.

It is a curious fact, that all the patients with cancerous fistula, and all but two in which it resulted from simple ulcer, were females; and in one of the two exceptional cases the sex is not recorded.

D. *Situation, Size, and other characters of the External Opening.*

1. *Situation.* The situation of the external opening in these cases resulting from disease of course depends, in a great measure, on the region of the stomach from which the fistula originates. Thus, if this be the neighbourhood of the pylorus, the external opening is generally near the umbilicus; or, if the disease commences in the anterior surface or near the fundus of the stomach, the external opening will occupy the epigastric or left hypochondriac region.

[1] Dr. Graves's 'Lectures on Clinical Medicine,' vol. ii, p. 232.

In four cases (8, 15, 16, and 22) the opening was situated between some of the left ribs, portions of which, or of their cartilages, had been removed. In Case 16 (St. Martin) this was the result of a gun-shot wound. In 15 and 22, and perhaps also in 8, the ribs appear to have been destroyed in the progress of a simple ulcer of the stomach. In Case 17, which originated in a perforating ulcer on the duodenal margin of the pylorus, the external opening was between the seventh and eight *right* ribs, below the right mamma. In connexion with this, it is important to allude to a case mentioned by Cruveilhier, in which a simple ulcer, commencing in the stomach, was found to have eroded the posterior surface of the ensiform cartilage.[1]

2. The *margins* of the opening are generally described as rounded, hard, or of cartilaginous consistence, in those cases resulting from either wound or simple ulceration. In two cases (11 and 21) the margins are noted as having been depressed or drawn in; and in a third (18) this retraction of the margins was so great, that the opening resembled a funnel. All these three were cases of simple ulcer of the stomach. In cases resulting from cancer, the margins of the opening may be ragged, or even gangrenous. In Catherine Ross they are red, glazed, and firmly cicatrized, and there appears to be a gradual transition between the skin and mucous membrane.

3. The *integuments surrounding* the opening for some inches are usually red, tender, and more or less excoriated, owing to the irritation from the constant escape of the fluid contents of the stomach.

4. The *size* of the external opening varies greatly. Thus—

In 7 cases it was doubtful (3, 4, 5, 7, 11, 17, 24);

In 6 cases it was less than 1 inch (10, 14, 18, 19, 20, 22);

In 7 cases it was about 1 inch in diameter, or easily admitted finger (1, 2, 6, 9, 12, 13, 16);

In 1 case it was 1½ by 1¼ inch (15.)

[1] Op. cit., ii, 566.

In 1 case it was 2 inches in diameter (8);

In 2 cases it was at first about 1 inch in diameter, but, shortly before death, increased to the size of a half-crown, or a whole crown piece (21, 23);

In 1 case it was 4 inches by 3 (25).

It will thus be seen that in none of the cases hitherto recorded did the opening at all equal in size that which exists in Catherine Ross (25).

E. *Escape of Food swallowed by Artificial Opening.*

In all of the cases this phenomenon was observed. In six cases (7, 11, 17, 18, 19 and 20) fluids only are stated to have escaped, and in these the opening was either very small, or no mention is made of its size. In all the other cases, whatever the patient swallowed immediately escaped from the opening. This escape of food has usually been prevented by some artificial contrivance, such as a plug of linen, gutta percha, &c.; but it has always been found difficult to prevent the fluid ingesta from oozing out by the sides of the plug. In the case of Alexis St. Martin, the necessity for such an artificial plug became, after a time, superseded by a natural one, consisting of a slight protrusion of the coats of the stomach, so that food only escapes on the depression of this. It is probably to this provision of nature for preventing the escape of food, that this individual's good health and long life are in no small degree to be attributed. A tendency to the formation of a similar natural plug was also observed in Cases 15 and 21; but in the case of Catherine Ross the opening is so large that the whole stomach becomes everted through it.

It is to be remembered, in reference to the diagnosis of such cases, that a fistulous opening in the abdominal walls, from which food escapes immediately on being swallowed, does not necessarily communicate directly with the stomach. We have already seen that in Case 17 the opening was rather on the duodenal side of the pylorus. Again, in the museum of Charing Cross Hospital there is a preparation

(G. 21) of a cancerous stricture of the arch of the colon, in which the colon above the stricture communicated with the duodenum, and also, through the abdominal parietes, with the external surface. In this case, any fluid taken into the stomach, flowing by the duodenum into the colon, made its appearance, in a few minutes, at the umbilicus.

F. *Duration of the Fistula, and possibility of Cure.*

The length of time during which an individual may live with a fistulous opening into the stomach, varies very greatly with the cause on which the fistula depends.

1. As might be expected, all the cases dependent on cancer have proved very speedily fatal, three months being the longest period during which a person has lived with a cancerous fistula. Of the six cases of cancer, the duration of life, after the formation of the fistula, was as follows:

In 1 case it was doubtful (7).

In 2 cases it was 3 weeks (12, 19).

In 1 case it was 4 weeks and 3 days (23).

In 1 case it was 5 weeks (24).

In 1 case it was 3 months (9).

2. Wounds of the stomach, as already stated, are generally mortal; or, in a few instances, become speedily cured. The fistula, however, to which they now and then give rise, may continue for years. Thus—

In Case 6 it lasted 2 months, and was cured.

In Case 1 it lasted many years.

In Case 10 it lasted 10 years, and was cured.

In Case 5 it lasted 11 years.

In Case 14 it lasted 8½ years at least; as the patient was not dead when the case was reported.

In Case 25 it lasted 4 years, and still alive.

In Case 16, it lasted 35 years, and still alive.

3. Of the cases resulting from simple ulcer—

In Case 17 the patient lived 26 days, but died with tubercles in lungs.

In Case 11 opening lasted a "short time," and closed up spontaneously.

In Case 18 opening closed after 3 days; 3 years after, it opened again, and on the 4th day patient died.

In Case 13 patient lived 3½ months, and died from effects of cold.

In Case 20 opening lasted 6½ months, and was cured.

In Case 3 patient lived 1½ year.

In Case 21 opening lasted 7 years, but was twice closed during this period for upwards of a year.

In Case 15 opening lasted 8½ years.

In Case 4 opening lasted 12 years.

In Case 8 opening lasted 27 years.

In Case 2 opening lasted "many years."

In Case 22 opening lasted 3 years, and still alive in 1853.

Thus, if we except the first four cases, in which either the opening closed up spontaneously or death resulted from independent causes, the most of the others lived for many years. It is to be observed that in three cases (11, 18, and 21) the opening closed up spontaneously. In Case 11 the cure appears to have been permanent; in Case 18 the fistula reopened after an interval of three years; and in Case 21 the opening was twice closed for upwards of a year, and as often reopened.

This leads us to consider the question of the possibility of curing such cases. In six of the cases the opening became closed (2, 10, 11, 18, 20, 21). Two of these cases (2, 10) resulted from wound; the other four from simple ulcer. In four of the six cases the opening appears to have closed spontaneously (2, 11, 18, 21); and in two of these (18 and 21) the fistula reopened. In two cases only does the obliteration of the fistula appear to have been the result of treatment. In Case 10 a fistula, resulting from an injury, of ten years' duration, was closed by simply making the patient, who had been in the habit of always going about, keep quiet in her bed for a few weeks; and in Case 20 a cure was effected by ingenious local appliances, of such a nature as to prevent the contact of the irritating contents of the stomach with the surrounding skin, and at the same time approximating the edges of the opening by means of

the gradually increased compression of a circular bandage, while at the same time the patient was nourished chiefly *per rectum*.[1] In none of the cases does any cutting or paring of the edges of the wound appear to have been attempted. Dr. Keith seems to consider such a procedure practicable in the case of Catherine Ross, provided that she herself would throw no obstacle in the way. It is much to be feared, however, that in any such operation the contact of the gastric juice and the distention of the stomach by food would very much interfere with union by the first intention, while, at the same time, there would be a risk of exciting dangerous inflammation of the stomach.

G. *General health of persons with Gastro-cutaneous Fistulæ.*

It is astonishing to observe how little influence the existence of a gastro-cutaneous fistula has upon the general health. In most of the cases resulting from wound or simple ulcer the patients are stated to have enjoyed excellent health. This was particularly remarkable in Wencher's case of a woman, who lived for twenty-seven years with such a fistula, following her ordinary avocations; and is still more so in the case of Alexis St. Martin, who, during the last thirty-five years that the fistula has existed, has married and become the father of several children, earning a livelihood for his family by a laborious occupation. In Catherine Ross the state of general debility is more to be attributed to a general derangement of the entire nervous system than to the effects of the fistula.

The principal abnormal symptoms which have been observed as dependent upon the fistula are, great thirst, increased appetite, obstinate constipation, deficient secretion of urine, and amenorrhœa. The most of these symptoms are directly traceable to the constant escape of the ingesta

[1] For a fuller account of the treatment in this case, see South's ed. of Chelius's 'Surgery,' vol. i, p. 477.

from the abnormal opening. This continual drain creates a craving for an additional supply, while at the same time it cuts off the material necessary for the production of fæces and urine. In Case 15 the patient is said to have eaten as much as any three other women, and in Catherine Ross the feeling of hunger sometimes amounts to actual pain. There may be but one stool every three, four, or even twelve days; and this has, in several instances, been observed to consist of hard rounded scybala, like the droppings of a sheep.

Vomiting appears to have been a very unusual symptom in all of the cases, although it may have been very urgent before the formation of the fistula. In Case 15, however, there was an irresistible desire, three or four hours after each meal, to remove the plug, and evacuate the contents of the stomach. This same person also could never sleep unless she had previously washed out the stomach with an infusion of chamomile.

In Case 8 a vicarious discharge of pure blood from the mucous membrane of the stomach occurred at every menstrual period.

H. *Physiological observations on the Stomach, &c.*

Cases such as those we have been considering afford the opportunity of observing during life in the human subject—

1. The physical characters and properties of the coats of the stomach, such as its peculiar movements, the colour, &c., of the mucous membrane, &c.; and

2. The physical and chemical characters of the gastric juice, and its digestive power over different nutritive principles and articles of diet.

I. Observations of the former class have been made in six of the cases (8, 14, 15, 16, 21, and 25).

In Case 8, when food was swallowed the stomach could be seen occasionally to contract violently so as to expel the food with force. No mention, however, is made of the condition of the diaphragm and abdominal muscles.

In Case 14 the surface of the stomach is described as "d'un rouge très vif, et plissé dans tous les sens." Undulating movements could also be seen passing along the surface, which were increased by the contact of food. When the patient made the effort of swallowing, a lighted candle held to the fistula became agitated.

In Case 15 the interior of the stomach in the morning, before any food was taken, was said to be "d'un rouge vermeil, enduit d'un mucus luisant." It was disposed in longitudinal plicæ; and two sets of undulatory movements were observed, one towards the pylorus and the other towards the fistula.

In Case 16 Dr. Beaumont describes the mucous membrane as of a pale pink colour, increased in hue when food entered the stomach. Vermicular movements were observed, which were augmented into a churning motion by the contact of food. Mechanical irritation of the stomach produced faintness, but not pain.

This last observation was also noted in Case 21.

In Catherine Ross (25) the mucous membrane is of a vermilion red colour, disposed in rugæ, along which undulating movements can be observed. The freest manipulation of the stomach only produces faintness, not pain.

The observation made upon Catherine Ross, as regards the mechanism of vomiting, is of peculiar interest. During the last century there was a great controversy as to the precise mode in which this act is effected, and even at the present day we find the greatest variety of opinions expressed by different physiologists concerning it. These various opinions may be reduced to the four following:

1. Down to the end of the seventeenth century the almost universal belief appears to have been, that vomiting was the result of the convulsive contractions of the stomach itself; and, more recently, Haller, Rudolphi, and others, have considered the contraction of the surrounding muscles as unnecessary, or, at most, only auxiliary.

2. Another class of physiologists, commencing with Bayle

of Toulouse[1] (A.D. 1681), and including John Hunter,[2] have maintained that the stomach is nearly or entirely passive during vomiting, and that this action is excited by extraneous pressure of the diaphragm and other abdominal muscles. The greatest supporter of this theory has been Majendie, who drew his conclusions from observing the non-occurrence of vomiting after dividing the phrenic nerves, so as to paralyse the diaphragm, and removing the abdominal parietes; and the fact that it did take place when a pig's bladder was substituted for the stomach, the muscles remaining intact.

3. A third class, while they have admitted that the act has a double origin, depending on the contraction of the stomach, assisted by that of the abdominal muscles, have yet maintained that it is only the muscles of expiration which by their contraction compress the stomach; and that, after the first inspiration, the diaphragm becomes relaxed. This opinion, that vomiting is mainly an *expiratory* act, was propounded by Lieutaud, so long ago as 1752;[3] and in modern times has been strongly advocated by the late Dr. Marshall Hall.[4] It is also the view which is taught by Dr. Carpenter in his 'Principles of Physiology.' "It is not true," Dr. Carpenter observes, "that the diaphragm actively co-operates in the effect of vomiting;"[5] although he admits that this muscle, by its being passively fixed, may supply a firm surface, against which the stomach is pressed.

4. Lastly, there are others who believe that, while the stomach itself contracts, the vomitive act is in the main effected by both the muscles of expiration and inspiration; in other words, that the diaphragm remains contracted after the expiratory muscles come into play, and that, by the simultaneously opposing forces, the stomach is so compressed as to be evacuated of its contents. Dr. David Anderson, in an inaugural thesis presented to the medical faculty of the

[1] 'Dissert. sur quelques points de Physique et de Médecine,' Toulouse, 1681.
[2] 'Hunter's Works,' edited by Palmer, vol. iv, p. 92.
[3] 'Mém. de l'Acad. Roy. des Sciences,' tom. lxviii, p. 230.
[4] 'Quarterly Journal of Science,' June, 1828, vol. xxv, p. 388.
[5] 'Principles of Human Physiology,' fifth ed., p. 69.

University of Edinburgh, in 1842, proved by experiment that the diaphragm is rigidly contracted during vomiting. This he ascertained by laying open, and introducing his hand into, the abdomen of dogs which were vomiting from the effects of tartar emetic.[1] The correctness of this view is also supported by the powerful advocacy of Messrs. Todd and Bowman,[2] and is corroborated by what has been observed in the case of Catherine Ross. This case shows that although the coats of the stomach itself do undoubtedly contract, the principal part of the act is played by both the diaphragm and abdominal muscles. The upper wall of the stomach can be distinctly seen to be pressed down by the diaphragm. The contraction of the stomach appears to be confined to the pyloric region, and to have for its object chiefly the closure of the pylorus, so that when the stomach becomes compressed between the diaphragm and abdominal parietes, the only exit for its contents, in the natural condition of the parts, must be through the œsophagus. From all the observations which have been made on the matter, the following would appear to be the mechanism of an act of vomiting:

1. A long inspiration followed by partial closure of the glottis.

2. Contraction of the pyloric end of the stomach, with relaxation of the cardia and œsophagus.

3. Persistent contraction of the diaphragm, with rigid contraction of the recti and oblique muscles.

In connection with this subject, I may be allowed to correct an erroneous statement of facts, which is to be found in several English works on physiology. Dr. Carpenter, in quoting a case, observed by a French surgeon, M. Lépine, to prove that the walls of the stomach contract during the act of vomiting, observes: "In this case, the abdominal parietes having been accidentally laid open in the human

[1] 'Lond. and Edin. Monthly Journal of Medical Science,' 1844 p. 1.
[2] 'Physiological Anatomy,' vol. ii, p. 213.

subject, and the stomach having wholly protruded itself, it was seen to contract itself repeatedly and forcibly, during the space of half an hour, until by its own efforts it had expelled all its contents except gases."[1] A similar account of the case has been given by Mr. Paget;[2] and by Messrs. Todd and Bowman;[3] the latter, however, quote from Mr. Paget. How completely different were the phenomena really presented by this case, will be at once apparent on reading the following extract from the original account.

"Pendant tout le temps que l'estomac fut hors de l'abdomen, M. Lépine ne l'a ni vu, ni senti se contracter, quoique, dans le but de provoquer ces contractions, il ait appliqué sur cet organe ses mains, préablement trempées dans l'eau froide. A peine la reduction était elle opérée, qu'aux nausées, et aux efforts *inutiles pour vomir* succédèrent de veritables vomissements, qui amenèrent au dehors les aliments, que le malade avait pris une demi-heure avant la blessure."[4]

II. There are only three cases which have hitherto been taken advantage of for making observations on the chemical and physical properties and the digestive powers of the gastric juice. (15, 16, and 22.)

1. In the first of these (15), that of Magdelaine Goré, several chemical analyses of the gastric juice, and of the food undergoing digestion, were made by M. Clarion, more than half a century ago. M. Clarion found the gastric juice to contain 9 parts in 1000 of muriates, and 16 parts of an animal principle, precipitated by both alcohol and tannin. He also ascertained that, although the contents of the stomach during digestion were always acid, the fluid collected in the morning before breakfast was perfectly neutral, and had a strong analogy to saliva.

[1] Op. cit., p. 69.

[2] Report on Physiology, 'Brit. and For. Med.-Chir. Rev.,' Jan., 1845, p. 273.

[3] Op. cit., vol. ii, p. 213, note.

[4] 'Bulletins de l'Acad. Roy. de Méd.,' 1844, vol. ix, p. 147.

2. Every one is familiar with the valuable observations made by Dr. Beaumont, in the case of Alexis St. Martin (16), as regards the relative digestibility by the gastric juice of different articles of diet, &c. It would be quite apart from the object of this paper to enter into these here, I would merely call attention to some chemical analyses of the gastric juice obtained from St. Martin, which have been made since Beaumont's observations, and which are less generally known. These analyses have been performed in 1833, by Professor Dunglison, of Philadelphia;[1] and within the last two years, by Dr. F. S. Smith, of the Pennsylvania College.[2] The results arrived at by these two experimenters are, however, somewhat conflicting. Thus Professor Dunglison obtained *hydrochloric acid* with great readiness; and concluded that this acid was the principal, if not the sole, source of the gastic juice: whereas Dr. Smith states, as the result of his experiments, that if hydrochloric acid exists at all, it is in very small quantity, and "that the main agent in producing the acid reaction, is *lactic acid*." Dr. Smith found the fluid from the empty stomach always neutral.

3. Within the last few years a valuable series of observations, as regards the physical and chemical properties of the gastric juice, have been made on Case 22, and recorded in a thesis by Otto v. Grünewaldt. Professor Schmidt obtained the following results from his analyses: first, that the human gastric juice contains about 36 parts in 1000 of pepsin; secondly, that it contains butyric, and probably also lactic acid; and thirdly, that it contains no free hydrochloric acid, although, according to the experiments of the same chemist, this acid exists in considerable quantity in the gastric juice of some of the lower animals. Lastly, the fluid obtained from the empty stomach, early in the morning, before a meal, was always neutral.[3]

[1] Dunglison's 'Human Physiology,' seventh ed., vol. i, pp. 585 6.

[2] 'Philadelphia Med. Examiner,' July and Sept., 1856, and 'Brit. and For. Med.-Chir. Rev., Jan., 1857, p. 241.

[3] See References in Table; also Carpenter's 'Principles of Human

4. One observation, which has been made in all these cases, has been confirmed in Catherine Ross. This is the non-acidity of the fluid obtained from the empty stomach. No other experiments have been undertaken upon her. As far as regards the size of the opening, no case could be more favorable for examining the interior of the stomach, or for introducing or removing food. Indeed, it would not be a difficult matter, by means of an elastic catheter, to draw off the contents of the duodenum for examination. The patient, however, is in such a debilitated state of health—bed-ridden for nine years and unable to stand, that it has been feared that any observations made on the properties of her gastric juice would at present be of small value. In addition to this, Catherine Ross's propensity to practise deceit would require careful vigilance on the part of the experimenter. In reference, however, to her general health, it may be observed, that since she has been under Dr. Keith's observation, she has gained considerably in flesh and strength; and if she could only be induced to get up and go about, the infatuated act of this poor woman might yet prove a benefit to science.

Physiology, fifth ed., p. 81. The analysis, as given by Dr. Carpenter, does not agree with the original.

With the author's respects

"Preliminary Account of an Inquiry into the Functions of the Visceral Nerves, with special reference to the so-called 'Inhibitory System.'" By Joseph Lister, Esq., F.R.C.S. Eng. & Edin., Assistant Surgeon to the Royal Infirmary of Edinburgh; in a Letter to Dr. Sharpey, Sec.R.S. Received August 13, 1858. Communicated by Dr. Sharpey.

My dear Sir,—The fact that the irritation of visceral nerves sometimes causes arrest of the movements of organs supplied by them, as shown by Edward Weber's experiment of stopping the action of the heart by stimulating the vagus, and by Pflüger's more recent observation that the application of galvanism to the splanchnic nerves produces quiescence of the small intestines, appears to me to have an intimate bearing upon the question how inflammation is developed through the medium of the nervous system at a distance from an irritated part; and as the nature of the inflammatory process has lately engaged my especial attention, I have been led to make an experimental inquiry into this "inhibiting" agency, the true interpretation of which is, as you are aware, still *sub judice*. I now propose to state the principal results at which I have arrived, reserving further details for a more extended communication which I hope soon to offer to the Royal Society.

The view which has been advocated by Pflüger*, and I believe very generally accepted, viz. that there is a certain set of nerve-fibres, the so-called "inhibitory system of nerves" (Hemmungs Nervensystem), whose sole function is to arrest or diminish action, seemed to me from the first a very startling innovation in physiology; and you may possibly recollect my mentioning to you in conversation, when in London last Christmas, my suspicion that the phenomena in question were merely the effect of excessive action in nerves possessed of the functions usually attributed to them. On further reflection upon the subject, the consideration of the contraction produced in the arteries of the frog's foot by a very mild stimulus, as compared with the relaxation of the vessels caused by stronger irritants acting through the same nerves, confirmed my previous notions.

* Eduard Pflüger ueber das Hemmungs Nervensystem, 1857.

For I could hardly doubt that the cause of the quiescence of the heart or intestines on irritation of the vagus or splanchnic nerves was analogous to that of arterial dilatation in the web, and that, provided a sufficiently mild stimulus were applied to the so-called "inhibitory nerves," increased action of the viscera would occur, corresponding to the vascular constriction.

To test the truth of this hypothesis, I made several experiments between the 17th of June and the 14th of July of this year, with regard to the movements of the heart and intestines. The means used for stimulating the nerves and spinal cord were sometimes mechanical irritation, but more commonly galvanism, applied with a magnetic coil battery of a single pair of plates, the strength of which could be regulated in a rough way, with great facility, by the height at which the acid solution stood in the jar and the extent to which the rods of soft iron were inserted in the helix. The mildest action employed was such as was but just perceptible to the tip of the tongue, placed between the fine silver-wire extremities of the poles, when the rods were fully in the helix, but inappreciable after their complete withdrawal; the spring carrying the magnetic bar being made to vibrate by a touch with the finger: the greatest action of the battery, on the other hand, was so powerful as to elicit sparks when the poles were applied to the tissues.

My attention was first directed to the intestines, and it may be well to mention first all the results obtained with reference to them. The animals operated on were generally rabbits, they being very easily managed, and also favourable for the purpose on account of the large amount of movement which occurs in their intestines. Chloroform was generally not administered, on account of its depressing effect upon the action of the nervous centres.

In the first experiment, the ends of the poles having been fixed to the spinous processes of the ninth and twelfth dorsal vertebræ, according to Pflüger's original method, and the intestines allowed to protrude through a wound in the abdominal parietes, a series of interrupted currents were transmitted, a very small amount of acid being in the jar, and the rods fully in the helix. The effect was complete relaxation and quiescence of the small intestines, which had been previously in considerable movement, while the muscles of the limbs were thrown into spasmodic action; but on the discontinuance

of the galvanism the previous intestinal motion returned. The rods were then removed from the helix, and the battery, thus diminished, was applied on several occasions, with markedly increased action of the intestines in every instance during the first twenty-five minutes. In the next half hour the increase of action from the galvanism, though still distinct, was less strongly marked; and at the end of that period, the rods having been reintroduced, the inhibiting influence was also found to be much less complete than before, indicating that the parts of the nervous apparatus concerned were in a less active condition, no doubt in consequence of exhaustion. The arches of the tenth and eleventh dorsal vertebræ having been removed before the experiments with galvanism, I subsequently introduced a fine needle into the exposed part of the cord, with the effect of causing in repeated instances increased movements of the intestines, which were especially striking on account of the occurrence of peculiar local contractions not seen at other times. Further observations upon this animal tended to confirm those which have been mentioned, as did an experiment of the same kind performed the next day upon another rabbit.

I afterwards found that the best mode of proceeding was to remove the skin and one or two layers of muscles from a portion of the abdomen till the parietes were sufficiently thinned to permit the intestines to be distinctly seen through them; by this means the complication produced by exposure of the intestines to the atmosphere was avoided, and the most satisfactory results were obtained; the increase of the peristaltic movements during the transmission of extremely feeble shocks being strikingly apparent and constant on every occasion. During the experiment performed in this way I noticed several times that a violent struggle on the part of the rabbit, when the intestines were in pretty free movement, was followed by absolute and universal quiescence of those organs for several seconds; this appeared to me of great interest, as proving that the inhibitory influence is certainly sometimes exerted in the natural actions of the animal, and is not merely the result of artificial stimulation.

In the course of the above experiments several other observations were made. In the first place I verified the statement of Pflüger, that if, when the intestine is lying relaxed under the inhibiting

influence of galvanism applied to the spine, a particular part be irritated, local contraction occurs, but is not propagated to neighbouring parts. This fact is of fundamental importance, since it proves that the inhibitory influence does not operate directly upon the muscular tissue, but upon the nervous apparatus by which its contractions are, under ordinary circumstances, elicited.

Another point which seemed to require investigation was the well-known increase of peristaltic action which takes place after death, and which continues in spite of cutting off the mesentery close to the gut. Those who believe in a constantly restraining function of certain nerves during life might argue that the intestine has always a tendency to such active movements, but is kept in check by the "inhibitory nerves," and released from their control when they have lost their power after death. A different explanation, first suggested, I believe, by Bernard, is that the increased action of the intestines is the result of failure of the circulation in the part; and to this view I felt disposed to agree, in consequence of having noticed curious irregular contractions in the arteries of the frog's foot from a similar cause. In order to decide the question, I tied three adjoining arterial branches in the mesentery of a rabbit, thus depriving about 3 inches of the intestine of its circulation; the parts so affected being accurately defined by the extent of absence of pulsation in the minute vessels close to the gut. In about a minute and a half, vermicular movements commenced in this part; the rest of the intestines being at the time very quiet. Powerful interrupted galvanic currents were then transmitted through the posterior dorsal region of the spine, with the effect of causing perfect quiescence of the whole of the intestine, including the part whose arteries had been tied. After cessation of the galvanism the movements recurred in the portion devoid of circulation, while elsewhere they were almost entirely absent. This experiment was repeated on another occasion with similar results. In one of the cases I divided the mesentery close to the gut, after ligature of the vessels, but no change took place in the character of the movements which had been previously induced, indicating that the increased action in these cases had been of the same nature as that which results from death. The arrest of the movement on the application of galvanism proved that the delicate operation of ligature of the mesenteric vessels had been performed without

injury to the adjacent nervous branches; and it therefore followed that the movement in the parts supplied by those vessels was not due to any injury of the nerves, but simply to the arrest of circulation. It further appears from these experiments, that, in whatever way the cessation of the flow of blood through the vessels operates in increasing the peristaltic action, it does so through the medium of the nervous apparatus, and not by directly influencing the muscular tissue. For, in the latter case, the movement would have continued in spite of the inhibiting influence, which, as we have seen, has no effect upon muscular irritability.

The fact that the movements continue in a portion of gut deprived of its mesentery, proves that the nervous apparatus by which the muscular contractions are induced and coordinated in *post mortem* peristaltic action, is contained within the intestine.

The distinction between the coordinating power and muscular contractility was very strikingly shown in the further progress of one of these experiments. The peristaltic movements of the portion of gut supplied by the ligatured arteries ceased entirely about twenty minutes after the vessels were tied, and the surface of the gut became there perfectly smooth and relaxed, contrasting strongly with the wrinkled aspect of other parts. But muscular irritability had outlived the coordinating power, as was shown by energetic, purely local contraction taking place in a part pinched. Similar observations confirmatory of this point were afterwards made upon a rabbit which had died of hæmorrhage an hour before.

The mechanism by which the muscular contractions are regulated is, doubtless, the rich ganglionic structure lately demonstrated in the submucous tissue by Dr. Meissner of Bâle*. Professor Goodsir gave me the first information of this anatomical fact on my mentioning to him the foregoing physiological proofs of the existence within the intestines of a coordinating apparatus distinct from the muscular tissue. I have since verified Meissner's observations, and found abundant well-marked nerve-cells in the submucous tissue of the Ox, exactly corresponding with his descriptions.

But while muscular irritability outlives the coordinating power in the intestines, the latter lasts much longer than the inhibiting

* Henle and Pfeufer's Zeitschr. 2nd series, vol. viii.

property in the spinal system, for I find that Pflüger's experiment does not succeed in a dead animal, unless performed soon after death, although the intestines may continue to move for a long time.

In another experiment I divided with fine scissors, at a little distance from the intestine, all the visible branches of nerves in a portion of mesentery corresponding to an inch and three-quarters of the gut, leaving the vessels uninjured. No effect was produced on the peristaltic movements, which happened to be pretty active at the time, and continued the same at the seat of the operation as elsewhere. To ascertain whether the division of the nerves had been thoroughly effected, I now transmitted powerful galvanic currents through the spine, as in former experiments; when all movements ceased in the intestine, except in the small piece whose nerves had been cut, which continued in vigorous action as before. The persistence of the vermicular motion after complete division of the mesenteric nerves shows that the movement which occurs during life, like that which takes place *post mortem*, is effected by a mechanism within the intestine; and its continuance in the portion of gut so treated, while other parts were relaxed, on the application of galvanism to the spine, proves that the inhibiting influence acts through the mesenteric nerves, whose integrity is necessary to the effect.

This being established, it follows that if a quiet state of the intestine, such as very frequently occurs in its natural condition, were due to a controlling agency on the part of the so-called "inhibitory system," the complete division of the mesenteric nerves supplying a portion of gut which is at rest, would liberate it from this restraint, and movement would be the result. I performed the operation in one case under such circumstances, but the portion of intestine concerned remained as tranquil as the rest.

To sum up the above, it appears that the intestines possess an intrinsic ganglionic apparatus which is in all cases essential to the peristaltic movements, and, while capable of independent action, is liable to be stimulated or checked by other parts of the nervous system; the inhibiting influence being apparently due to the energetic operation of the same nerve-fibres which, when working more mildly, produce increase of function.

After the above conclusions had been arrived at, my attention was

directed by Professor Goodsir to a paper by Dr. O. Spiegelberg, published last year, in which he shows that the movement of the intestines is increased by mechanical irritation of the cord. His results are particularly satisfactory, as having been obtained incidentally during an inquiry into the movements of the uterus, and so without any preconceived theory*. Spiegelberg also attributes the increased peristaltic action after death to arrest of the circulation; having found that the same thing occurs during life, when the aorta or vena cava is compressed above the origin of the mesenteric vessels.

To proceed to the experiments upon the cardiac movements: some of these consisted in irritation of the vagus in rabbits, and this was followed by different results in different instances: thus, on one occasion the pinching of the cardiac end of the left nerve, divided in the neck, was followed by considerable increase in the number of beats as felt through the walls of the chest, but similar treatment of the right nerve afterwards caused great depression of the heart's action. Again, in one animal the evidence obtained from mechanical irritation of the vagus was almost entirely negative. In another case, the left vagus having been exposed, feeble galvanic currents transmitted through the nerve, isolated by a plate of glass placed beneath it, were succeeded by slight increase in the number of contractions. The strength of the battery having been then increased by introducing the rods into the helix, it produced first irregularity, and then complete arrest of the action of the heart, which had been previously exposed. No sign of recurrence of contraction appearing, I filled the jar to the top with acid solution, and sent powerful currents through the vagus, with the instantaneous effect of reviving the action of the heart, which, on their immediate discontinuance, continued to beat, though feebly, for several minutes. During this time I again applied the galvanism very mildly, and the result was great increase in the number of beats on several successive trials. The apparent discordance of these facts is, I believe, partly owing to differences in the state of the nerves in different cases as respects irritability and exhaustion, as will be better understood from the sequel; and, on the whole, the experiments appear to show that, in a

* Henle and Pfeufer's Zeitschrift, 3rd series, vol. ii. pt. 1.

healthy state of the nervous system, very gentle irritation of the vagus increases the heart's action, while a slightly stronger application diminishes the frequency and force of its contractions. This conclusion is in harmony with an observation which I made incidentally upwards of a year ago, that irritation of the posterior part of the brain of a frog with a fine needle was repeatedly followed by improvement in the circulation, whereas it was by the application of a stronger stimulus, that of galvanism, to the same part of the cerebro-spinal axis that Weber first induced an inhibitory action on the heart.

It is said, on apparently good authority*, that division of the vagus in mammalia is invariably followed by increase of the action of the heart; this, if true, would be a strong ground for believing in an inhibiting influence constantly operating upon it through this nerve. But it is also stated that the same thing does not occur in frogs; and this circumstance appeared to me to throw much doubt upon the evidence regarding mammalia. I therefore made careful experiments on the effects of cutting both vagi, once upon a calf and four times upon rabbits; taking the number of the heart's beats immediately before and immediately after section of each nerve by the momentary stroke of a sharp pair of scissors. In no case was the rate increased at all by the operation, and the very gradual diminution in frequency that commonly took place appeared to depend on general exhaustion from other circumstances attending the experiment. In one rabbit, in which I had removed the skin and pectoralis major from the præcordial region, so as to see the movements of the heart distinctly through the transparent pericardium and intercostal muscles, I noticed particularly that the strength of the contractions, as well as their frequency, remained quite unaffected by the division of the vagi. From these facts I feel warranted in concluding that, whatever may occur under exceptional circumstances, there is certainly no constant control exercised over the heart's action through those nerves.

The influence of the spinal system upon the heart is, however, very apparent after a struggle, which almost invariably increases the frequency and force of the beats; and I found that this continued

* Pflüger, *op. cit.*

to be the case after division of both vagi, implying that those nerves are not the only channels through which this influence is transmitted. A new field of investigation was thus opened. For, supposing the inhibitory agency to be simply the greater action of an ordinary nerve, it would probably not be exercised exclusively by the vagus, but also by the other nerves connecting the cerebro-spinal axis with the cardiac ganglia, viz. the sympathetic branches in the neck; in which case the action of the heart should be increased or diminished, according to the strength of the stimulus, by the application of galvanism to the cervical region of the spine after the pneumogastric nerves had been cut.

In an experiment performed with this view, the poles having been fixed to about the fourth cervical and fifth dorsal spinous processes, and both vagi divided in the neck, galvanic currents only just perceptible to the tip of the tongue were first transmitted. This excessively feeble action of the battery, though apparently not very favourably situated for influencing the cord, produced marked effects upon the heart's action, increasing the number of beats, which were about forty in ten seconds, by from three to ten in that period. This effect having been observed for a considerable time, the rods of soft iron, which had been till then only inserted half-way in the helix, were pushed fully in. The battery, thus strengthened, instead of increasing, as before, the rate of the pulsations, diminished it by two in ten seconds on several successive trials. On again half withdrawing the rods, the galvanism, when applied, again increased the number of beats. A little more of the acid solution was afterwards poured into the jar of the battery, when the stronger currents which it produced reduced the number by about five in ten seconds.

Yet distinct as was this inhibiting influence, the shocks were still quite tolerable to the tongue even when the rods were fully in the helix.

These results were of great interest, as proving how slight an increase of the feeble stimulus which promoted the action of the heart sufficed to produce the opposite (inhibiting) effect. But it was by no means clear that the influence had not been exerted through cardiac branches arising from the vagi above the parts where they were divided, or even through the trunks of those nerves, which might

possibly have been affected by the galvanism acting through the superjacent spinal column. In order to eliminate the vagi completely, I divided in another rabbit all the soft parts in front of the spine, except the trachea and œsophagus, at the level of the cricoid cartilage, having previously cut each carotid artery between two ligatures. The incisions were carried fairly down to the bodies of the vertebræ, and outwards beyond the tips of the transverse processes, so as to ensure the section not only of the vagi and their branches, but also of the sympathetic cords, with any filaments of those nerves which they might contain. Also the poles of the battery were fixed to the spinous processes of the seventh dorsal and first lumbar vertebræ, so as to avoid all possibility of direct action of the galvanism upon either the vagi or other cardiac nerves. Feeble currents being then transmitted, diminution of the number of beats to the extent of two to four in ten seconds occurred in several successive trials, the results being so constant as to leave no doubt that they were produced by the galvanism.

It may appear almost incredible that such extremely mild galvanic currents, applied through the spinous processes of the posterior dorsal region, should be capable of thus affecting the heart; but that their effects were really very considerable, was clear from the further progress of this experiment and from others somewhat similar, which showed that this apparently trivial stimulation gradually exhausted the part of the nervous system through which the heart is acted on by the cord. Thus, in one case, currents only just perceptible to the tongue, transmitted for about thirty seconds at a time through the lower cervical and upper dorsal regions of the spine, at intervals of nine minutes on the average during two hours and twenty minutes, produced at first decided increase of the heart's action, but during the last hour failed to affect it at all. The strongest possible action of the battery which, as proved by other experiments, would, at the outset, have entirely arrested the cardiac movements, was then set on, but with no effect whatever on the organ.

When partial exhaustion has occurred, a much stronger galvanic stimulus is required, to produce the same effect upon the heart, than at the commencement of an experiment; and thus an action of the battery which, when first applied, causes marked diminution in the number of beats, may after a while come to have the opposite effect,

and increase the heart's action as decidedly as it had previously lowered it; while at an intermediate period it may seem to have no influence at all. This principle gives the clue to understanding what had before appeared incomprehensible in these experiments, showing that facts which at first seemed utterly inconsistent, were really perfectly harmonious. The case before related, in which revival of the heart's action resulted from powerful stimulation of the vagus, which, had the organ been contracting as usual, would have arrested its movements and probably finally destroyed them, will now be understood. I have seen other analogous cases of revival of action by very powerful galvanism, which under ordinary circumstances would have arrested it, viz. twice in the heart and twice in the intestines. The observation published so long ago as 1839 by Valentin*, that mechanical or chemical irritation of the vagus in the neck of an animal recently dead, and with the nerves consequently enfeebled, causes contraction of the ventricles, admits of a similar interpretation, as also does a corresponding fact regarding the splanchnic nerves, given without explanation by Kupfer and Ludwig, in a paper just published†, viz. that they lose their inhibitory influence a certain time after death, and acquire a motor power over the intestines.

Two more experiments require mention, as they exclude the possibility of the agency in them, of either the vagi or the part of the brain from which the vagi spring, having been performed upon decapitated rabbits. In one of these cases, the carotids having been tied near the head, the neck was completely severed behind the first vertebra, care being taken to avoid hæmorrhage from the vertebral arteries, and artificial respiration, for which provision had been made, was carried on for an hour and a half after decapitation. The results of moderate galvanism, applied to the posterior dorsal region of the spine, to which the poles had previously been attached, were at first not distinct, but afterwards decided increase of action was produced by it when applied at intervals during half an hour; the effect being perfectly apparent in the heart which lay exposed before me. Exhaustion of the nerves concerned having then taken

* Valentin, De Functionibus Nervorum, p. 62.

† Henle and Pfeufer's Zeitschrift, 3rd series, vol. ii. pt. 3.

place, the most powerful action of the battery failed to influence the character of the contractions.

In the other case, the poles having been fixed as before, and the head similarly removed, powerful galvanic currents were immediately transmitted. The pulsations of the heart in the opened chest at once fell from thirty-five to sixteen in ten seconds, but rose again to twenty on the removal of the stimulus.

Hence it is clear that the sympathetic branches connecting the cord with the cardiac ganglia have equal claims with the vagi to be called "inhibitory nerves." In fact this expression seems to me altogether objectionable, since there is good reason to think that the same fibres which check the movements, much more commonly enhance them. The only evidence afforded by my experiments that the inhibiting influence is ever exerted in the natural actions of the animal, consisted in the quiescence of the intestines sometimes seen after a struggle, and two doubtful observations of retardation of the heart's beats from the same cause. Indeed it appears very questionable whether the motions of either of these viscera are, under ordinary circumstances, ever checked by the spinal system, except for very brief periods; whereas the increased action of both heart and intestines, familiarly known to result from mental emotion, may last for a very considerable time. The fact that the nerves of these organs are capable of setting them at rest under conditions of extraordinary irritation is nevertheless a matter of great importance, especially in a pathological point of view, and appears to afford an explanation of facts in medicine hitherto little understood, —such as failure of the heart's action from violent emotion or pain, and the constipation which attends strangulated omental hernia.

From the observations of Spiegelberg*, it would appear that the uterine contractions are promoted by mechanical irritation of the cord, and arrested by transmitting a powerful stream of galvanism through the spine. Also the forcible expulsion of urine very frequently seen in the lower animals in consequence of fear, and the temporary palsy of the detrusor often witnessed in the human subject in surgical practice as the result of severe injury, seem to me

* Henle and Pfeufer's Zeitschrift, 3rd series, vol. ii. part. 1.

to imply that the bladder, too, while sometimes stimulated through the cerebro-spinal axis, is paralysed by its very powerful operation. Hence it seems probable that the movements of all the hollow viscera are liable to similar influence from the spinal system. At the same time it appears to be a mistake to regard this influence in the light of a strict control; for the experiments related in this letter show pretty distinctly that the contractions of the heart and the peristaltic action of the intestines are regulated, under ordinary circumstances, by the independent operation of the intrinsic ganglia.

Professor Schiff has, I understand, observed increase of the heart's action to result from very gentle stimulation of the vagus*, and has come to the conclusion, as stated by Spiegelberg in his paper before referred to, that the inhibiting influence depends upon nervous exhaustion. There are some circumstances which make me entertain great doubt as to the correctness of this view. In the first place, the very rapid recovery of the cardiac or intestinal actions when the inhibiting galvanic currents are discontinued, contrasts strongly with the length of time that the impairment of function resulting from a protracted experiment, and certainly due to exhaustion, lasts both in the intrinsic cardiac nerves and in those that connect them with the spinal system. Secondly, although very powerful galvanism not only arrests for the time, but permanently impairs the action of the heart, no such effect is observed to follow the inhibiting influence when it is caused by milder stimulation; indeed, according to my experience, less injurious effects are produced upon the heart by a protracted series of experiments of the latter kind than by a corresponding set with the currents still more feeble, that increase, while acting, the frequency of the contractions. But if the diminished rate of the pulsations were caused by a partial exhaustion of the cardiac ganglia, an opposite result might have been anticipated.

Again, there can be little doubt that dilatation of the blood vessels, in consequence of a stimulus, is due to an effect produced upon the nervous centres for the arteries, similar to that experienced by the visceral ganglia when subject to the inhibiting influence. Now an inflammatory blush of long continuance may subside rapidly when

* Henle and Meissner's Bericht, 1857.

the source of irritation is withdrawn. Thus I have seen redness which had existed for about three days in the human skin in consequence of tight stitches connecting the lips of a wound, give place at once to pallor on their removal. Had the arterial dilatation in this case been the result of nervous exhaustion continued during so long a period, such speedy recovery could hardly, one would think, have taken place.

These and other considerations, to which the already excessive length of this letter forbids me to allude, induce me to think it safest in the present state of science to regard as a fundamental truth not yet explained, that one and the same afferent nerve may, according as it is operating mildly or energetically, either exalt or depress the functions of the nervous centre on which it acts. It is, I believe, upon this that all inhibitory influence depends, and I suspect that the principle will be found to admit of a very general application in physiology.

I am, &c.,

JOSEPH LISTER.

From the Proceedings of the Royal Society of London. Vol. IX. N° 32.

N. Parkes M.D.
with Dr Ms compts

ON

GASTRO-COLIC FISTULA.

A

COLLECTION OF CASES AND OBSERVATIONS

ON ITS

PATHOLOGY, DIAGNOSIS, ETC.

BY

CHARLES MURCHISON, M.D., M.R.C.P.,

ASSISTANT PHYSICIAN TO KING'S COLLEGE HOSPITAL, LONDON, AND TO THE LONDON FEVER HOSPITAL; EX-PRESIDENT OF THE ROYAL MEDICAL SOCIETY OF EDINBURGH; AND FORMERLY PHYSICIAN TO THE BRITISH LEGATION AT TURIN, AND PROFESSOR OF CHEMISTRY IN THE MEDICAL COLLEGE OF CALCUTTA.

REPRINTED FROM

THE EDINBURGH MEDICAL JOURNAL, FOR JULY AND AUGUST 1857.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

ON

GASTRO-COLIC FISTULA.

A.—GENERAL OBSERVATIONS ON FISTULA OF THE DIGESTIVE CANAL.

THE subject of intestinal fistulæ, excepting those fistulæ which occur in the neighbourhood of the anal orifice, is one, which hitherto has not received the attention which it deserves. Yet there is no part of the digestive canal in which fistulous communications may not be established, either with the external surface, with another part of the same canal, or with some other naturally existing cavity. These fistulæ have been divided by Cruveilhier into two classes, supra and sub-diaphragmatic. To the former class, or those occurring in the portion of the digestive canal above the diaphragm, belong the following varieties:—

1. *Bucco-nasal:* between the mouth and nasal passages.
2. *Bucco-maxillary:* between mouth and antrum of Highmore.
3. *Salivary:* between salivary ducts and external surface.
4. *Between Mouth, Pharynx, or Œsophagus, and External Surface,* an occasional result of cut throat, etc.
5. *Between Pharynx and Larynx,* as sometimes results from phthisis laryngea. (Cruveilhier.) Two cases of this variety, which occurred in the practice of Dupuytren, are also recorded in the *Gaz. des Hôpitaux,* tom. v., pp. 249 and 309.
6. *Between Œsophagus and Trachea,* cases of which may be found by the following references:—

Dublin Medical Press, vol. iv. Dr R. Paterson, in *Edin. Med. and Surg. Journal,* Jan. 1849, and *Arch. Gén. de Méd.,* Ser. 4,

tom. xix., p. 214. Dr H. Green, *Treatise on Diseases of Air Passages*. Viglas, in *Prag. Vierteljahrschrift*, vol. li., Sup. p. 30. Cruveilhier, *Traité d'Anat. Path.*, ii., 537–8. Several cases in Guy's Hosp. Museum, see Dr Habershon, *Guy's Hosp. Rep.*, 3d Ser., ii., 215 and 221. Dr Wilks, *Tr. Path. Soc. Lond.*, vi., 179. Dr Ogle, *Tr. Path. Soc.*, vii., 52.

The fistulæ in these cases have resulted from simple or cancerous ulceration, but in Dr Ogle's case the opening was congenital. In most, the disease appears to have commenced in the œsophagus, where the opening has been much larger than in the trachea; but in one (Guy's Hosp. Museum, Prep. 1711[87]) it evidently commenced in the trachea.

7. *Between the Œsophagus and Bronchi;* of which the following are references to cases:—

Dr Sym, *Edin. Med. and Surg. Journal*, 1835. Two cases by Dr Berton in *Dublin Med. Journal*, vol. vii., p. 127. Dr Winn, *Med. Times*, Old Ser., vol. xiii., p. 386. Cruveilhier, *Traité d'Anat. Path.*, ii., 538. Salter, in *Lancet*, 1853, ii., 411. Habershon, *Guy's Hosp. Rep.*, 3d Ser., ii., 230.

In the majority of these cases, the communication has taken place between the œsophagus and *left* bronchus, a circumstance which is fully explained by the anatomical relations of the parts—the more longitudinal course of the left bronchus, and the situation of the œsophagus rather to the left of the mesial line. In one case only, have I found it stated that the opening was into the right bronchus; and here it resulted from the softening of a large scrofulous gland, lying between and connecting the two.

8. *Between Œsophagus and Pleura.*—A case in the Museum of St Bartholomew's Hospital (Ser. xxiv., No. 14) shows an abscess of the neck, which burst into the right pleura and œsophagus, producing a pleuro-œsophageal fistula and pneumo-thorax.

9. *Between Œsophagus and Aorta.*—This is a not unfrequent termination of aneurism of the aorta. A case also has been recorded by Mr Flower, in which a similar result followed a perforating ulcer of the œsophagus (*Dublin Med. Press*, xxx., p. 17, and *Assoc. Med. Journal*, i., 722).

10. *Between Œsophagus and Pericardium.*—Three such cases have been recorded:—

Dr Sym, *Edin. Med. and Surg. Journal*, 1835; Mr Trotter, *Tr. Path. Soc. Lond.*, 1847 (prep. of this case in Museum of St Mary's Hosp., C. a. 2); and Dr Parkes, *Tr. Path. Soc.*, ii., 40.

Fistulæ of the digestive passages, below the diaphragm, may be divided into Gastric, Cystic, Intestinal, Intestino-urinary, and Intestino-genital.

I. *Gastric-Fistulæ.*—Of these there are the following varieties:—

1. *Gastro-Colic Fistulæ*, or communications between the stomach and colon, the subject of the present memoir.

2. *Gastro-Duodenal*, between the stomach and duodenum, as in:

Two cases by Cruveilhier, *Traité d'Anat. Path.*, ii., 540, and Livr. xxvii., pl. 1, fig. 2. Dittrich, *Prag. Vierteljahrschrift*, xiii., p. 125. Barlow's *Man. of Pract. Med.*, p. 422. Barclay, *Med. Times and Gaz.*, 1850, i., 439.

There are only five cases which I have found in the same search, which has yielded thirty-three cases of gastro-colic fistulæ. Hence, I cannot subscribe to the statement of a recent writer, M. Houel,[1] that the former are more common than the latter. Two of these cases resulted from cancer, and three from simple ulceration of the stomach. In the last case, the opening was into the upper portion of the duodenum, although Cruveilhier and Houel maintain that it is always in the third part.

3. *Between Stomach and Jejunum or Ileum.*

Dr Brinton, *Brit. and For. Med. Chir. Review*, xix., 479.

4. *Between the Stomach and Pleural Cavities or Lungs.*—Two cases have been recorded of cancer of the stomach, in which an opening into the left pleural cavity resulted.

Case of Heyfelder's, quoted by Diruf, in *Zeitschrift für die Gesammt. Med.*, vol. xlii., p. 474; and Dr Hensley, in *Lancet*, 1847, i., 674.

There are also two cases in which the stomach communicated with an abscess in the left lung.

Dr Hughes, *Tr. Path. Soc. Lond.*, ii., 251. Dr Habershon, *Guy's Hosp. Rep.*, 3d Ser., i., 111.

5. *Between the Stomach and Pericardium.*—A case of simple ulcer of the stomach opening into the pericardium, is preserved in the Museum of King's College, London. (Digestive Syst., No. 37.) For another case of communication between the stomach and pericardium, see Graves's *Clin. Lec.*, ii., 237.

6. *Between Stomach and Portal Vein.*—A case of Broussais's is quoted by Diruf, in which a cancer of the stomach ulcerated into the portal vein.

Zeitschrift für die Gesammt. Med., xlii., 474.

7. *Between Stomach and External Surface of Abdomen.*—Numerous cases of this interesting fistula have been recorded.

Several cases in the *Éphémerides des Cur. de la Nature*, 1754. Three cases by Lieutaud, in *Hist. Anat. Med.*, 1767, ii., 327. Eight other cases by Gérard, *Des Perf. Spont. de l'Estomac*, 1803, p. 69. Dr Stokes, *Lancet*, Jan. 28, 1832. Lefèvre, *Journ. Comp. du Dict. des Sc. Méd.* Cook, *Dublin Med. Journal*, xiv., 271. Beaumont's *Case of Alexis St Martin.* Diruf, *Zeitschift für die Gesammt. Méd.*, xlii., 474. Robertson, *Edin. Monthly Journal of Med. Science*, 1851, i., 1. Schmidt's *Jahrbücher*, vol. lxii., p. 16. Balluff, *Gaz. Méd. de Paris*, 1855, p. 281.

[1] *Manuel d'Anat. Path.*, p. 495.

A remarkable instance of this fistula is also at present in the Aberdeen Royal Infirmary, under the care of my friend Dr Keith. The opening is large enough to admit three or four fingers with ease.

8. *Between Stomach and Peritoneal Cavity.*—Perforation of the stomach into the sac of the peritoneum may result from simple or cancerous ulceration. It is generally followed by speedy death, but a case is on record by Dr Hughes, in which recovery seems to have taken place. (*Guy's Hosp. Rep.*, 2d Ser., iv., 332.)

II. *Cystic.*—Of fistulæ of the gall-bladder there are five varieties:—

1. *Cystico-gastric*, or a communication between the gall-bladder and stomach. This is the rarest variety. An instance of it is mentioned by Cruveilhier (*Op. Cit.*, ii. 541), in which the opening was closed up by a gall-stone; and another is recorded by Chardel (*Monogr. des Dégéner. Skirr. de l'Estom.*, 1808, p. 190), of a cancerous gall-bladder opening into the stomach. Cases also are alluded to by Cruveilhier, of gall-stones being vomited, as a consequence of this lesion.

2. *Cystico-duodenal.*—This is the most common of the five varieties. The gall-bladder contracts adhesions to the duodenum, and ulceration spreads from the interior of the former into the latter. This ulceration generally, or according to some always,[1] is dependent on the pressure of gall-stones. In King's College Museum is a series of preparations, illustrating the early stages of this ulceration.—Dig. Syst., Nos. 257, 258, 259 bis, 264, and 270. The opening into the duodenum is often of considerable size, so as to allow the passage into the bowel of a large gall-stone. In two cases, exhibited to the Pathological Society of London (Mr Pye Smith, *Tr.*, vol. ii., p. 163, and Dr Vander Byl, vol. viii.), the gall-stone thus passed was so large, as afterwards to produce a complete obstruction of the small intestine. A similar preparation is in the Museum of the Royal College of Surgeons.—Pathology, 1182. Preparations of cystico-duodenal fistulæ are not uncommon in our museums. Besides the instances just alluded to, may be mentioned the following:—

Mus. King's Coll., Lond., Dig. Syst., 57 and 259; St Bartholomew's Hosp., Ser. xix., No. 11; St Thomas' Hosp., No. 1412; Charing Cross Hosp., G. 3; Cat. of Mus. of Boston Soc. for Med. Improvement, No. 565; Peacock, in *Tr. Path. Soc.*, Lond., i., 168.

The occurrence of this fistula is also alluded to in the works of Portal, Bonnet, Budd, and Rokitansky; and, indeed, by most writers on the pathology of the liver.

3. *Cystico-Colic Fistulæ*, or communications between the gall-

[1] Budd on *Diseases of Liver*, p. 208.

bladder and colon, appear to be intermediate in frequency between the two first varieties. They are, probably, oftener the result of cancerous ulceration than cystico-duodenal fistulæ. At least all the cases, which I have found recorded, have resulted from cancer.

Museum of Boston Soc. for Med. Impt., 565; Cruveilhier, *Op. Cit.*, ii., 543; Murchison, *Tr. Path. Soc.*, Lond., vol. viii.

4. *Fistulæ between the Gall-Bladder and external surface of Abdomen* are not very rare; perhaps next in frequency to the cystico-duodenal. The diseased gall-bladder contracts adhesions to the abdominal parietes, which gradually become involved in the ulcerative process, until an external fistula is established, through which gall-stones may be discharged. The following are references to cases:—

Civiale (case from wound) *Archiv. Gén. de Méd.*, Ser. 1st, xxviii., 437. Duplay, *Arch. Gén. de Méd.*, 2d Ser., i., 381. Dr Fretin, in do., 5th Ser., iv., 86. Three cases in *Gaz. des Hop.*, 2d Ser., ix., 212, Prag. *Vierteljahrschrift*, xliv., Sup., p. 70. Schmidt's *Jahrbücher*, vol. lviii., 62; lxxxii., 37; lxxxiv., 46; and lxxxvi. Museum of Boston Soc., No. 566. Chelius' Surgery, transl. by South, i., 716. Two cases in *Tr. Path. Soc.*, v., 156 and 158.

Hydatids (Nélaton, *Gaz. des Hop.*, 1854, p. 298) and abscesses of liver (Chelius' *Surg.*, by South, i., 716) may open externally, and afterwards discharge bile, so as to constitute biliary fistulæ.

5. *Between the Gall-Bladder and Peritoneum.*—If the gall-bladder contracts no adhesions to the surrounding parts, ulceration of its walls may end in perforation into the peritoneum, followed by fatal peritonitis. Such cases have been recorded by Andral (*Clinique Méd.*, tom. iv., p. 500), and by Ferral (*Dub. Med. Journal*, xxiii., 170); and, as a result of Typhoid Fever (*Report of London Fever Hospital*, 1850).

III. *Intestinal Fistulæ.*

1. *Between different portions of the Intestines.*—Fistulæ may be formed between the opposed surfaces of two different coils of either the small or large intestine, or between two parts of the same coil. These may result from tubercular peritonitis. A case is quoted by Andral, in which communication was established between two coils of the ileum (*Path. Anat.*, tr. by Townsend and West, ii., 135); and another is mentioned in the *Dublin Medical Press* (xxxv., 357), of a fistula between the ascending colon and sigmoid flexure, the result of cancer. Again, fistulæ may be established between the small and large intestines, as between the duodenum and colon.

St Thomas' Hosp. Mus., P. 40. Charing Cross Hosp. Mus., G. 21. Andral's *Path. Anat.*, tr. by Townsend and West, ii., 135.

In the first two cases, the disease was cancer; in the third, in which the duodenum communicated with the colon through the gall-bladder, the nature of the disease is not stated.

2. *Between the Intestines and External Surface.*—Fistulæ of this

nature may take place at any part of the intestinal canal, as in the duodenum (Dr Streeton, *Lond. Med. Gaz.*, iii., 43); the jejunum or ileum (St Thomas' Hosp. Mus., No. 1229); and diverticula of ileum (King, *Guy's Hosp. Reports*, 1843). Again, fistulæ between the colon and external surface constitute the not unfrequent accident of artificial anus; and, between the rectum and external surface, the very common fistula in ano.

3. *Between Intestines and Peritoneal Cavity.*—Perforations of the intestines into the peritoneum may result from ulceration of various kinds. One of the most frequent forms is that which occurs in the course of typhoid fever.

IV. *Intestino-Urinary Fistulæ.*—Fistulæ may exist between the intestines and different parts of the urinary apparatus. The most frequent are those between the rectum and bladder, or urethra, the recto-vesical and recto-urethral fistulæ of surgical authors. I have not met with any recorded instance of fistula between the ureter and intestine; but in one of the cases of gastro-colic fistula (IV.), the communication took place through the pelvis of the left kidney. Lastly, we may have,—

V. *Intestino-Genital Fistulæ*, which are most common in the female. Thus recto-vaginal fistulæ are an occasional result of parturition. A rarer instance has been recorded in the *Gazette Médicale de Paris* (1837, p. 87), of a fistula between the sigmoid flexure and left Fallopian tube; and Mr Quain mentions the case of a male, in whom there was a communication between the colon and vas deferens (*Lond. Med. Times*, xxi., 295).

Although the object of this memoir is more particularly the subject of gastro-colic fistulæ, it has been thought that the references to other fistulæ of the intestinal canal above given, which were collected during the same investigation, might prove of service to those interested in the subject.

B.—CASES OF GASTRO-COLIC FISTULA.

Gastro-colic fistulæ, or communicating openings between the stomach and transverse arch of the colon, are not of very frequent occurrence, although perhaps they are more common than is generally supposed. Many writers on special pathology have made passing allusion to their occasional existence, such, for instance, as Meckel,[1] Rokitansky,[2] Cruveilhier,[3] Lebert,[4] Bock,[5] Valleix,[6] Wunderlich,[1] Foerster,[2] Brinton,[3] Barlow,[4] Reeves,[5] and Houel.[6] Isolated cases of the disease have also, from time to time, been recorded; and illustrative specimens are preserved in some of our museums. As yet, however, our knowledge concerning them is very meagre. The object of the present paper is to bring together an account of as many as possible of the cases which have been recorded, and of the preparations which exist in our museums, in order to draw some general conclusions as to the pathology and means of diagnosis of the lesion.

I may mention that the inquiry was first suggested to me, some years ago, by my friend Dr W. Gairdner of Edinburgh. To Dr Gairdner, as also to Dr Kilgour of Aberdeen, and other friends, I am indebted for several cases privately communicated to me. I have also to thank Dr Brinton for several references to cases.

No effort has been spared to make the collection of cases as complete as possible. The English, American, and Continental journals and periodicals (upwards of forty in number), have been carefully searched. All the principal works on pathology, and on diseases of the stomach, have been consulted. I have personally examined all the anatomical museums of London; as also those of Edinburgh, Glasgow, Aberdeen, and Manchester; and have got friends to examine those of Dublin, Cork, and Belfast.[7] I have also searched the catalogues of several American and Continental museums, and have lost no opportunity of appealing to the experience of my medical friends.

Although, as will afterwards appear, it seems not improbable that, in some of the cases to which the ancient writers gave the name of Lienteria, there existed a communication between the stomach and colon, the first well-authenticated instance of the lesion is recorded by Haller, who speaks of it as "one of the most dreadful cases" he had ever known.

I. HALLER. *Opuscula Pathologica*, 1755, *Observation* xxviii., p. 60; *Pathological Observations*, 1756, *Obs.* xxiii., p. 48; *copied by Lieutaud in his Historia Anatomico Medica*, vol. i., p. 36.

History. A female, died November 1744. "Upon inquiry into the disease, I was told that, about eight years before, she was seized with an illness during her lying-in, of which she never perfectly recovered; and could neither speak clearly, nor bear any solid food ever after;

[1] Meckel, *Manuel d'Anatomie Générale*, tom. iii., p. 442.
[2] Rokitansky, *Pathological Anatomy, Syd. Soc.*, vol. ii., p. 43.
[3] Cruveilhier, *Traité d'Anatomie Pathologique*, ii., 539.
[4] Lebert, *Maladies Cancéreuses*, p. 473.
[5] Bock, *Lehrbuch der Pathologischen Anatomie*, i., 645.
[6] Valleix, *Guide du Médecin Praticien*, ii., 618.

[1] Wunderlich, *Handbuch der Pathologie*, vol. iii., part 3, p. 147.
[2] Foerster, *Handbuch der Pathologischen Anatomie*, p. 36.
[3] Brinton, *Perforating Ulcer of Stomach*, p. 41.
[4] Barlow, *Manual of Practice of Medicine*, p. 395.
[5] Dr Evans Reeves, *Diseases of Stomach and Duodenum*, p. 166.
[6] Houel, *Manuel d'Anat. Pathologique*, p. 495.
[7] The preparations in the museums of this country are seven. Guy's, St Thomas', St Bartholomew's, St Mary's, and the London Hospital Museums, each contain one specimen. There is also one in the Edinburgh Royal Infirmary, another in that of Aberdeen; and a wax model of another, in that of Glasgow.

but most of her sustenance had been small beer, which supplied her just with sufficient strength to beg about the town."

Post-Mortem. "The peritoneum, stomach, duodenum, colon, gall-bladder, and liver, were all grown together in one confused mass, shooting out on all sides small white fibres, which degenerated into a thick and soft body, by which the above parts were in a manner glued together. So strong was the cohesion, that the colon could not be detached from the stomach, and there was an open passage from that intestine to the stomach, formed by an ulcer. Hence the colon was empty, and the stomach, in that part which was contiguous to the colon, was very much disfigured with scirrhous tumours and abscesses, but sound at its connection with the œsophagus."

II. Dr Abercrombie. *Pathological and Practical Researches on Diseases of the Stomach.* Edinburgh, 1828. P. 40.

History. A gentleman, aged 56. Previous good health, except slight attacks of dyspepsia. Began to feel languid, and lose flesh, with occasional pain in abdomen. Two or three weeks after this, while walking in the street, seized with vomiting. Vomited matter had odour and appearance of fæces. Suffered no inconvenience; but in another week had a similar attack, and, three or four days later, a third. The vomited matter "consisted of thin healthy fæces, which could not be distinguished from that which he had passed from his bowels the same day." These attacks returned at various intervals; he might have three or four in a day, or be free from them for a week. Never vomited food; what was vomited "always resembled what was passed from the bowels." Bowels regular. Lived in this way for three months, and died exhausted. A week before death, copious hæmatemesis.

Post-Mortem. Stomach contracted, and adherent to parietes of abdomen, and to arch of colon. At place of adhesion, a softened mass, about two inches in thickness. Occupying the whole of the great curvature of the stomach, was a mass of ulceration. The pylorus, and whole pyloric extremity, were healthy. In the centre of the ulcerated part there was a ragged, irregular opening, fully two inches in diameter, which made a free communication with the arch of the colon; and around the opening there was also some ulceration of the mucous membrane of the colon. Small intestines empty; large contained fæces.

III. St Thomas' Hospital Museum. *Old MSS. Catalogue*, No. 1175, B.

"Large abscess (scrofulous) between the stomach and transverse arch of the colon, communicating with both, and also opening through the abdominal parietes." This extract has been copied from the catalogue. The preparation itself has been lost or destroyed, and the above reference is marked "Cancelled." There is no history, except that the preparation was known to have been an old one.

IV. St Thomas' Hospital Museum. *MSS. Catalogue*, 1535, B.

History. "The patient died hectic, without any suspicion having been entertained of disease in the kidneys or intestines."

Preparation. "Extreme result of tubercular disease of left kidney. Various adhesions with the surrounding viscera exist, and a communication has been established between the cardiac extremity of the stomach and the descending colon, through the pelvis of the left kidney. There was a large abscess in the muscles of the loins of left side, which had bared the kidney, but did not communicate with pelvis."—(Catalogue.)

The opening in the stomach is large enough to admit a finger; that in the colon, a swan's quill. The pylorus is not preserved.

V. St Bartholomew's Hospital Museum. *Series* xv., No. 14.

History. None.

Preparation. "Portion of the great end of a stomach, exhibiting a large cancerous ulcer of its coats. The arch of the colon is adherent to the diseased parts, and is penetrated by the ulcer, extending through it from the stomach."—(Catalogue.)

The cancerous mass is situated in the great curvature of the stomach, occupying a space equal to size of palm of hand. Its surface is lobulated, irregular, and ulcerated. It involves the pylorus, which must have been considerably contracted. The opening into the colon is more than an inch in diameter. The surrounding mucous membrane of the colon is but slightly involved in the disease.

VI. Glasgow Hospital Museum. *Preparation* 199.

History. None.

Preparation. Wax model of a fistulous communication between the stomach and transverse colon. A large cavity exists between the two, which has probably been distended with fluid. There are two small openings in the stomach, half an inch and quarter of an inch in diameter respectively; and one in the colon, measuring two inches by three-quarters. Mucous membrane of colon covered with papular ulcerations.

VII. *Case communicated to me by* Mr Edward Newton, *of* 30, *Fitzroy Square.*

History. A gentleman, aged 34. In the beginning of 1838, he began to suffer from pain in the abdomen, and soon after a tumour was felt in the situation of the pylorus, which was pronounced to be cancer. In May 1840, he suffered from obstinate constipation, and began to vomit fecal matter. The only food which he could take was pounded meat or broth. These symptoms were relieved for a time, but recurred at intervals. In December 1840, he died exhausted.

Post-Mortem. The disease was cancer of the pyloric extremity of the stomach. An opening, large enough to admit the finger, passed from this into the arch of the colon, which was firmly adherent to the stomach. The pylorus was constricted by the disease.

VIII. Dr Levenstein *of Berlin. Schmidt's Jahrbücher*, vol. iii., Sup., p. 105. *See also Casper's Wochenschrift*, 1840, No. 50; *Gazette Médicale de Paris*, 1841, p. 89, etc.

History. A shoemaker, aged 61. Strong constitution. Previous health good. In April 1839, began to complain of a peculiar pain in left hypochondrium, entire loss of appetite, very offensive, corrupt eructations, vomiting, borborygmi, and constipation. The vomiting occurred at first only once every four or six weeks, then every week, and by September (1839), almost daily. The vomited matters were often copious, sometimes bilious, at other times like the vomit from a cancerous stomach, but always covered with a thin layer of bile. The fæces were always scanty, either thick or thin; generally of a pale colour, like those of jaundice, but never resembling those of lienteria. Slight jaundice. The painful region of the abdomen was slightly elevated, dull on percussion, and yielded, on auscultation, noises similar to the cavernous rales of a pulmonary vomica. (From this symptom, Dr L. diagnosed some internal fistula of the stomach.) The man gradually sank, and died June 1840, death being preceded by universal dropsy. Never had any febrile symptoms, till within a few days of his death.

The colon was firmly adherent to stomach. In the great curvature of the latter, was an opening into the colon, as large as the palm of the
Post-Mortem. hand. This opening was divided into two by a slip of the mucous membrane of the stomach, in such a way, that each half corresponded to one of the two ends of the divided colon, which could only communicate with one another through the stomach.[1]

IX. De Gintrac. *Journal de Médecine de Bourdeaux*, July 1842; *copied into Schmidt's Jahrbücher*, vol. xl., 1843, p. 31.

A man, æt. 57, had previously enjoyed good health. Two years before death, digestion became slow and difficult, being accompanied often by
History. fetid eructations and vomiting. These symptoms increased, and profuse diarrhœa was superadded. Pain at epigastrium, but no tumour. Remarkable emaciation; pale, yellow countenance; small, frequent pulse; dry tongue; complete loss of appetite; gradual sinking; death.

At middle of great curvature of stomach an oval opening, 1¼ inch in diameter, connected with a similar opening in corresponding part of trans-
Post-Mortem. verse colon. The margins of both these perforations were intimately connected with one another by a thick, reddish, old membrane. Pylorus normal. Mucous membrane near cardia thickened, with small rounded vegetations. In lesser curvature, a large, rounded, firm whitish tumour, presenting a scirrhous structure. The mucous membrane of the stomach was covered with a yellow fluid, which was also found in the colon.

X. De Waters *of Frederick Town, U. S. Philadelphia Medical Examiner*, April 1845; *Edin. Med. and Surg. Journal*, vol. lx., p. 237; *Edin. Monthly Journal*, v. 853; *Vierteljahrschrift für die praktische, etc.*, vol. xii., Sup. p. 53.

A man, æt. 65. In July 1843, attacked with severe dyspeptic symptoms, which persisted. On Jan. 10, 1844, had hæmatemesis, followed by
History. severe colic and melæna. Hæmatemesis recurred at intervals for some weeks, always with pain. Once vomited puriform matter; hæmatemesis ceased, but no improvement. Emaciation; lemon-yellow countenance; marasmus; and death, nineteen months from commencement of illness.

Pyloric extremity of stomach adhered to liver and arch of colon. A mass of cancer at pylorus, more than one inch thick, with an ulcerated
Post-Mortem. surface. Between pylorus and colon an aperture, about half an inch in diameter, which allowed the contents of the stomach to pass into the colon. Mesenteric glands enlarged and indurated.

XI. Mr Whitbourn. *Lond. Med. Gazette*, May 2, 1845, p. 31; *Edin. Med. and Surg. Journal*, vol. lxv., p. 238; *Vierteljahrschrift für die praktische, etc.*, vol. xii., Sup. p. 53.

A widow, æt. 64, admitted into Guy's Hospital, Nov. 6, 1844. For five years before admission, had a protrusion at the umbilicus, and
History. for one year had been troubled with constipation and occasional abdominal tenderness. Soon after admission, the tumour at the umbilicus became hard and painful, and suppurated, discharging an unhealthy purulent matter, mixed with blood. The hardness was then observed to extend towards the left hypochondrium. On Nov. 17th, she had bilious vomiting, with obstinate constipation. On the 19th, bowels relieved with pain, and passage of shreds of disorganized mucous membrane. "From the 20th to 21st, she vomited

[1] Although fetid eructations are noted, no mention is made of the odour of the vomited matter in this case; yet, from the anatomical distribution of the parts, it seems impossible that these did not contain feces.—C. M.

three pints of feculent matter of dark colour, and liquid," bowels being confined. On the 29th, bilious vomiting and much pain. On Dec. 7th, she vomited two pints of feculent matter, of greater consistence, and supposed to have come from large intestine. The feculent vomiting recurred almost daily; and on Dec. 16th and 17th, was accompanied with copious hæmatemesis; much pain, brown tongue, and pulse 120. Dec. 28.—Strength declining, but no emaciation; œdema of feet.

January 9.—Considerable emaciation.

January 17.—Vomited nearly a pint of feculent matter; is sinking; died in the afternoon.

Cancerous deposits in liver, uterus, and lungs. The parts around the umbilicus presented a scirrhous hardness, and the liver and adjoining
Post-Mortem. organs adhered at this place to abdominal parietes. In the arch of the colon, about its middle, there was a wide opening into a slough cavity, the size of an egg, with indurated walls. From this there passed "a simple, free, sinuous canal into the stomach, near the mesian line." The edges of the aperture in stomach were thickened, and of a scirrhous hardness. (No mention of condition of pylorus.) The arch of the colon to the left of the opening was contracted, and its coats thickened. Cæcum and ascending colon distended.

XII. to XVII.—Dittrich. *Vierteljahrschrift für die praktische Heilkunde*, 1848, No. 1, p. 26; *and Oppenheim's Zeitschrift für die gesammte Medicin*, vol. xlii., p. 474.

Dittrich, in a paper on Cancer of the Stomach, speaking of the communicating passages which may result from this disease, states that, out of 160 cases, in six, he had found adhesion to, and perforation of, the transverse colon. Short notes of the *post-mortem* appearances observed in these cases are given by the same author in a series of Reports on Pathological Anatomy, published in previous numbers of the *Vierteljahrschrift*. See, for example, vol. viii., Sup., p. 101; vol. x., Sup., p. 101; vol. xiv., Sup., p. 139. In none of the cases is any allusion made to the symptoms during life, and the appearances described appear only to have varied in the extent of the disease and size of the opening. In three of the cases, at least, the opening was near the pylorus; and in these three cases, the diameters of the openings were about ½, 1, and 1½ inch respectively.

XVIII.—De Oscar Dieuf. *Oppenheim's Zeitschrift für die gesammte Medicin*, 1849, vol. xlii., p. 474; *and note in Gazette Médicale de Paris*, 1850, p. 644.

A servant maid, aged 30, admitted into University Hospital at Erlangen, January 23, 1849. Health good up to a year before admission,
History. when she began to suffer from amenorrhœa, leucorrhœa, headache, nausea, vomiting, and severe pains in the stomach immediately after eating. Vomited at first watery matter, but soon everything she swallowed, except milk. For three weeks, had suffered from a feeling of constriction in swallowing both solids and liquids, or even saliva. On admission, a constant pain in epigastrium, increased by pressure. Here can be felt a firm tumour, extending from left false ribs to left lobe of liver, with muffled but still tympanitic percussion sound, passing into that of liver and spleen. Can only lie on left side; when on right, has pain in left. After eating or drinking, complains and then of food, as a bitter mass, without any abnormal colour. Tongue clean; appetite bad; great thirst; one scanty stool in four days; stools pultaceous, but normal as to colour and other properties.

Feb. 2.—Vomiting more frequent. (A thin, greenish, odourless fluid.) Stools natural.

Feb. 5.—Stools more frequent; skin yellow; voice hoarse and sonorous.

Feb. 8.—This morning, after tasting something, vomited some ounces of a thin yellow-coloured fluid, which had undoubtedly the odour of feces. The vomiting returned during the following night, and was accompanied by diarrhœa. Matters voided by stool presented same appearance as those vomited, but impossible to say if they contained undigested food, as patient took nothing but milk No pain.—Diagnosis of Gastro-colic Fistula, by Professor Canstatt.

After this, the patient became rapidly emaciated, the voice failed still more, and collapse supervened. On Feb. 10, there was a return of the feculent vomiting. This was checked by an opiate enema, and did not return. Feb. 11.—Diarrhœa. Feb. 13.—Curdled particles of milk found in stools. No pain, and but little tenderness on pressure over stomach. Feb. 14.—Died at 8 A.M.

Post-Mortem. Stomach adherent, anteriorly, to abdominal wall; below, to transverse colon; and posteriorly, to pancreas. Cavity of stomach contracted; the whole organ, from cardia to pylorus, a mass of cancerous degeneration, its walls being several lines thick, and its mucous surface covered with soft, fungoid excrescences, having all the histological characters of medullary cancer. In the middle of the great curvature, the stomach was perforated by an ulcer, with an irregular margin, coloured yellow by feces, and larger than a crown piece, which opened from the stomach into the adherent transverse colon; in this last, the opening was only about the size of a halfpenny, and was divided into two unequal parts by a remaining bridge of mucous membrane. The opening had not the appearance of recent origin. That in the stomach was overlapped for the greater part by the surrounding cancerous matter, which acted as a sort of valve.

XIX.—Dr H. Davies. *Tr. Path. Soc. Lond.*, vol. i., p. 89; *Lancet*, 1846, vol. ii., p. 536; *Preparation and Drawing in Museum of London Hospital*, A. c. 52; *Post-Mortem Records of Hospital*, vol. i., p. 90.

History. A clerk, aged 54. First symptoms were those of severe dyspepsia. In June 1844, he was seized with fecal vomiting, the feces being partially solid, and not distinguishable from those passed in the ordinary way. (No mention made of undigested food in feces.) Attacks of fecal vomiting recurred at intervals, but might be absent for months; always came on when bowels were confined. Continued in this state for 2¾ years. Never had much pain, and continued at his work till within two or three months of his death. Ultimately died exhausted.

Preparation. A limited firm adhesion between arch of colon and lower part of fundus of stomach. Colon drawn up at this part to an acute angle. A perforating ulcer, nearly the size of a shilling, passes from stomach into adherent colon. Margin of ulcer in the stomach thin, perfectly smooth, crescentic, and very intimately connected with margin of corresponding ulcer in colon. Radiated puckerings of mucous membrane of stomach around opening, but no trace of cancerous deposit. Mucous membrane of posterior wall of colon (described by Dr Davies as) protruded into the ulcerated opening so as to form a kind of valve dividing it into two portions. No stricture, nor disease of pylorus. Cicatrix of a second ulcer in fundus.

XX.—Dr Bence Jones. *St George's Hospital MSS. Post-Mortem Book*, 1851, p. 96; *MSS. Case Book*, vol. xii., p. 171. *(Preparation not preserved.)*

History. Female, aged 45, admitted into St George's Hospital, April 16, 1851. Health good previous to October 1850. Began then to suffer from pain in abdomen, vomiting, great constipation, and œdema of feet. Two months before admission, a hard tumour had been discovered in the left side of abdomen; vomited everything she swallowed, and for

three weeks was supported by beef-tea injections. On admission, anæmic, much emaciated, low, and weak. Œdema of legs; general pain over abdomen, increased by pressure. Tumour felt to left of, and above, umbilicus. Vomiting ceased; motions dark and offensive; quick pulse. Continued to get weaker; and on May 4th sickness returned five or six times, bowels being confined.

May 5.—Vomiting more urgent; can retain nothing on stomach. "The matter vomited has a most offensive odour, very like that of gangrene."

May 6.—Died at 2 P.M.

Post-Mortem. Liver firmly adherent to stomach, but healthy. "The transverse colon was adherent to the anterior surface of stomach, which was occupied, as to its pyloric extremity, and also to some extent at both the anterior and posterior surfaces, by a mass of scirrhus, presenting on dividing the pylorus, extensive ulceration. This had moreover ulcerated forwards into the posterior surface of the colon by a narrow elongated opening, as it lay in contact, and firmly consolidated, with stomach. Great vascularity and ulceration of glands in the colon near the opening."

XXI. Mr Jones *of Caernarvon*. *Trans. Med. and Chir. Society of London*, xxxv., p. 35; *Dublin Med. Press*, 1851, p. 421; *Lancet*, 1851, ii., 586; *Med. Times and Gaz.*, xxiv., 651; *Archiv. Gén. de Méd.*, 5th Ser., i., 365.

History. A boy, aged five, first felt unwell December 10, 1850. Had erythema nodosum on arms and legs, languor, debility, and loss of appetite. These symptoms continued for more than a month, and he lost flesh. On December 31st, he was seized with violent pain in the belly, and vomiting of a dark-brown fluid, like pus from an ill-conditioned abscess. This vomiting lasted for four days, and recurred at intervals up to March 10th, with frequent attacks of pain and progressive emaciation. On January 3d, the vomiting was noted as very fetid; and Mr Jones felt no doubt that the vomited matters were stercoraceous in February, although the odour was not so decidedly fecal as it afterwards became. On March 10th, diarrhœa and loud borborygmi came on, with entire relief to the pain and sickness. Had fifteen stools a-day for five days, and then six for several days more. Improved rapidly; slept well; appetite voracious, but did not gain flesh. Continued to improve for two months. About middle of May appetite again failed, and bowels became torpid.

At beginning of June, after a dose of a mild laxative, vomiting returned. Vomited matters stercoraceous, light yellow, and semi-liquid.

All June and July, continued in the same state. Vomited every second day. Always felt better after vomiting; from being distressed and languid, became cheerful, and called for food. Aperients always caused vomiting, and did not operate on bowels.

At end of July, worse. Vomiting more frequent; bowels more costive; but no pain nor borborygmi since diarrhœa in March.

Gradually sank, and died August 2d.

Post-Mortem. Extreme emaciation. Some turbid serum in abdomen. A perforation, the size of a pin's head, in middle of transverse colon. Stomach small. An adhesion between it and the transverse colon, and an opening allowing the passage of the finger from the one to the other. Small intestines contracted (three feet) and empty. Colon dilated to twice its natural size, forming several coils, and appearing to occupy whole abdomen; filled with a thick yellow liquid, not distinguishable from that vomited. Mucous membrane everywhere healthy.

XXII. Cruveilhier. *Traité d'Anatomie Pathologique Générale*, tom. ii., p. 539, 1852.

History. None.

Post Mortem. Cancer of stomach. The cancer occupied the neighbourhood of the pyloric extremity, and opened into the colon at the angle of union of the ascending and transverse colon. The stomach also adhered to the liver and gall-badder, and a great part of its wall was destroyed at the part where it adhered to the former.

XXIII. Dr Duchek. *Schmidt's Jahrbücher der gesammten Medicin*, 1853, ii., p. 24.

Dr Duchek, in his review of cases in Dr Hamernjk's practice at the Hospital of Prague, observes that, out of twelve cases of cancer of the stomach, "bloody diarrhœa happened once, with perforation into the colon."

There is nothing more said as to the case.

XXIV. Dr Willigk. *Vierteljahrschrift für die praktische Heilkunde*, 1853, xxxviii., p. 24.

Dr W., in some observations on cases of perforating ulcer of the stomach, states that, among the interesting cases of this lesion, he had found one where the ulcer had pierced the walls of the colon, which were closely adherent to the great curvature of the stomach, establishing a communication between the stomach and colon. The two openings, the edges of which were closely cicatrized, were more than an inch in diameter.

No history of the case is given.

XXV. Dr W. T. Gairdner. *Case communicated to me by Dr G. Preparation in Dr Haldane's Collection. Post-Mortem Book of Royal Infirmary*, vol. xvii., No. 38. *References to Case in Edin. Med. Journal*, June 1855, p. 80; *Med. Times and Gazette*, xxxii., 19; *Dublin Hosp. Gaz.*, Aug. 1855, p. 208.

History. A man, æt. 40, admitted into Edinburgh Royal Infirmary, April 1855. Discharged from army, some time before, on account of "stomach complaints."

On admission, in great suffering; tenderness of epigastrium; moaning; with knees drawn up to abdomen; features collapsed, and expressive of pain; cold extremities; almost imperceptible pulse; extreme emaciation; apparently moribund. Stated, that he had had similar attacks repeatedly; that he had long been subject to uneasy feelings in upper part of abdomen, aggravated by motion, and also after eating, which was often followed by vomiting. Had for long been losing flesh; and, latterly, his appetite had failed.

Turpentine stupes were applied to abdomen; and next day, having quite recovered from collapse, following report was taken:—Abdomen moderately swollen; tympanitic. No trace of fluid effusion. Tenderness at epigastrium not extreme. No pulsation, nor appreciable tumour. Veins of abdomen not enlarged. Tongue large, flabby, moist, and clean. Bowels confined.

In a few days, under treatment (milk diet, enemata, and opium), he improved somewhat. Could retain a small portion of food. Once or twice vomited a quantity of half-digested matter, highly acid, smelling like yeast, and containing sarcinæ.

At the end of a fortnight, had plainly less suffering, and the paroxysms of pain (which was seldom entirely absent) were less severe. Could retain a small quantity of animal food best; but even this was always followed by a feeling of repletion, and occasionally by vomiting at very uncertain intervals. Once or twice some doubtful streaks of blood in vomited matter. Stools very scanty and rare; described (to Dr G.) as normal.

The weakness and emaciation, however, increased from day to day; the appetite entirely failed; and although supported by beef-tea enemata, he gradually sank, apparently from pure inanition, and died on May 12th.

Post-Mortem. Abdomen collapsed. No prominence in epigastrium. Stomach hidden under liver, a fold of colon occupying the epigastrium. On raising colon from stomach, finger broke through omentum into a cavity situated in the lesser sac of the peritoneum, bounded by the pancreas behind, and by the stomach and colon in front, and communicating with both of these last organs. Stomach small, and slightly thickened towards pylorus. On opening it, an ulcer an inch and a half in diameter, nearly circular, perforating all coats of organ, on posterior wall, half an inch from pylorus. Its edges only slightly thickened, with no morbid deposit like cancer. Two much smaller ulcers in its neighbourhood, apparently cicatrizing. Pylorus normal. In middle and back part of transverse colon, opposite the head of pancreas, a circular perforation, more than an inch in diameter. The ulcerated walls of both stomach and colon rested on head of pancreas. The passage from stomach to colon was oblique and somewhat funnel-shaped. Colon contained a quantity of greyish pulp, evidently food half-digested.

XXVI. Dr Kilgour *of Aberdeen. Case communicated to me by Dr K. Preparation in Museum of Aberdeen Infirmary*, F. b. 2. *Case-Book, St Luke's Ward*, vol. xii., p. 168.

History. A man, æt. 43, admitted into Aberdeen Infirmary May 23, 1851. Health good up to January last. Began then to suffer from dragging pains at epigastrium, which became attended with a loathing of food, and, by the end of the month, with sickness after meals.

On admission, much emaciated. Pain and tenderness in region of stomach increased after food, which is generally rejected one, two, or three hours after ingestion. Matters vomited, sour, slimy, of consistence of thick gruel, very acid, and sometimes of a greenish or brown colour. Abdomen very flat; no bulging; but in left hypochondrium, near its junction with epigastric and umbilical regions, can be felt a tumour, two inches in diameter, moveable, dull on percussion, not painful. Appetite good. Bowels very costive.

On June 14th he became suddenly worse, complaining of great pain in the belly, which continued without intermission through the night.

June 15.—In a state of collapse. Says he is sure he is dying. Abdomen distended, and very tender. "Thinks there is something burst in his belly." Much thirst; vomiting quantities of a brownish fluid; pulse quick and feeble; skin cold; voice inaudible.

From this attack he rallied, and on 18th vomiting had ceased; he had regained voice, and was much better.

He continued, however, to waste; the vomiting returned, so that he could retain hardly anything on stomach; the appetite failed; and he gradually sank, and died on August 6th.

Post-Mortem. Body much emaciated; abdominal wall close to spine. Intestines much contracted, except the colon, from middle of arch downwards. Stomach connected by strong adhesions to transverse arch of colon. Pylorus scirrhous, very much thickened, and calibre so contracted that a probe only could be passed through it. Near pylorus, two large ulcers, one about the size of a shilling, with thickened, red, and ragged

edges; the other, involving the pylorus, had been larger, but was partially healed. An inch and a half from pylorus, at the point of adhesion between the stomach and colon, was a distinct, irregularly oval aperture of communication between them, large enough to admit the little finger, and with smooth margins, only slightly thickened.

XXVII. Dr Habershon. *Guy's Hospital Reports*, 3d Series, p. 111.

History. A female died in Guy's Hospital, August 5th, 1857. "Fecal vomiting existed for some time before death."

Post Mortem. Intestines, stomach, and liver matted together by old adhesions. In left side of abdomen an abscess of some extent, bounded in front and to outer side by ribs, internally by stomach and spleen, above by diaphragm and right lung, and below by arch of colon. This abscess communicated with chest by an opening through diaphragm, and was here bounded by lower surface of lung and thickened pleura. It also communicated by two separate openings with the greater curvature of the stomach, and by one opening with the transverse colon. Openings in stomach circular, with smooth edges, not thickened. Abscess filled with partially coagulated blood, and upper end of spleen found sloughing in its interior; stomach and colon also contained a quantity of blood. Several ulcers in transverse colon, with considerable thickening of surrounding gut. An abscess between bladder and rectum, communicating with the latter. Mucous membrane of rectum ulcerated and intensely injected. "Strumous ulceration" of uterus. A vomica in upper lobe of left lung.

XXVIII. Dr Wilks. *MSS. Post-Mortem Book of Guy's Hospital*, 1855, No. 170. *Preparation in Museum.*

History. A labouring man, æt. 67, admitted into Guy's Hospital, August 15, 1855. First marked complaint was of dyspeptic symptoms, about a year before. These increased, with occasional great pain in pit of stomach and left side.

On admission, much wasted and enfeebled. An obscure, not well defined tumour in epigastrium. Much distress and uneasiness after eating; therefore only takes small quantities of fluids. Was understood to say, that he had not had sickness before admission, and had no vomiting while in hospital.

Continued to sink, and died September 20. A week before death, had slight anasarca, and purpura spots on hands and fore-arms.

Post Mortem. A large hard tumour felt at pylorie end of stomach, unconnected with liver, but intimately adherent to pancreas behind and colon in front. On opening the stomach, this was found to be a raised, fungating, carcinomatous growth, size of palm of hand, and with surface sloughy and hanging in shreds. From end of this mass, furthest from pylorus, an opening large enough to admit a goose-quill passed into colon. Finger could be passed through pylorus, but was surrounded by disease. Two other detached growths on surface of stomach in neighbourhood of first. Similar deposits in cæcum; but no alteration of mucous membrane surrounding opening in transverse colon.

XXIX. Mr Hooper May. *London Medical Times and Gazette*, January 1856, vol. xxxiii., p. 38.

History. A shoemaker, æt. 40, admitted into Brompton Hospital, September 18, 1855. Fifteen years before, had been subject to pain in stomach and vomiting after meals, but had quite recovered. Remained well till six months before admission, when slight cough and hæmoptysis came on, along with pain and oppression after meals, and a feeling as if food would not pass down.

On admission, anæmia; losing flesh; complexion dusky; pain after food.

October 6.—"For several days has complained of severe headache, with pain in epigastrium and sickness, and during the last two, and throughout to-day, especially after meals, has several times vomited a considerable quantity of yellow matter, having the odour and appearance of rather relaxed feces." Pain after eating; borborygmi; flatulence; tenderness at epigastrium, where "a firmness" can be felt on pressure; increasing anæmia and emaciation. After taking four grains of calomel had several motions, and vomited about same quantity of feces as that passed *per anum*.

October 10.—Vomited fecal matter on evening of 8th, and not since; but has, on several occasions, complained of fecal odour of breath, and taste at times intolerable; bowels freely open. A "splashing sound" heard on percussion, especially on right side of abdomen.

October 11.—Left leg much swollen and painful, so as to prevent sleep; great thirst and craving hunger.

October 15.—Much hiccup; vomiting of feces mixed with undigested food; appetite gone; bowels freely open; motions of same characters as vomited matter.

Continued to sink, and died on October 25, three weeks from commencement of fecal vomiting. For the last week, vomited everything he swallowed. A gastro-colic fistula was diagnosed during life.

Post-Mortem. Great emaciation. Great omentum puckered, and incorporated with stomach and arch of colon, where they were in contact. On tearing it up, an enormous opening into anterior wall of stomach, near pyloric end, and into transverse colon, at once exposed. About 4 inches of the colon were thus destroyed, and an inch in centre of this space so completely, that nothing but the longitudinal muscular band remained. The opening in stomach corresponded with this, as far as it went, the two being agglutinated by adherent omentum. But aperture in stomach extended an inch and a half farther, to within an inch of pylorus, this portion being closed up by omentum. Ulcerated edges of both stomach and colon dark, very jagged and thickened, but not hard. Stomach and intestines elsewhere healthy. Some feculent matter in stomach.

XXX. Dr Barlow. *Manual of Practice of Medicine*, 1856, p. 422. *Further particulars furnished in private letter.*

History. An elderly man became a patient of Dr B.'s at the Surrey Dispensary, about 1836. Had been long suffering from apparently intractable dyspepsia, with great pain and distension, immediately after taking food. Had occasional, but not urgent, vomiting; and Dr B. now "cannot positively say that it was not fecal."

Post-Mortem. Several perforating ulcers were found in the stomach. Most of these had opened into the portions of intestine, between which and the stomach adhesion had been established before the perforation occurred. Thus, one communicated with duodenum, and two with transverse colon, whilst one opened freely into a large sac or pouch, formed by thick fibrinous lymph effused upon the surfaces of the adjacent viscera. One of the openings into colon was situated at middle of great curvature, and measured a third of an inch in diameter; the other, nearer the fundus, just admitted a probe. The opening into third portion of duodenum was situated near pylorus. No constriction of pylorus.

XXXI. Dr Joseph Bell. *Glasgow Medical Journal*, January 1857, p. 42, *and private letter from Dr Bell.*

History. A female, aged 33, admitted into Glasgow Royal Infirmary, July 17, 1856. Enjoyed good health till nine months before, when she had a child. Never recovered strength, and soon complained of lancinating pains in abdomen, which continued with occasional severe exacerbations. Five months before admission, copious hæmatemesis.

On admission, much emaciated, and of a deep sallow colour. A large, hard, flat tumour felt in epigastrium, stretching to umbilicus and left hypochondrium, very painful on pressure, and yielding on percussion a clear, somewhat tympanitic sound. Frequent vomiting. Matters vomited were sometimes only ingesta, at other times they were very offensive; and Dr B., on the authority of the house-surgeon, has "no hesitation in believing that feces were vomited." Bowels loose. Debility rapidly increased; and patient died July 31.

Post-Mortem. Peritoneum of viscera and abdominal parietes studded with nodules of cancerous matter. Stomach, left lobe of liver, and colon firmly connected by a tumour, extending backwards to the spine. On opening stomach, it was found to communicate freely, in fact, nearly in its whole length, with transverse colon. The abnormal opening presented a dark fungous-looking appearance. A portion of the tumour intervened between the walls of the two viscera. Ulceration of colon evidently of more recent date than that of stomach. Microscopic elements of cancer in tumour.

XXXII. Dr C. Evans Reeves. *Case communicated to me by Dr R. Alluded to by him in Work on Diseases of Stomach and Duodenum*, 1857, p. 166.

History A male, advanced in years. Had suffered for some time from pain in region of umbilicus, most pronounced from 7 P.M. till 2 or 3 A.M., and after eating. Seven weeks before death, began to vomit fecal matter. For first two weeks, vomiting recurred at intervals of six or seven days; the quantity brought up being scanty, dark coloured, and liquid. Later, it occurred every second or third day; and during the last nine days of life it was incessant, everything but milk and iced water being immediately rejected. Even these, if taken in any quantity, either passed off instantly by the bowels, or were vomited with great straining, particularly if pressure was made over the descending colon, or if he moved in bed. When fluids were injected into colon, if much force was used, and a large quantity thrown up, they passed into stomach and were vomited.

He became very feeble and emaciated, and gradually sank.

Post-Mortem. Stomach very small. Mucous membrane deep purple. Pylorus free. Near the fundus, on great curvature, was an irregular opening, rather larger than a shilling, with ragged edges, situated in the centre of a cancerous ulcer, and communicating with the transverse arch of colon. The two organs adhered intimately, and the opening was of the same size in both. The walls of colon, to right of opening, were affected with cancer, and the canal so narrowed, that fore-finger could scarcely be passed through it. The colon formed an acute angle at point where communication existed. Hence it passed downwards, and was considerably dilated.

XXXIII. Dr Murchison. *Trans. Path. Society of London*, vol. viii.; *Lancet*, 1857, i. 480; *Med. Times and Gazette*, 1857, i., 471; *Preparation in Museum of St Mary's Hospital*, C. *b.* 11.

History. A coachman, aged sixty-three, admitted into St. Mary's Hospital under Mr Ure, November 11, 1853. Following particulars extracted from case book of Hospital:—Eighteen months before, whilst mounting a carriage, struck left side against the step. Six months after, abscess formed just below left costal cartilages; he lost appetite, suffered

from indigestion, and got very thin. Abscess, increased to size of a large orange, was opened, and pure pus escaped. Experienced great relief, and returned to work. Six months after, another abscess formed in same locality. This also was opened. The fistulous openings of both abscesses continued to discharge.

On admission, a brawny swelling, twelve inches by six, over left false ribs and epigastrium. In this, two fistulous openings, exuding a thin, clear discharge. One of these in epigastrium, near middle line. Through this, a probe could be passed two or three inches into abdomen, and its point freely moved about. Appetite bad; could eat no meat. (No mention made of vomiting.) Extremely emaciated.

While patient was under observation, a very fetid odour was frequently noticed to proceed from fistulous openings, and quantities of a fetid gas would escape from them.

At beginning of December, became worse. Fistulous openings assumed an unhealthy appearance, and discharged a quantity of fetid, fecal-smelling matter. Frequent rigours; great prostration; sallow countenance; œdema of legs; weak, irregular, and intermitting pulse; dry, brown tongue; vomiting; and costive bowels. Vomited matters had a fecal odour, but did not otherwise resemble feces.

Gradually sank, and died December 11th.

Post-Mortem. Three (a third had been made in hospital) fistulous openings, with sloughy margins, in left of epigastrium. These opened into a large cavity, with indurated sloughy walls, which communicated both with the stomach and transverse colon. Anterior wall of stomach, for about one-third of its extent from pyloric extremity, destroyed by cancerous ulceration, and replaced by that portion of abdominal wall forming anterior boundary of sloughy cavity. The whole of pyloric end of stomach, not destroyed by ulceration, occupied by a mass of scirrhus, with a fungoid, ulcerated, sloughy surface. This obstructed greatly the pylorie opening. The stomach at this part firmly adherent to colon. On slitting open colon, an aperture was seen to exist, through which a crow-quill could be passed into sloughy cavity. Margins of this opening quite free from cancerous deposit. Adhesions of liver, etc.

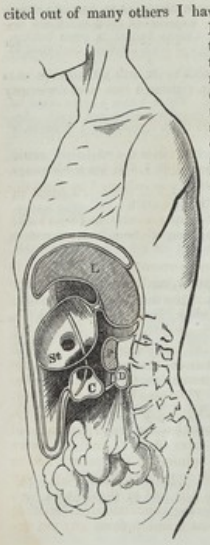
I shall now proceed to consider the conclusions, which may be legitimately drawn from the preceding cases, as to the Pathology, Diagnosis, and Prognosis of Gastro-colic Fistula.

C.—PATHOLOGY OF GASTRO-COLIC FISTULA.

I. *Mode of Origin.*—It is well known that, in ulceration, whether simple or malignant, of the stomach, or any portion of the intestinal canal, perforation, with consequent escape of contents into the peritoneal cavity, is often prevented by the diseased bowel contracting adhesions to neighbouring organs, which thus close up the deficiency in their coats. In this way, perforations of the stomach may be closed up by adherent liver, pancreas, spleen, diaphragm, abdominal parietes, colon, duodenum, etc. The comparative frequency of some of these adhesions, in the case of simple ulcer, has been pointed out by Rokitansky.[1] With regard to the colon, two such instances may be

[1] *Pathology. Syd. Soc. Transl.*, vol. ii., p. 23.

cited out of many others I have met with. In the museum of the Manchester Infirmary, is a preparation (No. 25) of a cancerous ulcer of the stomach, closed up by an adherent colon; and in Schmidt's Jahrbücher,[1] a case is recorded of a simple ulcer of the stomach closed up in a similar manner. It is seldom, however, that the original disease, by such adhesions, is arrested. The ulcerative process advances into the adherent organs, or the cancerous deposit invades them, and then sloughs out in successive patches, until at last, if these organs be hollow, fistulous communications result. The peculiarly favourable relation to one another of the stomach and colon for this result, is well shown in the annexed wood-cut, which also represents a fistulous communication between the two. Gastro-colic fistula then is, for the most part, one of the sequelæ of simple or cancerous ulceration of the stomach. Thus, out of the thirty-three cases collected in this paper, there were



21 from cancer, { 1, 5, 7, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20, 22, 23, 26, 28, 31, 32, 33.

and

9 or 10 probably from simple ulceration. { 2, 6?, 8, 19, 21, 24, 25, 27?, 29, 30.[2]

From this it would appear, that the proportion of cases resulting from cancer is more than double that from simple ulceration; and, as simple ulcer of the stomach is about five times as common an affection as cancer, Dr Brinton was not far wrong when he conjectured "that its proportion in the malignant disease is at least thrice (and probably six to ten times) as great as in the ulcer."[3] This accounts for the fact, that some pathologists, such as Rokitansky and Bock, speak of it as a result of cancer of the stomach, but make no mention of it under the head of simple ulcer. This greater rarity of gastro-colic fistula, as a sequela of simple ulcer, may depend on

[1] Vol. lxxxiv., p. 46.

[2] The numbers of the cases will be repeated in the course of the paper, in order to facilitate comparison.

[3] Article on Cancer of Stomach in *Brit. and For. Med. Chir. Rev.*, xix., 479.

three causes:—viz., the fact, that simple ulcer is much more rarely met with in that part of the stomach nearest the colon—the great curvature (in 5 only out of 220 cases);[1] and that there is a greater tendency in cancer to contract adhesions to neighbouring parts before perforation; while, at the same time, the cementing matter is of a less permanent quality than the lymph thrown out in the vicinity of a simple ulcer.

The absolute frequency of gastro-colic fistula, as a sequela of cancer of the stomach, has been considered by Dittrich and Brinton. Dittrich,[2] out of 160 cases, found this result in six, or 3·75 per cent.; but this proportion seems large, and probably Dr Brinton[3] is nearer the truth when he says, that out of 507 cases collected by him, this lesion was observed in eleven, or in 2·17 per cent.

In two of the cases included under simple ulceration (8 and 29), there was probably also extensive sloughing of the adherent coats of the two viscera.

Although I have considered the cancerous and simple ulcer in these cases as commencing in the stomach, and involving the colon secondarily, it seems not impossible that the disease may commence occasionally in the colon. Dr Habershon, in remarking upon his case (27), considered that the disease commenced in the colon; and, in some general remarks on ulceration of the stomach, observes, "A communication sometimes takes place from the colon, but this appears generally to extend from the intestine to the stomach, rather than from the latter to the former."[4] The cases here recorded, however, are quite at variance with such an opinion. Out of the thirty-three cases, there is every reason to believe that the disease commenced in the stomach in twenty-six; in four cases (3, 8, 21, and 32), there are not sufficient data for forming an opinion on the point; and in three only (6, 11, 27), does the disease appear to have been most advanced in the colon after death. In the last of these cases, also, it seems not improbable, that the disease commenced exterior to both stomach and colon, in a different manner from any yet described.

In this case, as also in case 4, I am of opinion that the lesion was produced by an abscess of the abdominal cavity, bursting into both the stomach and colon. In case 4, this abscess evidently originated in tubercular disease of the left kidney, so that the passage between the stomach and colon was through the pelvis of that organ. In case 27, the abscess also opened into the cavity of the chest; the ulceration of the mucous membrane of the colon and rectum does not appear to me any proof that the disease commenced in the colon, for we often find ulceration of the large gut in connection with abscesses of the abdominal cavity, as, for example, of the liver. Dr

[1] Brinton. *Brit. and For. Med. Chir. Rev.*, xvii., p 162.

[2] *Prag. Vierteljahrschrift*, 1848, i. 26.

[3] *Brit. and For. Med. Chir. Rev.*, xix., 479.

[4] *Guy's Hosp. Rep.*, 3d Ser., i., p. 103.

Graves[1] has recorded two cases, which are interesting as regards this mode of origin of gastro-colic fistula. These were cases of abdominal abscesses, which perforated the stomach from without. In one case, the abscess also opened into the pericardium; and there seems no reason why such an abscess should not open both into the stomach and colon. In case 33, it was a question, whether the original disease was cancer of the stomach or an abscess of the abdominal wall. The history gave some countenance to the latter view, but the *post-mortem* appearances rendered the former more probable.

Lastly, it is probable that a gastro-colic fistula may result from the softening of tubercular matter cementing together the stomach and colon, in the same way as a fistula may sometimes be formed between the œsophagus and one of the bronchi. Case 3 is described as a "scrofulous abscess" communicating with the stomach and colon. From the meagre description, however, and the fact that the preparation has been lost, the exact nature of the disease may be doubted. At the same time, there is a case on record by Dr Benecke,[2] which renders this mode of origin very probable. This was a case in which the stomach and colon were glued together by scrofulous peritonitis, and in which the stomach, at the site of adhesion, was perforated, evidently from without, by the softening of tubercular matter. It only required a repetition of the same process, in the direction of the colon, to have completed a gastro-colic fistula. As regards the mode of origin of the fistula, the thirty-three cases then may be classified as follows:—

Cancer commencing in stomach,		20 cases.
Do. colon,	(case 11)	1 „
Simple ulceration commencing in stomach, with sloughing,	in two cases	8 „
Do.	in colon, (6)	1 „
Abdominal abscesses originating external to both stomach and colon,		2 „
Softening of tubercular matter,		1? „
		33

2. *Closeness of Adhesion of Stomach and Colon, etc.*—In eighteen of the thirty-three cases, the stomach and colon are noted as intimately adherent; in seven cases (12 to 17, and 23) the closeness of adhesion is doubtful; but in eight cases (3, 4, 6, 11, 25, 27, 31, 33), there is mention made of a cavity of greater or less extent existing between the two. In three of these eight cases (11, 31, and 33), the cavity appears to have been hollowed out of a cancerous mass, and this cancerous mass may possibly have originated external to both stomach and colon,—for instance, in the retroperitoneal glands, as was thought to have been the case in 31. In case 3, the intervening cavity is described as a scrofulous abscess. In 25, the original disease was simple ulcer of the stomach; this opened into the lesser sac of the peritoneum, producing a limited abscess, which ultimately burst into the colon; in a similar manner, ulceration of the colon probably gave rise to the formation of the cavity in case 6. In 4, the cavity was the pelvis of the kidney; and in 27, an abdominal abscess.

[1] *Clinical Lectures*, vol. ii., p. 232.

[2] *Lancet*, 1849, ii. 664.

3. *External Fistulæ.*—In three of the cases, in which a cavity was observed between the stomach and colon, there also passed from this cavity a fistulous opening through the abdominal parietes (3, 11, 33). In 33, this seems to have existed before the opening into the colon.[1]

4. *Situation of the Opening in Stomach.*—An analysis of the cases gives the following results on this point:—

In 4 cases,	opening at or near fundus	(4, 5, 19, 32).
„ 10	„ great curvature	(2, 6, 8, 9, 11, 18, 27, 29, 30, 31).
„ 11	„ at or near pylorus	(7, 10, 12, 13, 14, 20, 22, 25, 26, 28, 33).
„ 8	„ doubtful	(1, 3, 15, 16, 17, 21, 23, 24).
33		

Thus, out of twenty-five cases in which the situation of the opening was noted, in eleven it was at or near the pylorus; in ten, in the great curvature; and in four, at or near the fundus. In two of the cases, in which the opening was situated in the great curvature (29 and 30), there was also ulceration in the neighbourhood of the pylorus; so that the cases in which the pylorus may be said to have been free from disease, were twelve in number (2, 4, 5, 6, 8, 9, 11, 18, 19, 27, 31, 32).

5. *The sizes of the openings* in the stomach, in the different cases, may be classified approximately as follows:—

In 2 cases	diameter less than	½ an inch (28, 30).
3	about	½ an inch (6, 10, 14).
6	„	1 inch (4, 7, 12, 19, 20?, 21).
6	„	1 to 1½ inch (5, 9, 13, 24, 25, 32).
2	„	2 inches (2, 18).
5	„	several inches (8, 29, 31, 32, 33).
9	„	doubtful (1, 3, 11, 15, 16, 17, 22, 23, 27).
33		

In case 33, although the opening in the stomach was large, that in the colon only just admitted a crow quill.

In three cases (8, 18, 19) the opening was divided into two by a slip of mucous membrane; and in case 8, in such a way, that each segment of the colon corresponded to one of the divisions, so that the only communication between the two segments of the colon was through the stomach.

6. *Size of Stomach and Intestines.*—In four cases (18, 21, 25,

[1] See remarks on this case in *Trans. Path. Soc.*, vol. viii., by author.

32), the stomach is noted as small and contracted; and in two (2, 26), the small intestines, as contracted or empty, as also the ascending colon in 26; while, at the same time, in four of these cases (2, 21, 26, 32) the descending colon is described as dilated, or containing feces. In case 11, the cæcum and ascending colon were much distended, but here there was a stricture in the arch, immediately to the left of the fistula.

7. *Contents of Stomach and Intestines.*—In two cases (9 and 29), fecal matter is noted as having been found in the stomach after death; and in two others (21 and 25), undigested food was found in the colon.

D.—SYMPTOMS AND DIAGNOSIS OF GASTRO-COLIC FISTULA.

Some of the symptoms about to be detailed have only been observed in a few, or in individual cases. It is to be recollected, however, that the observers of these cases have had no previous experience of the affection they were treating, nor any knowledge of the symptoms they might expect. In all probability, several of these symptoms will yet be found to be of more general occurrence. Some of them are so marked, that, even with the hitherto obscure knowledge of the subject, in two cases, at least, the existence of the lesion has been determined during life (18 and 29). In reference to the diagnosis of gastro-colic fistula, it will be convenient to consider the symptoms according to three different stages of the affection, viz., the previous disease, the period of formation of the fistula, and the fistula fully established.

I. *The Symptoms of the Disease which gives rise to the Fistula.*—The great majority of cases (28 at least out of 33) have been found to result from either cancer or simple ulceration of the stomach. The symptoms of these affections (epigastric pain, vomiting, cachexia, etc.) are sufficiently characteristic, and so well known, that it would be superfluous here to consider them. I would merely desire to impress the importance, of ascertaining the previous existence of either of these complaints, in cases of suspected gastro-colic fistula. Moreover, as the fistula is more than twice as frequently a sequela of cancer as of simple ulcer, the existence of an epigastric tumour would, *cæteris paribus*, be favourable to the supposition of its existence. Out of the twenty-one cases depending on cancer,

In 10, the history is either absent or imperfect (1, 5, 12, 13, 14, 15, 16, 17, 22, 23).
In 3, a tumour was absent or not mentioned (9, 10, 32).
In 8, a distinct tumour could be felt (7, 11, 18, 20, 26, 28, 31, 33).

Thus, out of eleven cases of cancerous fistula, a tumour was detected during life at least in eight. In case 29, also, in which there was no cancer, "a firmness" was felt on pressure over the epigastrium.

II. *Symptoms at the period of formation of the Fistula.*—It is probable that sometimes the fistula may form, without any characteristic addition to the previously existing symptoms marking its advent. Thus, in case 2, the first symptom of the fistula (fecal vomiting) came on suddenly, after some weeks of dyspepsia, when the patient was walking in the street, and was attended by no other unpleasant feeling. In many of the cases, however, the formation of the fistula appears to have been accompanied by some local symptoms, or by disturbance of the general system; and, perhaps this has more frequently been the case, than has been observed. Thus, in case 11, the symptoms indicating the existence of fistula were preceded by pain in the abdomen, and the passage of shreds of disorganized mucous membrane *per anum;* in 18, by diarrhœa; in 20, by general pain over the abdomen, urgent vomiting, and quick pulse; in 21, by violent pain in the belly; in 26, by the symptoms of peritonitis, sudden violent pain in the abdomen, vomiting, collapse, etc., while, at the same time, the patient himself felt "as if something had burst in his belly." In case 29, there was pain and sickness for several days; and in 33, rigors, great prostration, vomiting, brown tongue, and irregular, intermitting pulse. Cases 11 and 26 particularly deserve attention in future observations.

III. *The Symptoms and Physical Signs after the formation of the Fistula.*—When a communicating aperture has been formed between the stomach and colon, there is no barrier to prevent the contents of the one viscus passing into the other—fecal matter from entering the stomach—crude and undigested food from passing into the colon, and so being voided *per anum*. These results are what might have been naturally expected from the new condition of the parts; and they are what we actually find. Fecal vomiting, and the presence of undigested food in the stools, are the most frequent and characteristic symptoms of gastro-colic fistula. I shall now consider each of these symptoms, as well as some others, a little in detail.

1. *Vomiting.*—This has been an almost invariable symptom in all the cases observed during life, in which there has been a free communication between the stomach and colon; and in the majority of cases, the vomited matters have contained feces. The following analysis shows the frequency of the symptom in the thirty-three cases:—

In 11 cases, fecal vomiting (2, 7, 8, 11, 18, 19, 21, 27, 29, 31, 32).
In 3 „ vomiting very fetid (9, 20, 33).[1]
In 3 „ vomiting not fecal (10, 25, 26).
In 2 „ no vomiting (4, 28).
In 2 „ history imperfect (1, 30).
In 12 „ no history (3, 5, 6, 12 to 17, 22, 23, 24).

[1] In these cases, the fetor may have been due to the presence of feces, but this is not stated; and it is to be borne in mind, that the matters vomited from a cancerous stomach are sometimes very fetid, when there is no fistula.

Thus, out of nineteen cases, in which the history was clear, fecal vomiting was observed in eleven; and fetid (perhaps fecal), in three. But, of the cases, in which there was no vomiting, *fecal* vomiting could scarcely have been expected in either; in 4, because the communicating passage between the stomach and colon was long and circuitous (viz., through the pelvis of the left kidney); and in 28, because the opening into the stomach, as examined by myself, was only large enough to admit a goose quill, and was overlapped by cancerous matter. These two cases, then, may fairly be excluded; reducing the total number to seventeen, in eleven of which there was fecal vomiting; and in three, fetid. Considering the value of this symptom in the diagnosis of gastro-colic fistula, it becomes an object of importance to determine under what circumstances it may be absent. Dr Gairdner ingeniously endeavoured to account for its absence in his case (25), by the situation of the gastric disease near the pylorus, and believed "it would be found that fecal vomiting was to be looked for chiefly in those cases in which the pyloric opening was free, and in which, therefore, the stomach was not overloaded with its own proper contents, while vomiting of food was to be expected in the opposite class of cases."[1] This opinion of Dr Gairdner's is borne out by the facts now before us. In all the three cases in which the vomiting was not fecal, the pylorus was involved in the disease, as will be at once evident by comparing the numbers of these cases (10, 25, 26) with those given under the head of situation of the gastric opening. In all these cases, the opening was perfectly free; and in 25, very large.[2] In two also of the cases, in which the vomiting is only noted as fetid (20, 33), the disease was in the pylorus. Again, it also holds good, that in all those cases in which the disease was not situated at the pylorus, fecal vomiting occurred. Thus, of the four cases in which the opening was at or near the fundus, it is noted as having been present in two (19 and 32); of the other two, in 4 its absence has already been explained; and in 5 there is no history. Of the ten cases, in which the opening was in the great curvature, fecal vomiting was observed in seven, and fetid (very possibly fecal), in one (9); of the remaining two (6 and 30), there is no history, or an imperfect one. In one case (21) of fecal vomiting, the situation of the gastric disease is doubtful. But it does not appear that the fact of the disease being in the pylorus, produces an immunity from fecal vomiting. In case 7, there was persistent fecal vomiting, with cancer of the pylorus. Although, however, the pylorus is said, from recollection, to have been constricted, it seems more probable that the cancerous ulceration had advanced so far, as not to interfere with the passage of food. From the preceding facts the following conclusions may be derived:—

[1] *Edinburgh Medical Journal*, July 1855, p. 31.

[2] Could the absence of fecal vomiting in this case have depended at all upon the obliquity of the passage from the colon to the stomach?

1. Fecal vomiting is probably present in all cases in which the opening (except this be very minute) is situated in the fundus, or great curvature of the stomach, and may also be present when the disease is in the pylorus.

2. In all cases in which only food is vomited, the opening is at or near the pylorus, so as to preclude the passage of food.

The fact, that food only should be vomited, when there is great obstructive disease of the pylorus, is what might have been expected, and admits of another explanation besides that given by Dr Gairdner. When there is great obstruction at the pylorus, all the food will pass at once into the colon, and so down to the rectum, and little or none through the pylorus into the small intestines. But if no food enter the small intestines, no feces can be formed before arriving at the opening in the arch of the colon, and none can enter the stomach. This diminished function of the small intestines accounts for the fact, that after death, in several cases, they have been observed contracted and empty.

In cases of cancer of the pylorus, vomiting might be less after the establishment of the fistula than before; the food in the former case escaping at once into the colon.

It is not to be forgotten, that fecal vomiting may originate from other causes, such as any obstruction of the bowels, as from strangulated herniæ, intussusceptions, organic stricture, etc. The history, and the other concomitant symptoms of such cases, are generally sufficiently marked, to prevent the possibility of their being mistaken for gastro-colic fistula. At the same time, it is possible to have organic disease of the stomach, accompanied by stricture of the colon, as in a case recorded by Mr Holmes, in the Edinburgh Medical and Surgical Journal;[1] and, if fecal vomiting occurred under such circumstances, the diagnosis would be difficult. There is one distinction, however, between the fecal vomiting in any of these affections and that from gastro-colic fistula, which it is important to bear in mind. In the case of obstructive disease, there is also obstinate and intractable constipation; whereas, in gastro-colic fistula, there may be diarrhœa, or, if the bowels are costive, there is no great difficulty in moving them.

2. As another consequence of feces entering the stomach, there may be a *fecal odour of the breath*, and an intolerable *taste of feces*, as in case 29; or there may be peculiarly fetid eructations, as in cases 8 and 9. In two, if not in all these cases, there was also fecal vomiting.

3. Again, in cases where a cavity exists between the stomach and colon, which also opens outwards through the abdominal parietes (3, 11? 33), *food, as well as fecal matter, may escape through the external opening*. In case 3, there is no history; and in 11, it is doubtful if the external opening passed into the cavity. Fetid gases

[1] Vol. viii., p. 151.

and fecal matter, however, were observed to exude from the external fistulæ in case 33; and, although there is no mention of food having been observed in this case, it is possible that this may have been overlooked. In connection with this point, there is an interesting preparation in the museum of Charing Cross Hospital (G. 21). This is a cancerous stricture of the arch of the colon, in which the colon above the stricture communicated with the duodenum, and also, through the abdominal parietes, with the external surface. In this case, "any fluid taken into the stomach, flowing by the duodenum into the colon, made its appearance in a few minutes at the umbilicus."

4. *Passage of Undigested Food per Anum.*—It is to be regretted, that in so few of the cases was any observation made upon this interesting point. In seven only is there any mention made of the appearance of the stools, after the probable formation of the fistula (2, 8, 18, 19, 25, 29, 32). In 18, they resembled the matters vomited, and contained curdled particles of milk (the sole article of diet); and in 32, milk and other substances, if not vomited, "passed off instantly by the bowels." In case 29, the stools had the same character as the matters vomited, which consisted of "feces mixed with undigested food;" and in 2 and 19, the stools and vomited matters are described as indistinguishable. In 25, the stools were described to Dr G. (by nurse?) as normal; yet half-digested food was found in the colon after death. In case 8 only, are the stools said to have contained no undigested food; yet they were imperfectly formed, being pale, like those of jaundice; so that milk, of which, probably, the patient's diet principally consisted, would have been difficult of detection in them.

For the same reason that fecal vomiting is rarer when the pylorus is obstructed, it may be expected that future observations will show that undigested food in the stools is then most common. The food cannot pass through the intestinal tract to form normal feces, but escapes at once, by a short cut, to the anus. The greater the obstruction at the pylorus, the more readily will the food pass at once into the colon (provided the aperture of communication be of sufficient size); and, consequently, the less probability will there be of fecal vomiting, and the greater of undigested food being observed in the stools.

Undigested food may be found in the stools under other circumstances than that of gastro-colic fistula; but these, as yet, do not appear to be very clearly understood, for medical men too often rely, for the appearance of the stools, upon the observations of the nurse or of the patients themselves. This symptom is occasionally observed in the diarrhœa of children.[1] Dr Lyons,[2] likewise, has recently observed in the Crimea, a form of diarrhœa, in which "the

[1] West, *On Diseases of Children*, p. 479.
[2] *Report on the Pathology of the Diseases of the Army in the East*, p. 8.

egesta often differed in appearance but little from the ingesta; thus solid fragmentary particles of various kinds of food, animal as well as vegetable, could be readily detected in the feces, still preserving their ordinary physical qualities." This affection was attributed to an atonic condition of the alimentary canal, and to the difficult assimilation of the "preserved meats and vegetables," on which the troops were fed. None of the cases proved fatal. The same symptom may also result from ulceration of the pylorus, producing a widening of this orifice, and destroying its action as a sphincter during digestion. Schönlein[1] describes such a case, in which pieces of fish, potatoes, etc., were found undigested in the stools. He also mentions the manner in which he succeeded, during life, in distinguishing the affection from gastro-colic fistula. He gave the patient a meal, of which the ingredients were coloured with cochineal, in order to see how soon the colouring matter would appear in the stools. This meal was followed by seven stools; and it was only in the seventh (almost twelve hours after the meal) that the colouring matter first appeared. Hence he concluded that his case was one of widening of the pylorus, and not of gastro-colic fistula.[2]

Although the causes which may give rise to the presence of undigested food in the stools have not been much studied, the observation is one of great antiquity. It is this affection which Hippocrates, and all the old authors, described under the name of *Lientoria*. Galen, in his commentary on one of the aphorisms (lib. vi., aph. 1) of Hippocrates, tersely observes: "Levitas intestinorum (vel lienteria) est velox exitus eorum quæ comeduntur atque bibuntur, quæ talia deficiuntur, qualia fuére devorata." This lienteria was supposed to depend on two causes—paralysis of the stomach and intestines, or ulceration from acridity of the humors; the former being distinguished by the absence, and the latter by the presence, of pain. It seems highly probable that gastro-colic fistula existed in some cases of the so-called lienteria.[3]

5. *Emaciation.*—This was particularly noticed in twelve out of the twenty-one cases of which there is any history; and in all, it probably existed. In most, it no doubt depended on the original disease; but in case 18, it is noted as particularly rapid after the formation of the fistula; and in case 11, as only commencing after this.

[1] Schönlein, *Klinische Vorträge*, 1842, Case 37, p. 365.
[2] Dr W. T. Gairdner writes me, that he has observed a case similar to Schönlein's. He was inclined to attribute the half-digested food in the stools, not so much to abnormal patency of the pylorus, as to destruction of the gastric glands in the pyloric region, and consequent deficiency of gastric juice. The recent observations of Corvisart, that the active principles of the gastric and pancreatic juices counteract each other when mixed together, might also afford an explanation.—(*L'Union Médicale*, t. xi. No. 50, 1857.)
[3] For numerous references to the works of the old authors on this subject, see the translation, by Dr Adams, of *Paulus Ægineta*, Syd. Soc., vol. i., p. 522.

6. Among *other symptoms of less importance*, may be mentioned the following:—The appetite, for the most part, failed entirely; but in one case (29), it was noted as craving, and accompanied by great thirst. In one case (26), the vomiting, which had previously existed, seemed to cease for a time, after symptoms which indicated the perforation. But sudden cessation of vomiting may take place in the latter stages of ordinary cases of cancer of the stomach. In case 25, the vomited matters contained sarcinæ. In five cases (2, 10, 11, 25, 31), hæmatemesis is noted as having occurred; and in case 10 this seems to have been the most prominent symptom during life, and was accompanied by severe colic and melæna. In three cases (8, 21, 29), loud rumbling noises were frequently heard in the abdomen. The bowels were generally very costive (11, 19, 20, 21, 25, 26, 33), which symptom, when neglected, always was followed by an aggravation of the fecal vomiting; in four cases, however, there was diarrhœa (9, 18, 23, 31); in 10 and 23, the stools contained blood.

An alteration or absence of the voice was observed in three cases (1, 18, 26). Some stress has been placed upon this, as a diagnostic symptom, by Diruf;[1] but, in the two last cases at least, this appeared to result merely from the state of collapse of the general system; and in 26, the voice returned with the disappearance of collapse.

In six cases (8, 11, 20, 28, 29, 33), death was preceded by general dropsy, or anasarca of the lower extremities; and in one, (28), by purpura.

In most cases, there was pain referred to the epigastrium, or some part of the abdomen; but in two cases the absence of this was remarkable. In 21, there was no pain for many months before death, although this at first had been severe; and in 19, in which the fistula existed for upwards of two years, so little inconvenience was experienced, that the patient was able to follow his ordinary employment till within three months of death.

7. *Auscultation and percussion.*—It is not probable that either of these means of diagnosis will be of much service. At the same time, from observations made in certain of the cases, they demand a passing notice. Dr Levinstein, in case 8, heard, on applying the stethoscope over the left hypochondrium, gurgling râles, similar to what are heard in a vomica of the lung; and from this he diagnosed the existence of some internal fistula of the stomach, which, from other symptoms, he concluded to be connected with an abscess of the liver. Notwithstanding the approach to accuracy of Dr L.'s diagnosis, the value as a diagnostic symptom of gurgling râles heard over the stomach, appears very questionable. In case 29, a "splashing sound" was heard on percussion of the abdomen. Again, in two cases (18, 31), a hard tumour was felt in the epigastrium, which,

[1] Oppenheim's *Zeitschrift*, vol. xlii., p. 491.

in place of being dull, yielded a tympanitic sound on percussion. In case 31, this sign attracted the particular attention of Dr Bell, who was perfectly ignorant of Diruf's previous observation. It may have been due to the excavated nature of the tumour.

8. *Vomiting of Enemata.*—A very interesting observation was made by Dr Reeves in case 32. Fluid enemata, if much force was used in their administration, passed from the colon into the stomach and were vomited. This fact suggests an important aid to our diagnosis. If in any case a *coloured* enema was rejected by vomiting, the existence of a gastro-colic fistula would be rendered almost certain. The passage of the enema into the stomach would be favoured by elevating the lower part of the body during the experiment.

From the symptoms which have just been enumerated, it must be obvious that the diagnosis of gastro-colic fistula need seldom be a matter of doubt.

1. We have, in the first place, almost invariably, the symptoms of either cancer or perforating ulcer of the stomach, for a greater or less period.

2. Then we have symptoms, more or less marked, indicating the formation of the opening.

3. When the opening is fairly established, there is very generally vomiting of feces.

4. Fecal vomiting, supervening upon the symptoms of cancer or perforating ulcer of the stomach, would render its diagnosis tolerably certain.

5. Where fecal vomiting is absent, there will be the greater probability of our finding lienteria; and this, with the other symptoms and signs enumerated, would in general leave little doubt as to the nature of the case.

6. When there is lienteria, without fecal vomiting, there will probably be found disease of, or near to, the pylorus, or a very large fistulous communication.

7. In a case with neither lienteria nor fecal vomiting (which probably will never occur, except the communicating aperture be very small), it is unlikely that the lesion would be suspected; but even then its existence might be guessed from observing a greatly increased rapidity of emaciation, and a relief from previously existing pain, combined with the results to be derived from coloured enemata and percussion.

I had lately an opportunity of seeing a patient in the Edinburgh Royal Infirmary, under the care of Dr W. T. Gairdner, whose symptoms render the existence of a gastro-colic fistula highly probable.

Case.—A man, æt. 45, admitted into the Royal Infirmary, April 25, 1857.

History before Admission. Always had good health till the end of December 1856, when he was seized with pain in the chest, violent dry cough, etc. Soon after this, abdomen began to swell, and diarrhœa came on, which continued up to admission. For a fortnight before, had pain after meals, and vomited a great deal.

Considerably emaciated. Abdomen distended and tender. A tumour, with ill-defined edges, felt in right lumbar region, stretching into epigastric and umbilical regions. This tumour appears to vary somewhat in position, but is never absent. There is always more or less, but never perfect dulness, on percussion, over it.

Observations since Admission.

About May 20th, he was first observed to pass in his stools small pieces of half-digested meat, the largest about an inch long by half an inch in thickness, along with membranous masses of vegetable matter. This symptom has since continued. Pieces of ham have been found in the stools, a few hours after being swallowed, quite red and undigested. On one occasion, indigo pills were administered, and the colouring matter was passed *per anum*, two or three hours after. Since two or three weeks after admission, he has had but seldom vomiting, and not much pain. The vomited matters were never known to have a distinctly feculent odour, but were nauseous, and of a brownish colour on one occasion.

The patient is still (July 3) under observation; and for some weeks there has been little change in his symptoms.[1]

The following pathological history would explain the symptoms in this case:—

1. Obstructive disease (cancer) of pylorus, indicated by tumour, pain, vomiting, emaciation, etc.

2. Gastro-colic fistula, by lienteria, cessation of vomiting, and diminution of pain; absence of fecal vomiting depending on obstructive disease of pylorus, as already explained.

3. It is to be borne in mind, however, that the symptom of lienteria may be fallacious, as proved by the cases of Schönlein and Dr Gairdner;[2] but the result of Schönlein's test seemed to favour the existence of fistula.

E.—PROGNOSIS IN CASES OF GASTRO-COLIC FISTULA.

Considering the fatal nature of the diseases, of which gastro-colic fistula is merely one of the sequelæ, the prognosis in all cases must be unfavourable. In addition to this, the speedy escape of nutriment, without absorption into the system, and the disgusting symptom of fecal vomiting, must increase the danger, and render life still more wretched than before. At the same time, it is satisfactory to know, that patients may live for a considerable period after the formation of the fistula. In eleven of the recorded cases, the period during which the patients lived after its probable formation, was ascertained to be as follows:—

Case 20, lived 1½ day.	Case 11, lived 2 months.
„ 13, „ 6 days.	„ 2, „ 3 „
„ 33, „ 10 days.	„ 21, „ 6 „
„ 29, „ 3 weeks.	„ 7, „ 7 „
„ 26 & 32, „ 7 weeks.	„ 19, „ 2¼ years.

[1] *July* 10.—He has since been dismissed from the infirmary. His symptoms were somewhat relieved for a fortnight before he left; the stools appearing more fecal, and there being no vomiting. Nutritious enemata have been given hitherto without much effect. W. T. G.

[2] See page 31.

In the last case, the patient went about, and was able to follow his employment as a clerk, till within three months of his death. In this case the pyloric opening was free. The prognosis will always be more unfavourable, when there are symptoms of pyloric obstruction, with great lienteria.

The fact, that the formation of the fistula may be attended by relief to previously existing pain, has already been alluded to.

In bringing these remarks to a close, I have only to express the hope, that the attention which has been drawn to the subject, will elicit new information from future observers.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

HINTS

ON

VENTILATION,

BY

JOHN LOUCH, M.R.D.S., M.S.A.,

ARCHITECT, SURVEYOR, AND VALUATOR.

PRICE ONE SHILLING.

MAIDSTONE:
PRINTED BY JAMES BRINE, 10, WEEK STREET.

1860.

This little Work formed Eleven Papers on the subject of Ventilation, and appeared in the Dublin Medical Press; they are again presented to the public, at the request of many friends of the Author, who is indebted to the following Works for the quotations contained in it:—Civil Engineers and Architects' Journal; The Builder; Tredgold on Warming and Heating; Farady on Heat; Doctor Arnott; Doctor Reid; Report of Health of Towns Commission; Chambers's Journal; &c., &c.

15, *Molesworth Street, Dublin,*
September, 1860.

FROM THE DUBLIN MEDICAL PRESS,

Of Wednesday, July 14th, 1858.

SANITARY REFORM.

"The old saying, 'that it is an ill wind which blows nobody good,' will now probably be verified by an additional example; for that which wafted the odours of the Thames into the Houses of Lords and Commons cannot fail to prove beneficial elsewhere: it has supplied a most convincing argument to the advocates of measures affecting the public health. We shall now probably have a solution of the difficulty encountered in all great towns on the banks of rivers; for in relieving aristocratic noses from offensive emanations, we may learn to protect plebeian lungs against the causes and consequences of them: we seldom have evils corrected until people in power suffer from them in their own persons. How the Thames is to be 'deodorized,' we leave to our contemporaries on its banks, although they have hitherto failed to set it on fire; it is our business to deal with the Liffey, and even it we would gladly leave to some candidate commissioner of some board of health in embryo. What we want is to avail ourselves of the opportunity to remind our readers that the preservation of health is as much their business as its restoration; that they are the persons to prevent much of the mischief which it seems great engineers cannot remedy when it becomes overwhelming; and that on them devolves the duty of discovering the causes of evils which others must remedy. Hospitals, workhouses, lunatic asylums, and jails, they are to watch over and to regulate, so that they become not the source of disease, or the seat of it when in progress; while the causes of poisonous effluvia and miasmata in town or country they have to discover, and the means of contagion to cut off. We do not say that they are now bound to do all this; we only assert, that sooner or later the thing must be done systematically and effectually, and that they are the persons who must do it: this is inevitable, for neither political amateurs nor personal pets can be entrusted with any such duty. The selfish and short-sighted ratepayer may say and think to the contrary, assuming that 'the doctor' for his sixty or seventy pounds a year must do this also, in addition to his dispensary, vaccination, bridewell, police and military duties; 'we'll stand no such nonsense,' say they, 'the rates must be reduced and taxation kept down, for all the parliament's saying,' but they will find in the sequel that no such flimsy fabric will hold water: for if workhouses and their hospitals must be supported, infirmaries maintained, dispensaries efficiently worked, and lunatic asylums paid for, so must permanent establishments be created to enforce cleanliness, ventilation, perfect drainage, and proper con-

struction of dwellings: no cheap-and-nasty expedients will answer; if done at all, it must be done effectually. All this we say to remind our brethren, so that they may be prepared for legislation on this subject; for if London be purified, so must every other city, town, and village in the three kingdoms, Ireland included; partial measures not being now in high estimation: in fact we would warn our provincial brethren to be prepared for such work, and resolved not to do it without proper payment. In the meantime they can be trying their hands by a little wholesome reformation above and beyond sewers and dunghills, and in places not generally supposed to require it—viz., in the nurseries, bedrooms, and culinary regions of their patients, where, although they may not have to encounter the perfumes provided for the Lords and Commons, they may discover many things equally injurious to health, and quite as productive of disease. Our correspondent, Mr. Lotch, has been lecturing them on ventilation, an object of the first importance amongst these to which we are alluding; and as he deals with the subject so perspicuously, we leave it in his hands; but there is something about the house, particularly in the sleeping-rooms, which ventilation, especially when defective, cannot and does not remove, and that is the volatile products of respiration and perspiration which settle on ceilings, walls, and furniture. This it is which, detected by its smell only, becomes the medium by which infectious diseases are transmitted, and when accumulated by time is probably the cause of disease, or modifications of disease, for which we cannot otherwise account, and to it must be attributed the offensive odour perceived in crowded apartments, even when the inmates and furniture are removed. Sewerage and ventilation may be as perfect as the circumstances of the case admit, and yet this source of mischief will remain; for to what other cause can we assign the communication of infectious and contagious diseases long after the apparent purification of apartments in which the sick have lain. In hospitals, workhouses, and barracks, this evil is corrected by cleansing of floors and furniture, and lime-washing of walls and ceilings. The medical attendant can adopt his sanitary precautions and enforce his rules there, but in dwelling houses this cannot be accomplished without great determination and disregard of consequences. The floors of sleeping apartments are so clothed with woollen carpets, the walls and ceilings so coated over with paper-hangings and the paint, appropriately called distemper, that purification is out of the question, while the accumulation of vast quantities of curtains and ponderous furniture sets even ventilation at defiance. People may declaim about the evils of narrow streets and polluted rivers, but we defy them to contend with the evils which bad taste and ostentation create in their own dwellings; to them they must submit without repining, and against them, even in his own house, the doctor's efforts are unavailing. As a preparation, therefore, for a movement to promote sanitary reform, do we suggest, by way of beginning, an onslaught on carpets, curtains, paper-hangings, and cumbrous articles of furniture; that successful, the rest will be found comparatively easy: engineering difficulties are easily overcome, but habits, tastes, and prejudices are not easily conquered."

CONTENTS.

	Page
Damp in Dwellings	9
Malaria...	10
Effects of Damp in Basement Floors	11
Mode of Preventing Damp from Exhalations	12
Sewers	12
Ventilation of Sewers to be avoided...	13
Humidity of the Air	14
Gas for the purposes of Light and Heat	15
Closed Stoves	15
Ancient Ecclesiastical Buildings	16
Hospitals or Rooms occupied by Sick Persons	16
Courts of Justice	16
Influence of the Atmosphere	17
Ventilation of Collieries	17
Spontaneous Ventilation	18
Flues from large Fire-place Openings	19
Return Smoke...	19
All Air of a Room must be Changed	20
General Lying-in Hospital, Lambeth	20
St. Patrick's or Swift's Hospital, Dublin	22
Consumptive Hospital, Brompton	23

	Page
York County Hospital	24
Bristol General Hospital	28
Guy's Hospital	29
Advantages of Cool Air	30
Ill Consequences of Warm Air as a means of Ventilation	31
Fire-place as a Medium of Ventilation not to be depended upon	32
Dr. Chowne's Experiments on Tubes in a Room	33
Ventilation is distinct from Heating...	35
Windows	35
Military Hospitals in Kent	35
Air Flues as connected with Chimneys to be avoided ...	37
Barrack Buildings	37
Plan for Ventilating Hospitals	38
Ventilation of Private Dwellings	39
Barrack Buildings—Recent mode of Ventilation adopted a failure	40
Palace of Westminster, London—Mode of Ventilation as proposed	40
Experiments to prove the effects of Carbonic Acid confined in a Chamber	44
The Selection of Materials to be used in Buildings ..	44
Places of Worship	45
Stables	46
Farm Buildings	47

VENTILATION.

THIS important subject is one which should occupy the thoughts of those who are solicitous about Sanitary Reform, and is of such vast interest to those employed in the erection and improvement of dwellings, that I feel a few hints arising from an experience of some years may be of use.

Damp in Dwellings.

Early in my practice it became necessary that I should consider the subject of Damp in Dwellings, and search out its causes in many localities; and, I must confess, that in many instances I have been at a loss to account for it, but by perseverance have succeeded in guarding against its effects. And, to guard against the effects of damp should be a primary consideration under the head of Ventilation. Whether we take into consideration the mansions of the aristocracy, the portions of them allotted to their domestics, the dwellings of the labouring class, or houses in *cities or towns*,—all should look to this, and to the many evils arising from damp and noxious vapours.

Damp in dwellings may proceed from many causes; such as bad and insufficient building, bad materials, want of drainage,—all of which may be remedied. But that arising from the exhalation from the earth is difficult to be overcome; and I regret to see it so

very much unattended to, even in the most costly dwellings. Hence arise all the ill effects attributable to *miasma*, and which could have been prevented by a first attention to the plain fact, that all earths will emit exhalation, even if drained to *the fullest extent*.

The handsome artificial sheet of water, or the luxuriant plantation, *close to residences*, may be fine and attractive; but while they are so, many are the sad proofs of their poisonous influence, as producing fevers, dysentery, cholera, and a multiplicity of other diseases. How can an hospital ward be well ventilated when at night the *poisonous miasma* is proceeding from its basement? Or how can the mansion be ventilated, when the decomposing vegetable and putrid matter, from the decaying leaves of trees, are spread around it; or the exhalation from the lake is passing in by every opening of the doors and windows? and all this proceeding while the inmates are at rest at night.

Malaria. Like most other poisons, malaria is capricious in its action, inscrutable in its nature, and appears to be under the influence of no uniform laws. Although it is said to be the effect of the decomposition of vegetable and animal substances, and to be always found in the neighbourhood of marshy districts, yet it is occasionally seen where no vegetable is to be detected, and no marsh exists. Notwithstanding, however, the difficulties which impede the investigation of the inquirer, a large amount of valuable information has been collected on the subject of malaria, by which some notion may be obtained of the peculiar conditions under which it is originated, and the laws which govern its action on the animal economy. This subject is not exclusively important to the professional man, but it is one in which every individual of the community feels a deep interest. It has been justly observed that if the sword has slain its thousands, malaria has slain its tens of thousands. It is disease, not the field of action, which digs the grave of armies; it is malaria by which the brave and intrepid spirit, fitted for better things, is quenched. This is the destroying angel—the real pestilence which walks at noon day—and to which all the other causes of mortality are but as feeble auxiliaries. In the work of destruction to human life, caused by malaria, we have only to compare the average duration of life in countries free from its influence, with those where it is known to exist in all its virulence. The average life in this country (Ireland) is said to be fifty: in Holland it is only twenty-five—the executioner is malaria. There are districts in France where the average is but twenty-two, twenty, and eighteen.

That the effluvia which arise from stagnant water or marshy ground are the immediate or exciting cause of disease is now universally admitted. What the nature of that effluvia is, is not known; even the presence is known only by its effects on the human constitution.

It is not my province to enlarge on all the causes or effects of malaria. I only wish to confine myself to such observations resulting from my profession as an architect; but I may be permitted to say, I have witnessed many sad instances of its effects on the human constitution, as caused by damp in dwellings.

Effects of Damp in Basement Floors. The damp arising from the exhalation of the earth, ascends up the walls of buildings to a considerable

height, and soon destroys the timbers, and saturates the materials composing ground floors, and is the chief cause of all the ill effects from miasma in dwellings, and making ground apartments so particularly unwholesome.

Mode of Preventing Damp from the exhalations.

My practice for many years has been to sink the ground composing the *basement* floor of a building to at least two feet under its level, and sinking the ground for three feet around it, removing all the *earth* taken out to a distance, filling in the entire space with clean broken stones, or clean gravel free from any loamy matter, and over this spread two or three inches of clean mortar. The footing course of masonry of all the walls of buildings *should be built dry, free from mortar;* these precautions, with proper drainage, will effectually prevent damp in ground floors, and will make them in a measure as wholesome as the upper floors of dwellings. I have known floors of places of worship, although they were six feet from the earth, become rotten from the effects of the exhalations from the earth, and had to be replaced at a great cost, *the ground of which had been well sewered.* The upper parts of buildings may be made dry by proper eave and other gutters from the roofs, and the external face of the walls cemented.

Sewers.

The sewers from all buildings should be particularly attended to, that they are made water-tight, and constructed to prevent vermin getting into them, having proper outlets, and all the sinks should *be trapped*, to prevent the escape of foul air from them into, or convenient to, the buildings. Particular care must be observed that rain-water pipes from the roofs shall not enter into the sewers at the bottom, but merely

drop into stone basons properly trapped. The various joints of those down pipes (if the pipes are let into the sewers) are so many means for the escape of the gases from the sewers, and pollutes the air which penetrates the buildings, dealing out disease of the worst consequences.

When a building is thus secured against damp and foul vapours, then methods may be resorted to to insure the free ingress and exit of air.

Ventilation of Sewers to be avoided.

In speaking of sewers, many schemes have been suggested as necessary to remove from them the various gases generated by the decomposition of putrid matter lodged and passing through them, and much controversy has ensued in consequence. Now, to overcome such a task as to try and ventilate *sewers*, would be as difficult as to remove the poison generated in its great abundance and intensity in marshy and swampy lands. All that can be expected to be done, is to prevent its escape into our dwellings, by proper sink-traps, &c. Be assured if you keep the poison confined within its limits it will be *innocious*, and why impregnate the air of cities or towns with it to ventilate sewers.

I was concerned in a case in the year 1844, where it was considered necessary to erect a mausoleum about eight miles distance from Dublin. It was the wish of the guardians of the noble head of a family that the remains of as many of his ancestors which had been buried in the churchyard, should be raised, and placed in a bottom vault of the building. About fifteen coffins, all composed of lead, were raised for this purpose. Some had lain many years in the ground, and when taken up the escape of gas from

them was so great that the surrounding air was impregnated with it; every person at the work and in the place was affected—*some severely attacked with cholera that night*. I was one of those so attacked, and under Providence I am indebted to the late Dr. Graves and to Mr. Vance that it was not fatal. Had we never interfered with the coffins, there was no other cause that could have affected us. I could multiply cases, if necessary, to prove that the escape from sewers has been fatal in the extreme during the period of the visitation by cholera.

I find the more tenacious the clay the greater will be the exhalations.

In order to insure perfect ventilation in *residences*, all causes likely to impregnate the air surrounding them and in them must be removed. *A first consideration is pure air*. Surely when it is known that the exposed surfaces of banks of lakes, ponds, rivers, and such like, will emit a poison that is of such intensity as to be capable of killing instantaneously whoever is exposed to it, care will be taken to prevent its effects; undrained meadow land is said to be a source of this great evil; mud and matter washed down by rivers to the sea shore when exposed to the sun's rays on various continents, have been the cause and grave of the European, while the interior of those countries are often considered most salubrious.

Humidity of the Air.

The humidity of the air in this country (Ireland), to which is attributed the luxuriance of its vegetable kingdom, causing so many and frequent changes in its atmosphere, even during a day, or in a few hours, presents many difficulties that will at all times appear

when it is necessary to apply the subject of ventilation to buildings.

Gas, for the purposes of light and heat.

Gas, for the purposes of light and heat, is now used to such an unlimited extent, that care should be observed to render our habitations as free from noxious vapours as possible. Professor Farady says: "That coal gas is really in many cases, when made in London, not far off in its degree of gravity from fire damp," (fire-damp when mixed with air is an explosive mixture); and he proves that gas always remains, as it were, suspended in a chamber, if an outlet is not made for its escape; and gas mixed with our breath by respiration is similar to fire-damp and air.

Dr. Arnott writes: "At this day in London, Edinburgh, and other large cities, the solecism is presented in hundreds and thousands of instances of elegant rooms for the reception of company, where all the luxuries of the palate are offered, but where you cannot get a mouthful of air that has not passed through the lungs of a neighbour, and which is not mixed with the smoke and fumes of gas or other lights, fires burning without chimneys, as they virtually are."

Closed Stoves.

In cases of death from stoves, Leblanc has found there was more or less carbonic acid in the air. He observed even one per cent. of this gas to destroy an animal in two minutes—a fact which explains many of the circumstances which appeared some years ago in the evidence of some London chemists respecting the influence of closed stoves. It is obvious that their structure was dangerous. Leblanc found that a candle was extinguished in air containing only four and a half to six per cent. of carbonic acid. In such an atmos-

phere life may be kept up for some time, but respiration is oppressive. Even three per cent. in the atmosphere killed birds, and yet we have statements which affirm that upwards of three per cent. has been detected in London theatres.

Ancient Ecclesiastical Buildings.

The ancient ecclesiastical buildings having been erected so lofty, and their windows always at a great height from the floor, the best system of ventilation was afforded; while, through a mistaken principle of acoustics, many of those erected within the last thirty years are so very low, that when crowded the air in them becomes oppressive, and the means for its removal is often found deficient.

Hospitals or Rooms occupied by Sick Persons.

Hospitals, or rooms where sick persons may be, require the greatest care to ensure the ready escape of vapours; as, for instance, it is known that a person labouring under smallpox may contaminate the air of a room to such a degree, that a healthy person breathing this air but for a short time may become affected with the disease, although the infected may never have come into actual contact with the infecting person. The free dilution of the poisons with pure air will destroy or render innoxious those malignant agents.

Courts of Justice.

Courts of justice are often, owing to excessive crowding of the buildings, rendered so hot as to become intolerable, and from the defective ventilation those present are constantly re-breathing a vitiated atmosphere, and even where attempts have been made to remove this evil, it is only by producing strong draughts or currents of air, which are even still more prejudicial to health.

It is important that in all buildings the readiest

means shall be taken for the removal of vitiated air. In a "Report of Gas Explosions in Collieries," the Commissioners write:—"Again, much depends, all other circumstances being equal, upon the kind of roof or covering rock of coal-bed,—*if this is sufficiently porous, as many sand-stones are*, the conditions for the escape of fire-damp upwards through the superincumbent rocks are more favourable than when the roof is composed of clay or agillacious shell."

Influence of the Atmosphere.

The atmosphere has an influence on the air of all apartments; it has a sensible influence in the mines on the fire damp. In a low state of the barometer gas is evolved in considerable quantity. When the barometer is high, instead of the issue of gas, there is a sensible current of air which enters into the cracks of the roof; at this state men may work with the naked candle, but when the evolution of fire damp commences, Davy's lamps are employed.

Air will press in every direction fifteen pounds on every square inch exposed to it, and this should be kept in mind, and that the tendency of the air is to assume rotatory movements, and thus induce the removal of vitiated and the supply of fresh air, whenever expansion or any other cause produces a disturbance in the atmospheric balance.

Ventilation of Collieries.

The too common use of single shafts in collieries, in cases where others might have been sunk—the single shaft divided into two or three portions by wobden partitions named brattices, a down current of pure air descending through one division, and the foul air from the colliery workings rising up through another,—has often been reprobated. It is evident the objection to this mode is that wooden partitions

are generally destroyed in the case of an explosion, and the way of escape for vitiated air interfered with. Two distinct shafts, not far distant from each other, is considered the best; there is no doubt but the fresh air will descend one of them, while the vitiated air will ascend the other. This fact I wish to be kept in mind during the reading of these papers.

Spontaneous Ventilation.

In many instances the principle is overlooked in spontaneous ventilation, that where openings are provided for the escape of impure air, others must also be provided for fresh air, *vice versa*. It must not be forgotten that air, like other fluids, can only fill a given space; or, as one of our earliest writers remarks, "that unless openings are properly adapted to suffer air to pass freely through, the external air proves a stopper to the internal, and only mixes with the next in contact." We know that the air in a well remains stagnant and pent up; but, as has been remarked, if two wells or shafts are sunk at a given distance from each other, and a horizontal passage cut from the bottom of one well to the other, so soon as the communication is made there will be a tendency in the air to descend one shaft and ascend the other, whenever the temperature of the external air varies from that below. In describing his engine, Dr. Desaqulier says:—"At every stroke eleven cubic feet of air is driven in, or as many sucked out; if the axis of the cranks turns sixty times iff a minute, one man in that time might change a whole cube space of eight feet." And by his estimate, a man breathes a gallon (about two hundred and eighty-seven cubic inches) of air, per minute, and a candle (six to the

pound) will burn nearly the same quantity. This agrees with modern calculations. At the lowest estimate, three hundred cubic inches are contaminated by a man per minute, although Tredgold and others take the quantity at eight hundred cubic inches, and a single candle alone three hundred cubic inches—ten cubic feet being considered as requisite per minute to afford a wholesome atmosphere for each person.

In the means used by various persons for the ventilation of ships, and the machines spoken of, all state the fact of drawing out vitiated equal to the quantity of pure air driven in. Such is the principle in mines, and it should be so in ventilating buildings.

Flues from Large Fire-place Openings.

It is a practice attended with the happiest results, in forming kitchen fire-places, or other large fire-openings, that two flues are brought up from them, known to the bricklayer as split flues; they empty at different points in the same chimney stack; they are generally built similar in size and finish to the other fire flues from a building. I have not known any instance where this is done that the chimneys smoked, and I have always found that one of them acted as a down while the other acted as an up draught: they commence their division a short way above the opening of the fire-place.

Return Smoke.

Few but must have felt the unpleasant effects of what is termed return smoke in apartments where the fire-place may not be occupied. If the flues are properly built, where they are all let out on the same plain at the top of the chimneys, then the tendency is, the smoke of one flue may be sucked down the next flues. This will be prevented by putting pots of equal height on all the flues of the chimney stack,

and leaving a space of a few inches between them, that the air may freely pass round each chimney pot. If flues are not properly divided, no art can cure the return smoke.

All the Air of a Room must be changed.

M. Lassaigne has shown by a series of investigations that, contrary to a common opinion, the air in a room which has served for respiration without being renewed, contains carbonic acid alike in every part, above as well as below; the difference in proportion is but slight, and where appreciable there is some reason to believe that the carbonic is in greater quantity in the upper parts of a room. These experiments establish the very important fact, that all air in a room must be changed, in order to restore its purity.

Dr. Wyman makes the following remarks on this point: "Although carbonic acid is a much heavier gas than atmospheric air, it does not from this cause fall to the floor, but is equally diffused through the room. If this gas is formed on the floor without change of temperature, this diffusion will not take place rapidly. In the celebrated *Grotto del Cane*, carbonic acid escapes from the floor and rises to a certain height, which is pretty well defined to the sight on the walls; below this line, a dog is destroyed as if in water; above it, he is not affected. An analysis of the air above and below a brazier has been made, and it was found equally contaminated, the former containing 4.65 per cent., and the latter 4.5 per cent. of carbonic acid.

General Lying-in Hospital, Lambeth.

The system adopted in ventilating the General Lying-in Hospital, Lambeth, is:—The whole roof is rendered, as far as practicable, air-proof, divided into two separate compartments; the one to contain

cold fresh air, for the supply into the various wards on each floor, and which seems to answer every desired purpose well; the other for foul air, into which all the ventilating flues discharged the effluvia rising up from the various rooms, and instead of immediately escaping into the open air, this foul air is confined in the chamber until it descends a shaft to the bottom of the building, dips under the partition, and then ascends a second shaft into the open air; it has to descend the shaft about fifty feet, and dip under the partition, and ascends seventy feet before becoming exposed to the open air.

On this subject there has been some controversy. I find the committee of the hospital *deny the efficacy of this system of ventilation altogether*, and one of the governors also writes thus:—"It is true that the system which was introduced into the hospital more than fifteen years ago, at an expense of £3,000, proved not only inefficient, but positively injurious to the health of the patients."

In no case is the importance of a proper system of ventilation more clearly seen than in many of the hospital reports which have proved such valuable contributions to medical science. Public establishments are familiarly known to the medical profession, in which, when the ventilation was imperfect, no case of compound fracture ever *recovered*; few or none survived the amputation of a limb; mortification attacked every wound, however trivial; the prostration of strength became so great that men who had at first stood the severest operation without murmur, subsequently cried like children from the slightest pain; and, indeed, cases have actually presented themselves

where the apparently lifeless corpse, subdued and oppressed far more by the atmosphere with which it was surrounded than by the disease to which it was supposed to have fallen a victim, has actually been known to revive after removal to the dead *room*—a separate apartment, where the play of a wholesome atmosphere flowing unrestrictedly upon it revived the fading flame of life, after it was to all appearance gone, and where health and strength were ultimately restored.

St. Patrick's, or Swift's Hospital, Dublin.

About the year 1821 or 1822, the St. Patrick's, or Swift's Hospital, in Dublin, had its corridors or wards heated on the principle of the hypocaust, and the large fire-places done *away with*. Looking to the construction of this hospital, it will be seen it consists of a centre, with two buildings running *parallel* from its rere, containing wards or corridors—each about 320 feet long by 14 feet wide, and of sufficient height; off these corridors are the various cells and day rooms—the former are twelve feet by eight feet six inches, and opposite to the cell windows are similar windows in corridors. The buildings are in height two floors above the ground floor. It was always considered *salubrious*, and in which the patients lived to an old age. Soon after the introduction of the hypocaust as a mode of ventilation and heating, the mortality amongst the patients appeared to be very great, and caused considerable anxiety to the *governors* and physicians in charge. It became necessary to remove it; as soon as this was done, the health of the patients was restored, and since that *period* the large fire-places (three to each corridor) have been used with iron guards to them.

(I see, on looking to the plans of most of the new asylums for insane, that the open fire-place is used as the mode of heating the corridors.) Since the period first mentioned, the windows, both in corridors and cells, have been considerably enlarged, the doors constructed to do away with as much as possible the prison appearance, and the patients receive as much liberty as their circumstances will admit of.

Consumptive Hospital, Brompton.

The ventilation of the Consumptive Hospital, Brompton, is thus described in the *Builder* for October, 1847:—"For ventilating purposes air may be either drawn in or out, or driven in or out. In the present case, as a more economical mode, the second has been adopted by Dr. Arnott. The application of steam power renders the apparatus complete. It consists of two wooden quadrangular boxes, in which two hollow pistons move up and down alternately by means of a rod attached to either end of a wooden beam, on the principle of a steam engine. The beam is moved by a steam engine of about one-horse power. When the wooden piston descends in the box, a valve opens in the upper half of the box, through which the air is admitted. When the piston ascends, the valve is closed, and the air is propelled into a central chamber. The same effect is produced in the lower half of the box; and thus in both boxes each ascent and descent of the piston impels a quantity of air into the central chamber situated between the two. From the central chamber the air rises into another apartment filled with plates of copper, heated by an adjacent fire. These are placed at a distance of half an inch from one another, and the air passing between them is thus raised to the desired temperature. The warm atmos-

phere now passes into a large tube, which ascends to the top story of the building, but in its course gives off a branch at each flat or landing. These are divided into ramifications, the narrowest of which go to the nearest apartments, and the widest to the most distant. They run along the skirtings of each room, into which they open by valvular fissures. The impure air of each room escapes by other valvular apertures, opening into the chimney as high as the ceiling. The apparatus is said to be capable of throwing into the building *three thousand cubic feet of air*, which, as the hospital contains a hundred inmates, is an allowance of twenty cubic feet per minute to every person."

York County Hospital.

York County Hospital is a large building erected in the year 1850; it has three floors above the basement. The basement is arched, and eleven feet high in the clear, consisting of seventeen different apartments, amongst which is the air chamber, and an apartment for heating and ventilating apparatus. The ground floor is fourteen feet six inches high, and the two upper floors each fifteen feet high in the clear. On the ground floor there are twenty apartments; the chamber floor has ten apartments, and the attic floor has a similar number of apartments. The principal wards are forty-two by twenty-three feet each.

In the year 1852 the committee printed a report on the warming and ventilation, out of which I beg to give the following extracts:—"The committee appointed to report on the warming and ventilation of the New County Hospital, are apprised that a similar report has been some time since requested from one of their members by the Central Board of Health; that the committee of the Consumptive Hospital at

Brompton have also been desirous of obtaining information on this subject; and, that the Select Committee on Ventilation and Lighting of the House of Commons have been referred to the York Hospital as the most complete model of the method of warming and ventilating invented by Dr. Arnott.

"On a matter of so much public interest, the committee are anxious to furnish all the facts with which they are acquainted, and at the same time to avoid all inaccurate statements and unfounded assumptions. Heretofore little accurate thought has been bestowed on this very important subject, and few buildings have been erected, even for the restoration of health, in which any intelligent attention has been paid to the means of obtaining a proper *climate* within. It is certain, however, that this is a condition for recovery quite as essential as food or medicine; and in the York Hospital it has received an attention more than is usual, but not more than is justly due to it."

"Very inartificial modes of ventilating and warming may suffice for persons in health, who are not confined for the whole twenty-four hours to the same chamber, and whose windows and doors may be frequently opened without inconvenience; but they are inadmissible in hospitals, where the unintermitting inhabitation of the principal wards, and the augmented susceptibility of their inmates to the influence of currents of air, allow no intervals for refreshing an apartment, and no latitude for irregular draughts."

"Dr. Arnott, whose plans have been adopted here, prefers a single mechanical force of propulsion, combined with the spontaneous up-draught of a shaft, as affording the most uniform and manageable means of

ventilation. The method which he employs of driving air through a building is exactly the same by which a supply of coal gas is moved through the mains and pipes of a town; and in his application of it to the York Hospital, he has carried this method to a high degree of mechanical perfection. To simplify the motive power, he has substituted the pressure of a small column of water, alternately admitted and cut off by a stop-cock, for the force of a steam-engine, elsewhere employed by him; and to render the small power which would be thus commanded available, has exhausted every artifice of contrivance to avoid friction."

"The principle of propelling the air through the chamber in which it is warmed, and thence through the building, possesses a superiority over any method of ventilating by exhaustion in this respect, that the tendency of the process is to produce a plenum rather than a vacuum, and there is consequently no danger of the regular ventilation being interfered with by an undesired in-draught through accidental apertures. The more entirely, however, the undesigned influx of air is prevented, the more necessary it is to be satisfied that the supply provided is sufficient for its purpose. Now, it is one of the chief merits of this invention, that the propelling pump, being constructed on the well-known principle of a gasometer, accurately measures its own work. The hollow cylinder of this gasometer, which acts as the piston of the pump, is six feet in diameter, and four and a half feet deep, moving up and down in a water-joint, with two sets of curtain valves, above and below, so as to inhale and exhale at each ascent and descent of the beam; it drives, therefore, at each double stroke two hundred

and fifty cubic feet of air through the building; and as it works at the rate of nine such strokes in a minute, the number of cubic feet which it delivers in that time is 2,250, or 135,000 in an hour; and if we take the number of patients in the hospital at 70, the allowance of air per head is 1,928 cubic feet in the hour; if at 80, then the allowance is 1,685 cubic feet each. When the air has entered the channels provided for it which encircle the wards, it is admitted into the rooms by a double slit in the whole length of the skirting boards, and thus diffused rises to the valves in the cornice, whence it proceeds into the vertical shaft by converging flues, which are calculated to allow of its exit in, and equal to that of its entrance."

"The air chamber is intersected by a range of thin copper cases fixed at narrow intervals, between which the air passes to be warmed, and within which the water that communicates the warmth circulates more or less quickly, as allowed by an adjusting cock. The circulation is maintained by water stoves, of which one or more may be employed as more or less warmth is required, and in which a perfect command of the combustion of fuel and circulation of water regulates the amount of heat."

I am obliged to refer for further information of this mode of ventilation to the "Plan and Section of the Apparatus for Distributing Hot and Cold Air in use at the York New County Hospital," as annexed to the report, which I beg to remind the reader was made in the year 1852. As a heating apparatus, I understand it has done its work well; but after an experience of six years as a mode of ventilating, it was necessary to discard the apparatus altogether and they are now

ventilating and warming the house by means of open fires and free ventilation, by opening the doors and windows when the weather permits. Since this has been adopted, and Dr. Arnott's engine done away with, there was no return of erysipelas, although there were some very bad cases in the house; before this alteration it was scarcely ever free from the disease, besides the atmosphere is very much purer,—an every-day remark now by persons who were accustomed to visit when the engine was going. There is no doubt but this failure is caused by the mode used for the exit of the vitiated air from the different wards; and I do say with confidence that heating air before it is conveyed into the apartments as a ventilating system, has too much of the hypocaust about it.

Bristol General Hospital.

"Two fire-places have been put in each ward of the Bristol General Hospital, so that the temperate air from the apparatus may be dispensed with when required. The hospital accommodates 170 in-patients, and the wards contain an average of 1,098 cubic feet per patient; the fresh air is taken by a shaft in the airing ground and under the basement into the warming apparatus, and thence to all parts of the building. The vitiated air is drawn out near the ceilings by means of flues connected with a large extraction shaft, 100 feet high above the level of the wharf, wherein the air is always kept highly rarified by coils of hot water pipes, and a large cistern through which nearly boiling water is circulating day and night for the supply of all the baths, lavatories, and sinks. It is supposed that with the doors and windows all closed, upwards of 120 cubic feet of air per minute is drawn out through each ventilator, 14 by 9 inches; and with

the doors or windows open, the quantity is increased upwards of 150 feet per minute. The theory is, that the extraction is so regular and powerful, that the atmosphere of the worst wards will never be foul; nor can it be communicated to the other wards by means of the corridor, there being always an in-draught, it is asserted, from the corridors to all the wards."

I have not as yet any means of stating the effect of the mode adopted in the hospital, but it is so like that of the York County Hospital, I am afraid the same results will follow. The difficulty, or, I might say, the impossibility, of getting the vitiated air to traverse all the horizontal flues before it will arrive at the up-draught shaft, is now established by the many examples on this subject.

Guy's Hospital.

A description of Guy's Hospital is thus given:—

"The centre is flanked by two square towers surmounted by octagonal turrets, which act as fresh-air shafts to supply air to the building. In the rere is a lofty square tower surmounted by an octagonal lantern and a spire of open iron-work. This tower is the extraction or ventilating shaft, and receives the vitiated air from the wards, as well as the smoke from the furnaces and fire-places. There are no chimney stacks in the building. The structure is five stories high. The space allotted to each patient in the wards averages 1,600 or 1,700 cubic feet. The whole building, with the exception of the central hall and great staircase, and some of the officers' apartments on the ground floor, is artificially warmed and ventilated. The cubic contents of the spaces so warmed and ventilated is five thousand feet. The system of ventilation adopted is, the fresh air is taken from the high level, in order to

ensure greater purity, descends by the fresh-air shafts to the basement, where it is conducted by horizontal channels, *parallel* to the main walls, to the lower openings of vertical air-flues carried up in the thickness of the walls of the different stories. Along the sides of the fresh-air channels hot-water pipes are laid, which warm the air in winter. Every flue being thus supplied with air warmed by contact with heating surfaces placed wholly in the basement, corresponding flues conduct the vitiated air from each floor to a main vitiated air-trunk in the roof, terminating in the lofty ventilating air-tower at the rere of the *central hall*, the air of which is rarified by the waste heat from the furnaces of the hot water and steam boilers. The system of ventilation thus established, is perfectly independent of the obvious means of ventilating the wards when the weather permits of the windows being opened. In the construction of the wards express reference has been had to obtaining all the advantages derivable from what is called *natural* ventilation."

Advantages of Cool Air.

I have so far laid before you the expensive and complicated methods that have been adopted in ventilating several of the large hospitals in England, and with some of them the results (which were seldom known until fatal consequences ensued) which, as I before mentioned, are to be attributed to the mode of exit for vitiated air; and much may be owing to *injecting* the several wards and apartments *with heated air*. The advantages of a cool atmosphere to breathe in is greater than most people imagine; for, according to the experiments of Crawford, Lavoisier, and others, it appears the consumption of oxygen is less by 1-12th

in an atmosphere of the temperature of 79 deg. than in one of 54 deg. The diminution is greater than would result from the mere rarefaction of the air, and is attributed to a high temperature counteracting the chemical changes which the blood undergoes in the extreme vessels. Elevated temperature also increases the quantity of insensible perspiration, and nearly in proportion to the quantity it is elevated, according to the experiments of Berger and Delaroche.

Consequences of Warm Air.

In a room warmed by heating the air, a person not actively employed does not feel a comfortable degree of warmth when the thermometer is below 62 deg., though in cold weather that temperature feels oppressive to one who has been in the open air; indeed, the transition from breathing air at 20 deg. to air 62 deg., which must often happen, is too great to be made without being felt. There must, therefore, be a sensible advantage in having to approach such a room through halls and passages kept at a low temperature, in order that the change may be gradual, and therefore less injurious. If one's clothes have acquired any degree of dampness from a moist atmosphere, a chilling coldness is felt on entering a room filled with hot air. This feeling is caused by the powerful effect of warm air in abstracting moisture. It absorbs moisture in the state of vapour, and this vapour requires a great quantity of heat for its formation in the state which chemists term *latent heat*. It is easily understood that a person in damp clothes or in a state of perspiration cannot enter into a room filled with hot air without some danger; and the warmer the air of the room is, the greater this danger will be; because the evaporation will be quicker, and the sensation of cold

more intense. The continual absorption of moisture from the human body, which is a consequence of being in a bath of warm air, produces *headache*; the eyes feel wearied and painful, and the whole frame is disordered. If you saturate the air of the *room* with moisture, these sensations do not occur, or at least in a less degree. An apartment warmed chiefly by radiant heat is in a great measure free from these disadvantages, because the air is always cooler than the objects receiving heat from the fire.

1,000 volumes of air at 32 deg. become 1,375 at 212 deg. When the air is expanded by heat, it of course becomes lighter, and rises through the atmosphere in a similar manner as water, though much more rapidly. No less than eight ounces of carbon taken into the system in food, is supposed to be consumed daily by a man for the purpose of maintaining a proper temperature in his body, by being brought into contact with the oxygen of the air he breathes; he ought, consequently, to produce carbonic acid largely in his system as he does so, throwing it off by breathing.

Fire-place, as a medium of Ventilation, not to be depended on.

And I find that the fire-place as a medium of ventilation is not alone to be depended on. An experiment made by Mr. Daniel illustates that the ventilation of a room warmed by an open fire is defective; he says, the temperature of a room being 45 deg., "I found the point of condensation in it to be 39 deg. A fire was lighted in it, the doors and windows shut, and no one allowed to enter; the thermometer rose to 55 deg., but the point of condensation remained the same. A party of eight persons afterwards occupied the room for several hours, and the fire was kept up; the temperature increased to 58 deg., and the point of

condensation rose to 52 deg. This experiment shows that, notwithstanding the constant current of air up the chimney, the quantity of vapour increased till the air in the room was nearly saturated. It would have been removed as it was formed if the ventilation had been from the upper part of the room, and with it the effluvia and impure air."

It is of necessity that I am obliged to give extracts from different authors in my various papers on ventilation. The object I have in view is to state practically the workings of the methods as applied in different situations; and there have been so many *failures*, particularly when it should be otherwise expected from the eminence of the *men* who have undertaken the ventilation of hospitals and other buildings, *I wish to show the result of the means pursued by them*, and to give the authorities as best bearing on those results. Often in practice an appeal must be made to works *in some measure to be relied on*.

Those persons who have been engaged in the various elaborate modes of ventilation, had they considered the principle of a descending flue from a stove or a fire-place, *that it must be continuous, like a syphon*, they could have hardly expected that the vitiated air from a chamber could be brought by the horizontal flues, as constructed in many of the buildings, to a main shaft, and by it into the upright shaft.

Dr. Chowne's Experiments on Tubes in a Room.

Dr. Chowne found that in a tube open at both ends, placed upright in a room screened from the direct rays of the sun, and in which the air is perfectly quiescent, there is an upward current; and this current is sufficiently strong to turn a light horizontal

disk of paper suspended within the upper orifice of the tube. This may appear incredible; but as every cranny in the room was carefully closed, as the door was double, and the joints of the inner one covered with list, there remains no reason to doubt the fact. The longer the tubes, the more rapid was the revolution of the disks; and a similar effect was produced by increasing the diameter of the lower part of the tube so as to give it a conoidal form. Differences in the rate of velocity were observed as the temperature of the day altered from sunrise to sunset; the exclusion of light caused a retardation; and placing a shallow vessel containing strong sulphuric acid beneath the tube, so as to abstract the aqueous vapour, produced an entire stoppage of the disk after the lapse of thirty minutes. To test the effect on the whole atmosphere of the room, three bushels of quicklime were spread in shallow vessels on the floor. The rotation stopped in the smallest tube at the end of fifty minutes; and after ninety minutes, it all but ceased in the largest. Increasing the quantity of aqueous vapour by spreading damp cloths on the floor produced an opposite effect; there was a small increase in the rotations. It is by such experiments as these that we arrive at a true knowledge of what is required by ventilation; and, as I have before mentioned, all that can be expected of an architect is to prepare, in his buildings during their erection, such means for obtaining an *atmosphere* that would insure comfort and health to its inmates. We are told by Dr. Toynbee, "that 500 people in a church during two hours give off fifteen gallons of water into the air, which, if not carried away, saturates everything in the building, after it

has been breathed over and over again, in conjunction with the impurities it contains from each individual." The use of lamps, gas, and oil was shown by him to deteriorate the air, and to add much moisture to it. Thus the necessity of a ready and quick escape for the *vitiated air* to be removed as fast as generated.

Ventilation is distinct from Heating.

The proper ventilation of all buildings is to be considered as a distinct branch from heating. To ventilate a building on the best principles should be a first consideration, and to prevent the great evil of making ventilation subservient to heating, however desirable it may be to unite the two. Heated air, as a matter of economy, may be used in halls, corridors, or staircases, but it should never be used in living or workrooms of any kind in this country as a mode of ventilation. I have before mentioned that openings should be made for the entrance of fresh air equal to

Windows.

those for the escape of vitiated air. Thus, a casement window that is made to open as doors, is not so wholesome as a window hung in two divisions, so that one division will open up while the other can be opened down. Too much care cannot be observed that all windows may open *up and down* in *dwelling rooms*. Some of the various hospitals entirely depend on this system for ventilation, thus leaving the removal of the vitiated air to the care of the attendants; while the difficulty of overcoming neglect requires means to be adopted in other hospitals by flues, ventilators in the ceilings, and such like, all of which have their objections.

Military Hospitals in Kent.

One of the military hospitals in Kent contains eight wards, each about 64 feet long by 23 feet wide, and 13 feet 6 inches high, together with

smaller wards, and the usual apartments for military hospitals. There are nine windows, with two fire-places, in each ward. The mode of ventilation adopted is, a tube of wood is placed between the joists as large as the space between them will admit of, and each end passes through the walls, with large perforated metal ventilators outside of them. Each of these tubes have three openings, about 16 inches in diameter, from the wards, which are perforated with several inch augur holes; there are four of these tubes, having twelve circular perforated openings in the ceilings of each of the wards, together with the upper lights of the various windows being made to open as desired. In winter the air is driven down through the openings in the ceilings on the patients, rendering it necessary to have them almost continually closed, and making this mode of ventilation imperfect. Placing three openings in each of these tubes, with the ends of them open to the air, the result is that the cold air must be blown in, in volume, particularly if the fires are lighted.

Another garrison hospital comprises several wards, each about 33 feet by 18 feet, calculated to accommodate ten patients in each; the entire number of patients is about 328. The various wards on each floor are arranged on one side of the corridor, which is seven feet wide, and lighted by the front windows. Each ward has one window at the end. The door from the corridor is at the other end, and the fire-place at the side; it has two ventilators in the partition wall to the corridor, and two at the end in which the window is. These four ventilators are formed up next the ceiling, and have slides to open and shut as required. The corridor is ventilated by the windows only. This is

an improvement on the hospital last mentioned. I believe the mode of ventilation that has been used in many of the military hospitals is nearly similar to these two examples, *and thorough draught is generally complained of*.

Air Flues, as connected with Chimneys, to be avoided.

I am at present altering a large building in the country, that had been erected for an industrial school. Some of the large rooms had flues constructed in the chimney stacks with the fire-flues; those works had been well executed. I have noticed on the ceilings of each room, about those ventilators, the traces of the return smoke from the fire-flues. I have known other buildings in which such flues are built up in consequence of the return smoke; thus proving the necessity there is of avoiding the building of air-flues in the chimney stacks. If air-flues are built, they should have their outlets away from the chimneys.

Barrack Buildings.

I have noticed the plan, in some of the barrack buildings, of flues being built in the outer walls for the admission of cold pure air into the rooms, which is taken from under the eave course, and there are similar flues for the escape of vitiated air, which convey it up into the roof, where it passes out by large perforated slate ventilators placed in the gables. The fresh air flues are let into the apartments near the floor, with slides to them. As this causes draught, they are oftener closed than open. I see there is another plan just now about to be adopted. Two openings to each room are made between the windows, about nine by fourteen inches square, passing through the outer walls; on the inside a piece of wood is placed, so that the air cannot blow down,

and an angle is formed with it, which leaves an opening presented to an angle to the ceiling, which is covered with zinc, perforated with holes from one-eighth to one-tenth of an inch in diameter; these are for the inlets of pure air. A shaft is brought up from the ceilings in two angles of the rooms, made of well-seasoned wood about eight inches square in the clear; these are to pass direct up through the roof and let to stand over the slating, and the top capped and sides over the slating with luffer boarding, &c.; these shafts are on the same side with the fire-places. I can have no doubt but that they will act as down draught shafts, and will be continually stopped.

Plan as recommended by me for Ventilating Hospital Wards.

Some time ago I was applied to to give an opinion as to the best mode of ventilating an hospital about to be erected. There were to be nine rooms on each floor, each about thirty-six feet by twenty-three feet, and thirteen feet six inches high. The plan I recommended was, to have boxes made of strong smooth slate neatly put together; they were to be nine inches by four and a half inches in the clear, with the two ends open, and to be as long as the thickness of the walls would admit of; the two ends to have fine perforated metal plates secured over them; the space inside to have six divisions of perforated zinc, or fly wire, properly secured across them. I should have mentioned that the rooms were lighted with two windows at one end, the fire-place being at one side, and the door in the centre of the other end. I proposed placing four of these ventilators in the end, and as high up as the ceiling would admit of; that is, one over each of the windows, and one at the angles. I also had the upper lights of the windows made to open on

pivots, but to be hung three inches up from the meeting rails; they would open at the top in, say one foot, and to have cranked iron bars secured to sash frames for the sashes when open to rest on; the space between the meeting rails would present openings of near three inches; and for the purpose of diffusing the air coming in, there should be fine perforated zinc secured to the lower sash meeting rail, the entire width of the sash. Gussets should be formed of plain zinc or wood on the sash frames, to fill the spaces at the sides formed when the upper sashes are open back on the cranked iron bars; these are to prevent the air blowing in on the inmates. I have some confidence in this plan, as I know the efficacy of fine perforated zinc being placed in the upper sashes of the windows, where one of the usual squares of glass is replaced with it, after an experience of many years. Those acquainted with the beautiful principles of an instrument known as the respirator, will see at once the object I have for forming the ventilators as I describe; while an abundance of pure air will be at all times admitted into an apartment, all vitiated air will be effectually removed as fast as it is generated, and draughts perfectly prevented. Should these results be proved, the grand principle of hospital ventilation will be attained. I shall have great pleasure in showing the plans of various hospitals, and the mode of ventilation as adopted, to any person calling on me for this purpose.

Ventilation of Private Dwelling Houses.

In most of the private dwelling-houses the usual method of ventilation, when gas is used in rooms, is to place up over the lintels of the windows, or next the ceilings, perforated metal plates the size of bricks at the inside and outside of openings in thickness of the

walls. In some cases persons shut these, as they feel the draught from them; but I have found by the introduction of fine perforated zinc inside of the metal plates a preventative for the draught, while there is no annoyance from the gas. Some introduce tin tubes from the top of the gas lustre between the joists, which pass through the outer walls with a turn on them. Those tubes are said to be very dangerous, as they frequently become so hot as to set the timbers on fire. In a work which I have recently read, it has been accounted that the origin of many of the fires in London has been traced to this cause.

Barrack Buildings—Ventilation as adopted, a failure.

I have before given a description of the mode then being adopted under the directions of the commission on barrack building, which has been carried out at different barracks at a great cost. I mentioned my opinion at the time, that the shafts so formed for the exit of vitiated air would "act as down-draught shafts, and will be continually stopped." This opinion was formed about five months ago. I can now state with confidence that for a safe mode of ventilation it has proved a complete failure. The men suffer exceedingly at night from the quantity of cold air that blows down on them, and they are obliged to stop them up. In speaking of barrack-rooms, it must be borne in mind that there are generally twelve men in each, where the entire of the business required for their comfort is done during the day, and they sleep in them at night, hence the difficulty of adapting a mode of ventilation suited to all hours.

Palace of Westminster, London.—Mode of Ventilation as proposed.

It is generally understood, the modes that have been used to heat and ventilate the Houses of Parliament in London were on a most extensive scale,

exercising a vast amount of skill and experiment, together with a great expenditure of capital, amounting to many thousands of pounds; and looking to the exertions of Dr. Reid, Mr. Gurney, and others, with the talents displayed by the various committees, whose members must have possessed some knowledge of it, we expect to gain at once instruction so as to settle the question of the best mode of ventilation. In the year 1846 there were questions submitted by the Chief Commissioners of Woods to four gentlemen of experience, with regard to the warming and ventilation of the new Houses of Parliament, who considered Dr. Reid's system as he had explained it to them. It consists of one general scheme for the warming and ventilation of the whole of the extensive buildings, bringing in the atmospheric air from a considerable altitude at the Victoria and Clock Towers, at the two extremities of the building, carrying off all vitiated and impure air by a set of channels, both conveyed to a central tower, proposed to be of greater elevation than the towers already mentioned. "Although the scheme may be practicable, they were of opinion that it is too much complicated in its details and ultimate management to render such a system advisable to be applied to the whole building, including all the subordinate offices and private residences contained therein, and that the application of the system, considerably modified, should be restricted to such portions as comprise the new Houses of Parliament, their corridors, libraries, kitchens, and refreshment room." They considered that there would be advantage in applying the system, modified, in two divisions of the building, viz., to each House of

Parliament, and the various rooms belonging to it, separately and independently of each other; that it would be unnecessary to apply the system to the committee rooms, as they were capable of being warmed and ventilated in a more simple manner; and whatever warming and ventilation may be required to the Speaker's residence, and the other private residences, should be effected separately and independently of any other parts of the buildings, and from each other. The height of the Victoria Tower is about 342 feet.

In Mr. Goldsworthy Gurney's evidence before the Lords' Committee of the Houses of Parliament, to a question put to him, "If he objected to the system of ventilating from the floors or sides of the room instead of the upper portion of the apartments?" he stated that there was a practical objection to this system, which will be seen by looking at the subject carefully. Those retrogade currents produced from the entering at a low level into a room are practically very objectionable; they promote an increased rate of evaporation upon the skin, which produces a sensation of cold. Although the temperature of the air in motion may be warm, say seventy or eighty degrees, it will produce a temperature very considerably lower on evaporating surfaces. The skin of a human body is essentially an evaporating surface, and suffers much from this law. The feeling of cold in the parts of a room where retrogade or direct currents act, is not due to actual temperature of the currents, but to the increased rate of evaporation produced by them. Evaporation by retrograde currents rapidly absorbs heat. A person cannot get out of retrograde currents

if they enter near the floor, or at a low level; but if the openings are made in the ceilings, or at a high level, they are mixed with the atmosphere of the room above, and cease or become destroyed before they can possibly reach any one standing on the floor. There is a great objection to air for ventilation entering a room anywhere at a low level; it must necessarily impinge on the person.

Dr. Reid's principle was adopted in the House of Commons and lobby, and he took the fresh air from the Clock Tower. Sir C. Barry adopted the plan for the libraries, committee rooms, and House of Lords, taking the fresh air from the Victoria Tower; but the system of ventilation, as adopted by Dr. Reid, was found to be too complicated, and the want of mechanical arrangements prevented it succeeding. I find the present mode of ventilation adopted by Mr. Gurney is as follows:—The air is taken from the court yards, or over the roofs of corridors, and the Terrace river front, and old Palace yard, through openings into apartments; it is passed through canvass screens, and, if the weather permits, a fine disk of distilled water, thereby cleansing it. It is thence conveyed by flues especially made and arranged to each room, and also admission of fresh air by the windows. The vitiated air is withdrawn through valves above the ceiling of each room by flues to chambers in the roofs, and there discharged by the sides of the smoke flues, or into other shafts by means of fires in the same. There is provision for heating the air before it is conveyed to the several corridors, rooms, &c., as may be desired. There can only be one true principle

—that is, imperceptible currents of fresh and tempered air as required.

Experiments to prove the effect of Carbonic Acid confined in a chamber.

By experiments that have been made, it is now established that carbonic acid confined in a chamber will pass through brick walls, even if plastered with mortar on the inside, and through the plastering of ceilings, provided all is perfectly dry; but should they be damp or wet, or if the walls are built with stone, the carbonic acid will not thus pass off. This proves the necessity of the selection of such materials in the erection of dwelling houses, &c., as will assist the ventilation of apartments, particularly such as are calculated for the assemblage of a large number of persons. The walls when built of stone should be lined on the inside with bricks, or studded, and lath and plastered. Ash-pits, privies, soil-pipes from water-closets, sinks, and such like, should never be placed inside dwellings for this reason. Many years since, the walls and ceilings of the wards of an hospital in Dublin were oil painted, the governors being of opinion that they would remain cleaner, and be more in unison with the feelings of the occupants than the common lime whitewash. The mortality was so great before this error was discovered, that it was supposed a plague had set in in those rooms. It was evident that the carbonic acid could not penetrate the walls and ceilings as before, and a portion of it thus pass away. The experiment was tried of scraping all the paint off the walls and ceilings; or, I believe, the old plastering was entirely removed from them, and replastered and lime whitewashed; thus they restored the salubrity of those rooms, and ensured a

The selection of Materials to be used in Buildings.

speedy restoration to health of the patients confined in them since.

Places of Worship.

In places of worship, where large numbers congregate for two hours at a time, it is usual to place one or more large perforations, formed of ornamental open-work, in the ceilings, to allow the carbonic acid formed by the respiration to pass into the roof above the ceiling, and from thence through luffer-boarded openings into the air. Frequently the vitiated air returns from the roof, and the wind blows in through the luffer-boarded openings, and comes down in volume on the heads of the congregation. To prevent this evil, I have seen large shutters so constructed by pulleys and ropes that the openings may be closed at pleasure. Now, if a tube of zinc or other air-tight material, with a large bell mouth, (similar to a glass tube over a lamp) was placed over those openings, and brought out through the ridge, and properly capped, to prevent the wind blowing down, with the aid of the windows at each side, a safe and perfect mode of ventilation would at all times be ensured. If three of those were placed over the ceilings of our courts of justice, we should not hear any more complaints of the dreadful effects of the want of proper ventilation. School rooms and such like should be ventilated in a similar manner. In some of the churches where I have been professionally engaged, I have brought flues twelve inches square, two from the under-ceiling of each transept gallery, and two flues above, with openings from near the ceiling of the church (that is, eight flues from the transepts); they come out on different parts of the parapets, with perforated metal at bottoms, to be opened or shut as

required, and they have been found to assist the ventilation very much. Flues of similar construction built in the walls, let out at the parapets or at the eaves, I have found to be very useful from large kitchens, bakehouses, &c.

I have endeavoured to show that the effects of carbonic acid from respiration should be removed at the top of apartments, and not at the bottom, as some have attempted, producing similar results to those recorded in the report of the committee to the members of Health of Towns Association, wherein it is mentioned that a new house was erected at the Zoological Gardens in Regent's Park to receive the monkeys: when finished, sixty monkeys were placed in it, which had already borne several winters in England; a month afterwards fifty of these were dead, and the few remaining ones were dying. The cause of this was the belief that the room would be best ventilated at the skirting by apertures in it round the room; that the weight of the carbonic acid produced by the respiration of the animals, because heavier than the other air, would separate from it and escape below. This mistaken principle of ventilation is the cause of many sad and afflicting cases of consumption, while the simple opening up and down of the sashes of a window will render an apartment in a short time salubrious, by allowing the confined carbonic acid to dilute speedily with the oxygen of pure air.

Stables. In the year 1847 I had erected at the mansion of a nobleman, in the county of Kildare, an extensive range of stable offices. The mode I adopted to ventilate them was: the doors were in the centre of side walls of each of the stables; two windows were

placed on the same side, as high up as possible, the sashes of which were hinged to the sills of the sash frames so as to open in by cranked bars secured to the frame, to enable them to open to an angle of 45 degrees. Similar windows were over the doors. In the centre line of the ceiling of each stable were placed two tubes ten inches square, with bell mouths at bottom opening to sixteen inches square, going from the ceiling to the ridge board; over those, on the ridge, were placed round tubes made of strong lead, about eight inches diameter, standing over the ridge about one foot; a cap is formed on them coming down to near the ridge stone, of a much greater diameter, also of lead, and secured to the tube. It will be seen that this mode of ventilation prevents the air coming down on the heads of the animals.

Farm Buildings. In the years 1849-50 I had erected several farm buildings on the estate of another nobleman in the county of Meath. I used similar ventilating methods in the various cattle sheds, taking every precaution that there were no openings over the heads of the animals. An experience of some years now shows the efficacy of those modes, as I have the assurance of the tenants occupying the latter, that during the entire rage of the epidemic around them, which carried off a number of cattle, those offices have been perfectly free, and that they sustained no loss whatever; and in the former, the noble proprietor, when wishing additional stabling to be completed at a recent period, requested me not to alter the ventilation I had adopted for the other stables.

I have now brought under notice every matter I consider necessary on ventilation, and I have con-

fined myself to such matters as may be carried out in all buildings as suited to our requirements; and hope that sanitary measures may become an essential part of the profession of an architect. I do not wish in the least degree to *dictate*, but an experience of many years' practice shows me that a continued study of the branches of sanitary architecture is one of the best qualifications in a member of my profession.

APPENDIX.

The author begs to state that he has been professionally engaged by the following:—

Harold's Cross Church.

Erected in the years 1836-7, and enlarged in the year 1856.

Kingstown Mariners' Church.

31st, May, 1838.

Dear Sir,—I am directed by the Trustees of the Kingstown Mariners' Church to convey to you their satisfaction at the manner in which the entire work of the Chaplain's house attached to this Church has been executed; and at the same time to express their thanks for your attention to all their suggestions, and uniform kindness in attending their meetings, when, it is to be feared your compliance must have been often attended with much personal inconvenience. Indeed, the entire buildings have been executed in a manner highly creditable to your judgment and professional skill as an Architect.

I am, dear Sir, yours truly,

John Lough, Esq. Robert Daly.

Carysfort Church, Black Rock.

The old Church was enlarged; and, at a subsequent period, the old portion was taken down and re-built to match the new portion.

Baggott Street Asylum Church.

The alterations to this Asylum, together with the extensive Laundry buildings.

Jews' Synagogue, Mary's Abbey.

New floored, with new joists, pews, &c., and alterations, &c.

St. Matthias' Church, Dublin.

New transepts, chancel, stairs to galleries, and other alterations.

Primitive Wesleyan Methodists.

Presented by the Members of Conference of Primitive Wesleyan Methodists, July, 1841, to John Louch, Esq., Architect.

We feel that benefits of very considerable importance have been conferred on our Society in general, from the plans and estimates of Preaching-houses and Residences for Preachers which Mr. Louch has furnished gratuitously for some years past. Owing to his superior professional knowledge, the appearance and convenience of the buildings have been greatly improved, as well as a considerable reduction in the cost of their erection effected; and, as a small mark of the estimation in which we hold his valuable services, we beg his acceptance of a copy of Dr. Adam Clarke's Commentary, to be presented to him by the members of our Building Committee.

A. Stewart, Vice-President.
G. Robinson, Secretary.

Mount Jerome Cemetery, Harold's Cross.

Entrance Lodge, Gateway, surrounding Walls, and Survey.

Right Honorable Earl of Howth.

Repairs and Alterations to Howth Castle.

Wesleyan Methodist Alms House.

The Trustees of the Methodist Alms House for Aged Females, at their first meeting held in their newly erected building in Grantham Street, unanimously resolved to present to John Louch, Esq., Architect, a copy of Taylor's edition of Calmet's Dictionary of the Holy Bible, in 5 vols., quarto, in testimony of their sense of obligation to him for his valuable professional services, voluntarily and gratuitously rendered, by the supply of plans and specifications for the erection of, and superintending the work to its completion, an habitation for our aged widows, to replace the Whitefriar Street Asylum, the lease of which is nearly expired.

Charles Shaw.
George Ledford, Secretary.

2nd April, 1858.

Late Countess of Clanricarde.

Repairs and alterations to her Ladyship's Mansion in Domnick Street.

Late Sir John Blake, Bart.

Repairs, &c., to House in Ely Place, Dublin.

Right Honorable Lord Viscount Powerscourt.

By his Lordship's Guardians, to report and superintend various buildings on this estate during his minority.

Honorable C. J. Trench.

Schools at Breckna, Queen's County.

Right Honorable Earl of Roden.

To erect the additions to Jollymore Park House, County Down.

Most Noble Marquis of Drogheda.

For the very extensive additions, together with the Offices, to Moore Abbey, County Kildare; Demesne Wall, principal entrance; Farm Buildings at Clara; Agent's House and other Houses, at Monastereven.

Right Honorable Earl of Arran.

Designs for Entrance Hall, &c., to Saunders Court House, County Wexford.

James E. Anderson, Esq.

Designs and all the Working Drawings for Mansion on his Estate near Waterford.

Late James Boyse, Esq.

Extensive Additions to Bannow House, County Wexford.

Late Rev. Henry Moore.

Additions to the Glebe House, Ferns, County Wexford.

Right Honorable Earl of Darnley.

Alterations to Clifton Lodge; also extensive ranges of Farm Buildings and Residences on different parts of his Lordship's Estates in the County of Meath.

Late Right Honorable Lord Viscount Lorton.

To examine and report on various Works required to Rockingham House, County Roscommon.

Late Honorable Major Forrester.

Various alterations and additions to Ashtown Cottage, County Dublin.

Late Right Honorable Lord Viscount De Vesci.

Alterations and repairs to Abbey-Leix House, Queen's County.

Honorable and Rev. William Wingfield.

New Glebe House at Abbey-Leix; also completing the buildings of Glenbawn House, at Abbey-Leix.

Sir Francis Hopkins, Bart.

Repairs to the Mansion at Rochfort, County Westmeath.

Edward J. Medlecott, Esq.

New Mansion at Dunmurry, County Kildare.

Alderman Richard Atkinson.

His House and Offices at Hyfield, Rathgar, County Dublin.

Thomas Lefroy, Esq., Q.C.

His House and Offices, Earlsfort Terrace, City of Dublin.

J. E. V. Vernon, Esq.

Alterations, &c., to Clontarf Castle, County Dublin.

Late James Ferrier, Esq.

Houses at Booterstown, County Dublin.

Of the Business Houses which have been erected or fitted out by the Author, are the following:—

Messrs. West & Son, College Green.
Messrs. W. Fry & Co., Westmorland Street.

Messrs. Forrest & Son, Grafton Street.
Messrs. Higgenbotham, Grafton Street.
Messrs. R. Atkinson & Co., College Green.
Messrs. Waterhouse, Dame Street.
Messrs. Hyam & Co., Dame Street.
Messrs. Warren, Dame Street.

The following Houses have been erected for different Proprietors, under the direction of the Author.

Six Houses at Warren Point, Clontarf.
Two Houses at Howth.
Nine Houses, Upper Prince Edward Terrace, Black Rock.
Four Houses, Pembroke Road.
Eleven Houses in neighbourhood of Bullock.
Seven Houses near Kingstown.
Six Houses in Kingstown.
Six Houses, Mount Pleasant
Four Houses at Sandford Place.
Two large first-class Houses on Wellington Road.
Two Houses at Vesey Place, Monkstown.
Eight Houses on Waterloo Road.
Two Houses at Lansdown Road.
Two Houses, Haddington Road.
Four Houses at Mount Street Crescent.
Two Houses, Fitzwilliam Place.
Six Houses, Epworth Terrace.
Four Houses, Gawille Avenue, Rathgar.
Four Houses, Rathmines Road.
Two Houses, Kenelworth Square.
Two Houses, Rathgar Road.
One House and Offices at Rathfarnham.
Four Houses, Peter's Place.
Three Houses, Adelaide Road.
Five Houses, Baggott Street.
&c. &c. &c.

Many years have been devoted to the measuring and valuing the various artificers' works in buildings, and in valuing houses and lands, for the purposes of mortgage, sale, division, or as evidence before the different tribunals requiring the property for public or railway purposes, and for various government departments in Ireland.

Printed by James Brine, 10, Week Street, Maidstone.

ON THE NATURAL CONSTANTS

OF THE

HEALTHY URINE OF MAN,

AND

A THEORY OF WORK FOUNDED THEREON.

BY

THE REV. SAMUEL HAUGHTON, M.A., F.R.S.,

FELLOW OF TRINITY COLLEGE, DUBLIN.

READ BEFORE THE ASSOCIATION OF THE KING AND QUEEN'S COLLEGE OF PHYSICIANS.

DUBLIN:
PRINTED AT THE UNIVERSITY PRESS,
BY M. H. GILL.
1860.

[From the Dublin Quarterly Journal of Medical Science, Aug. 1859; and Aug. 1860.]

CONTENTS.

		Page.
1.	Division of Excretions and Work,	2
2.	Daily Excretion of Urea, according to former Writers,	3
3.	Excretion of Urea by Beef-eaters,	5
4.	„ „ Vegetarians,	6
5.	Parabolic Curve of Urea,	9
6.	Urea excreted by Sheep,	10
7.	Urea found after death in the Pericardium,	10
8.	Daily Excretion of Uric and Hippuric Acid,	11
9.	Daily Excretion of Phosphoric Acid,	12, 13
10.	Excretion of Phosphoric Acid, not due to the Disintegration of the Nervous Centres,	13, 14
11.	Analyses of Food,	19, 20
12.	Comparison of Nitrogen, ingested and excreted,	22
13.	Similar Comparison, by M. Barral,	23
14.	Coefficient of Vital Work (or number of Grains of Urea excreted requisite to keep 150 pounds weight of Man alive) determined,	27
15.	Coefficient of Mechanical Work found,	28
16.	Coefficient of Mental Work (Study),	32
17.	Coefficient of Mental Work (Routine),	32
18.	General Conclusions,	33, 34

ON THE NATURAL CONSTANTS

OF

THE HEALTHY URINE OF MAN,

&c. &c.

The following investigation was suggested to me by a remark of Bernouilli, to the effect that the total quantity of work of which a healthy man was capable appeared to him to be constant, no matter in what description of labour he was employed. The facts, as he understood them, were against this view: and he was forced reluctantly to give up his opinion. Before I read the opinion of this great man, I had arrived, from reflection and observation, at a similar result; and I think good reasons may be assigned, in the present state of our chemical knowledge, for a reasonable belief in this opinion. It is generally believed that the daily food we consume undergoes a process of oxidation before it is finally removed as waste matter from the system. This may be, and doubtless is, far from the whole truth; but, as far as it goes, it is true, and its investigation to its legitimate consequences must lead to correct views of nutrition and labour. It occurred to me that the natural and proper measure of the value of a given diet was the amount of work it would produce in a healthy animal, such as man. This was Bernouilli's idea, but he applied it simply to mechanical labour of which man was capable, and so fell into error, natural to the deficient knowledge of his time.

The following appears to be a juster view of the subject:—

1. A man in health consumes, per day, a certain amount of food and drink, and excretes per day an equal amount of waste

[a] See vol. xxvii. p. 374.

matter, supposing him to be full grown, in good health, and not gaining or losing flesh:—

2. The excretions are effected in four ways: *per halitum, per cutem, per vesicam, per anum*, and are more highly oxidated than the food and drink consumed.

3. The difference of oxidation of the *ingesta* and *excreta*, converted into work, should account for, and be equal to, the total labour effected per diem.

4. The work done per day may be divided into—

a. The effort necessary to live, or the work spent on the performance of organic functions—*Opera vitalia.*

b. The work, converted into heat, requisite to keep up the animal temperature—*Opera calorifica.*

c. The mechanical work effected by bodily labour—*Opera mechanica.*

d. The unknown, and hitherto unmeasured, work done by the mind—*Opera mentalia.*

It is an opinion of physiologists, and appears to be well founded, that the animal heat is fully accounted for by the oxidation of food effected *per halitum*. There remain, therefore, three excretions to consider, and three kinds of work to account for by their means, viz.:—

Excreta.—1. Per cutem; 2. Per vesicam; 3. Per anum.

Opera.—1. Vitalia; 2. Mechanica; 3. Mentalia.

I shall not attempt, in this communication, to discuss the general question, but shall confine myself to those subjects on which I have experimented directly, viz., the excretions *per vesicam*, and the mechanical labour of body, and work of mind, of which man is capable.

My experiments on urine were all made on the urine of healthy men, and were directed chiefly to the determination of the daily quantities of nitrogen and phosphorus excreted. I was forced into this course of chemical experiments by finding, in various authorities consulted by me, the most various estimates of a quantitative character relative to these subjects. For the convenience of reference, I shall divide my paper into four parts:—

I. The daily discharge of urea in healthy urine of man.

II. The daily discharge of uric acid in healthy urine of man.

III. The daily discharge of phosphoric acid in healthy urine of man.

IV. Comparison of the foregoing with the daily work, bodily and mental, and with the daily food, of man.

Part I.—*The daily discharge of Urea in healthy Urine of Man.*

The earlier writers on this subject refer the quantity of urea either to the fluid ounce, or to 1000 parts: both of these modes of estimating urea have been properly discarded by later writers, who estimate the quantity of urea discharged per twenty-four hours, considering this to be the natural period of man's cycle of labour, food, and rest.

The English medical writer on Urine who appears to be held in most repute is the late Dr Golding Bird, whose work has passed through five editions. He does not appear to have ever made any experiments himself, but is content to quote[a] from Becquerel's work.

I.—*Daily Urea.*

Daily urea of man, ...	270 grains.
Daily urea of woman, ...	240 ,,

The following determinations of urea in 1000 parts have been given by other writers:—

II.—*Urea in* 1000 *Grains.*

Berzelius, ...	30·10 grains.	
Lehmann, ...	31·45 ,,	
Ditto, ...	32·91 ,,	
Ditto, ...	32·90 ,,	
Becquerel (man), ...	13·84 ,,	1019 sp.gr.
Ditto (woman), ...	10·36 ,,	1015 ,,
Simon, ...	12·46 ,,	1011 ,,
Ditto, ...	14·58 ,,	1012 ,,

The discrepancies in the foregoing results show that it is absolutely necessary to fix upon some standard of comparison, such as the total quantity per twenty-four hours; otherwise our results will be vitiated by the drinking of a glass or two more or less of water per day, and by other trivial causes. Lehmann's three results are stated to be derived from twenty-four hours' urine of a well-fed and healthy young man, but the total quantity passed is not stated.

[a] Fifth Edition, page 75.

Elsewhere M. Lehmann obtained the following interesting results from experiments made on himself:—

III.—*Urea per* 24 *Hours of M. Lehmann.*

Animal Diet.	Mixed Diet.	Vegetable Diet.	Non-Nitrogenous Diet.
821 grs.	501½ grs.	347 grs.	238 grs.

The following results, given by M. Lecanu, are also referred to a period of twenty-four hours:—

IV.—*Urea per* 24 *Hours, determined by M. Lecanu.*

Adult men, ... 431·9 grs.
Adult women, ... 294·2 ,,
Very old men (84–86), ... 124·8 ,,
Children (under 8), ... 138·2 ,,

Bischoff also has determined the daily quantity of urea passed by a well-fed man, of thirty-one years of age.

V.—*Urea per* 24 *Hours, at* 31 *Years of Age, by M. Bischoff.*

Urea, ... 585 grs.

On the preceding Tables the following remarks may be made:—

No. I. is to be rejected altogether, as it is totally erroneous by defect, and the deficiency must have arisen from the use of some imperfect method of determining the urea, such as by the nitrate or oxalate.

No. II. is unintelligible without a statement of the total quantity of urine passed per day in each case.

No. III. I believe to be excellent, as far as it goes, but it rests upon experiments made upon a single individual.

No. IV. omits to state the diet of the individuals experimented upon, and is, therefore, less interesting than it would otherwise have been.

No. V. agrees admirably with my own results.

The foregoing summary contains, I believe, all that was known on this subject at the time I commenced my experiments, which I should never have undertaken had I found it possible to obtain reliable information in the books.

I made some preliminary trials as to the various methods in use among chemists for the determination of urea quantitatively, and found Liebig's volumetric process, by means of nitrate of mercury, to give the most accurate and consistent results.

Having decided on the mode of analysis to be employed, I divided my subjects for experiment into two classes:—

1. Well-fed, flesh-eating, wine-drinking men.
2. Well-fed, water-drinking vegetarians.

The following Tables contain the result of my experiments:—

Table A.—*Beef-eaters.*

No.	Urea, per Day.	Urine, per Day.	Specific Gravity.	Urea, per fluid ⅓.	Solids, per Day.
	Grains.	Ounces.		Grains.	Grains.
1	465·09	34	1023·8	13·70	817·25
2	677·25	62	1019·0	10·11	1065·69
3	644·62	52	1018·5	12·40	998·55
4	554·00	50	1015·8	11·08	818·00
5	630·00	45	1028·2	14·00	1330·20
6	484·30	41	1024·8	11·81	1051·25
Mean,	**575.87**	**47.3**	**1021.7**	**12.18**	**1013.49**

No. 1. Age, 37; weight, 126 lbs.; occupation, walks 5 miles per day, studies 5 hours per day; food, 8 oz. fresh meat; 8 oz. white bread; 10 oz. vegetables.

No. 2. Age, 35; weight, 126 lbs.; occupation, partridge-shooting, office-work 3 hours, anxious about business matters; food, mixed diet.

No. 3. Age, 19; weight, 126 lbs.; occupation, studies 5 hours; walking and athletic games, 2 hours; won the prize for the long jump at the Trinity College Foot Races; food, mixed.

No. 4. Age, 39; weight, 174 lbs.; occupation, walks 2 miles, office-work, 6 hours; food, mixed.

No. 5. Age, 40; weight, 189 lbs.; occupation, walks 4 miles, studies 4 hours; food, mixed.

No. 6. Age, 40; weight, 145 lbs.; occupation, walks 5 miles, studies 2 hours; food, mixed.

From the preceding results it follows that 576 grains of urea per day is the natural discharge, *per vesicam*, of the class

of men experimented upon. This agrees with Bischoff's and Lehmann's experiments made upon themselves.

I shall make no observation at present on the range from 100 grains below to 100 grains above the average, as I do not believe it to depend on any single circumstance, such as weight, bodily exercise, &c., but upon a combination of circumstances which I hope fully to discuss in the concluding part of this paper.

I have also to add that every one of these specimens of urine gave, on the addition of nitric acid, at 50° Fahr., after standing twenty-four hours, a precipitate of crystals of nitrate of urea. This circumstance I do not believe to be a proper test of excess of urea in urine.

The following Table contains the corresponding results of my experiments on vegetarians:—

Table B.—*Vegetarians.*

No.	Urea, per Day.	Urine, per Day.	Specific Gravity.	Urea, per fluid ℥.	Solids, per Day.
	Grains.	Ounces.		Grains.	Grains.
1	367·50	70	1014·00	5·25	1012·90
2	578·81	81	1014·76	7·14	1236·87
3	315·00	45	1015·23	7·00	712·50
4	366·12	56	1012·41	6·54	717·08
5	342·55	43	1017·17	7·87	775·60
Mean,	**393.99**	**59**	**1014.71**	**6.76**	**890.99**

No. 1. Age, 63; weight, 173 lbs.; occupation, walks 3 miles per day, office-work 1 hour; food, 1 egg, 6 oz. bread, vegetables *ad lib.*, 16 fl. ℥ milk, 40 fl ℥. water.

No. 2. William Wall, 16th Regt. (2nd Battalion), in Military Prison, Dublin. Age, 22; weight, 132 lbs.; occupation, 3 hours' shot drill, 1¼ hour ordinary drill, 3½ hours oakum picking; food, breakfast, 8 oz. oatmeal, ½ pint of milk,—dinner, 9 oz. Indian meal, ½ pint of milk,—supper, 8 oz. of bread, ½ pint of milk.

No. 3. William Tysack, Military Train, in Military Prison, Dublin. Age, 31; weight, 146 lbs.; occupation and food, same as last.

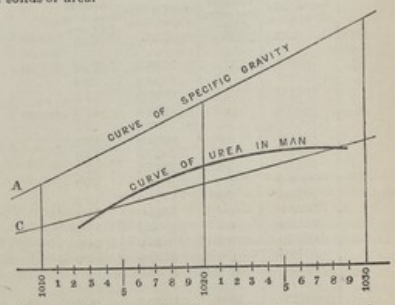
No. 4. Thomas M'Donald, 18th Regt. (2nd Battalion), in Military Prison, Dublin; age, 22; weight, 146 lbs.; occupation and food, same as last.

No. 5. Same man as No. 3, a fortnight subsequent.

From the preceding, it follows that 394 grains is the natural discharge of urea from a well-fed vegetarian per day. No. 2 appears to be exceptional, and above the usual average; but, even allowing for this, it would appear that the average for vegetarians is considerably above what is considered usually by medical men in this country as the proper average of well-fed men, on the authority of Golding Bird. It is quite certain that a physician, applying Bird's rule in his private practice, would, probably, pronounce 90 well-fed men, out of every 100 who consulted him, to be labouring under diabetes insipidus: although, from the foregoing experiments, they should be entitled to be considered as in the enjoyment of excellent health.

I shall now attempt to connect the quantity of urea in healthy urine with the specific gravity.

The result of all my experiments is shown in the annexed diagram, in which the base line represents the specific gravity, and the vertical ordinates, the number of grains per fl. ℥, either of solids or urea.



The right line A, marked *Curve of specific gravity*, is drawn by making the ordinate equal the abscissa, minus 1000; thus, 1015 sp. gr. has an ordinate of 15 units measured off; 1020 sp. gr. has an ordinate of 20, and so on.

The line C is formed by dividing the ordinates of the line A in half; and the *curve of urea in man* is formed by taking for each specific gravity observed, an ordinate equal to the number of grains of urea per fl. ℥ corresponding.

This curve of urea intersects the line dividing the ordinates of the curve of specific gravity in half, at points corresponding to the specific gravities 1014 and 1028. If, therefore, the specific gravity of healthy urine be 1014 or 1028, the number of grains of urea per fl. ℥ will be 7 or 14 respectively. For all specific gravities intermediate between these limits, the number of grains of urea per fl. ℥ will be *greater* than half the excess of the specific gravity above 1000; and for specific gravities *below* 1014 and *above* 1028, the number of grains of urea per fl. ℥ will be *less* than half the excess of specific gravity above 1000.

The following Table contains the summary of all my experiments on this subject:—

Table *derived from Experiment, showing the relation between the Specific Gravity of Healthy Urine, and the Quantity of Urea contained in it.*

Specific Gravity of Urine.	Grains of Urea per oz. of Urine.	Specific Gravity of Urine.	Grains of Urea per oz. of Urine.
1010·0	. . .	1020·0	12·2
1010·5	. . .	1020·5	12·4
1011·0	. . .	1021·0	12·6
1011·5	. . .	1021·5	12·9
1012·0	. . .	1022·0	13·1
1012·5	5·0	1022·5	13·3
1013·0	5·7	1023·0	13·4
1013·5	6·6	1023·5	13·5
1014·0	7·2	1024·0	13·7
1014·5	7·8	1024·5	13·8
1015·0	8·4	1025·0	13·9
1015·5	8·8	1025·5	13·95
1016·0	9·2	1026·0	14·0
1016·5	9·6	1026·5	14·0
1017·0	10·0	1027·0	14·0
1017·5	10·4	1027·5	14·0
1018·0	10·9	1028·0	14·0
1018·5	11·2	1028·5	13·95
1019·0	11·5	1029·0	13·9
1019·5	11·9	1029·5	. . .

It is evident from the diagram, p. 7, that the curve which represents the relation of the quantity of urea in healthy urine to the specific gravity of the latter is not a right line; or, in other words, that the quantity of urea is not *proportional* to the specific gravity.

The curve laid down in the diagram is a representation of the Table, and may be algebraically expressed by a parabolic curve, as follows:—

Let y denote the grains of urea per fl. ℥ in any specimen of urine, not containing either sugar or albumen; and let x denote the excess of specific gravity above 1000. Then,—

$$y = A + Bx + Cx^2. \qquad (1)$$

And, since when $x = 14$, $y = 7$; and when $x = 28$, $y = 14$; and the curve then reaches its highest point, it is easy to show that,—

$$\begin{aligned} A &= -14 \\ B &= 2 \\ C &= -\tfrac{1}{28} \end{aligned} \qquad (2)$$

From these values it can be shown that the curve of urea (1) may be put into the form,—

$$[28 - x]^2 = 28\,[14 - y], \qquad (3)$$

which is the equation of a parabola whose parameter is 28.

It is easy to verify this equation by the Table already given. For example, let the specific gravity be 1018, then we have,—

$$\begin{aligned} (28-18)^2 &= 28\,(14-y) \\ 100 &= 28\,(14-y) \\ y &= 10{\cdot}5 \text{ grs.} \end{aligned}$$

Let specific gravity = 1024, then,—

$$\begin{aligned} (28-24)^2 &= 28\,(14-y) \\ 16 &= 28\,(14-y) \\ y &= 13{\cdot}43 \text{ grs.} \end{aligned}$$

These results, and others similar to them, obtained from the parabolic curve (3), differ slightly from those found directly by experiment, and given in the Table, showing that the parabolic curve does not accurately represent the curve of urea; but it evidently does so within a degree of approximation much less than is required by medical men, for whose purposes the following bedside rule may prove quite sufficient:—"*Half the excess of the specific gravity of urine* (*not containing either sugar or albumen*), *above* 1000, *is the number of grains of urea per fluid* ℥."

This rule gives the line C, p. 7, for the curve of urea, with which it agrees at the specific gravities of 1014 and 1028.

The following determination of urea in sheep's urine may prove interesting, for comparison with the urine of man; the range of specific gravity is greater, and the proportion of urea to specific gravity is also greater:—

	Specific Gravity.	Urea, Grains, per fluid ℥.
Urine of ram, ...	1057·06	37·33
Urine of ewe, ...	1008·93	6·41

Before concluding this part of my paper relating to urea, I may add the following interesting facts, although they do not properly belong to the subject of healthy urine. I examined carefully for urea, having first got rid of albumen, the serous effusion of the pericardium of two subjects, both women, one of whom had died of Bright's disease, with dropsy; and the other had perfectly healthy kidneys. The result of the examination was as follows:—

	Grains, per fluid ℥.
1. Subject with diseased kidneys, . . .	3·05
2. Subject with healthy kidneys, . . .	1·31

The difference in the two cases is sufficiently striking, and the total quantity of urea in the dropsical effusion of No. 1 must have been very great; yet the quantity in the case No. 2 is also so large as to make us hesitate as to the conclusions to be drawn from the qualitative determination of the presence of urea found, on a post-mortem examination, in the pericardium, or in the ventricles of the brain.

Part II.—*The daily discharge of Uric Acid in healthy Urine of Man.*

The following Tables contain the results I obtained from my experiments, with respect to Uric and Hippuric acids. They are so different from those usually received that they would require explanation, which I should offer, were it not that I believe that both uric and hippuric acids are accidental in healthy urine, though the former is always found in small quantity; the latter, hippuric acid, occurred to me only once, though it was carefully sought in each case. When I say that uric acid is accidental in healthy urine, I do not mean to say that it occurs like chlorine and sulphuric acid, the quantity of which depends directly on the chloride of sodium, and sulphate of alumina and potash consumed with the food; but I

do mean, that no uric acid whatever should occur in the urine of a man in perfect health, but that all the nitrogen of the urine should pass off in the form of urea, a more highly oxidated product than uric acid.

The following determinations of uric acid passed in twenty-four hours have been given:—

VI.—*Uric Acid discharged per Day.*

	Grains.
Becquerel, ...	8·10
Neubauer, ...	5·08
Ditto, ...	7·63
Lehmann, ...	18·17

It is stated by Liebig, that 7·5 grains of hippuric acid are discharged by the urine per day; this is quite at variance with my results, as will appear from the following Tables:—

Table C.—*Uric Acid per Day (Beef-eaters).*

No.	Grains per Day.
1, ...	1·02
2, ...	11·88
3, ...	1·04
4, ...	7·40
5, ...	5·29
6.	0·71
Mean, . .	**4.55**

Table D.—*Uric Acid per Day (Vegetarians).*

No.	Grains per Day.
1, ...	0·50
2, ...	0·71
3, ...	1·69 partly hippuric acid.
4, ...	2·48
5, ...	2·03 partly hippuric acid.
Mean, . .	**1.48**

When we consider that the quantity of urea passed by the persons considered in these Tables, per day, is 576 grains, and 394 grains, respectively, we may fairly consider the uric acid discharged simply in the light of a minute fraction of nitrogen, which has escaped complete oxidation, and as a matter rather of chemical than physiological interest.

The only case in which I detected hippuric acid was that of William Tysack (Nos. 3 and 5, Tables B and D). His urine

had a remarkable smell, compounded of that of sweet hay and apple-juice, and differed in its sensible qualities from the other urines examined.

All the preceding experiments were made on not less than from 20 to 50 ounces of urine, and I am certain that all the uric acid was obtained.

Part III.— *The daily discharge of Phosphoric Acid in healthy Urine of Man.*

There is no subject respecting which more difference of opinion exists than with reference to Phosphoric acid eliminated by the kidneys; some investigators considering that it is as accidental in its character, and as dependent on food, as chlorine or sulphuric acid; while others regard it as the direct product of the disintegration of nervous tissue, and, as such, entitled to our regard as the measure of nervous work done by the system. In whatever point of view it is to be considered, it is obviously important to determine its daily amount in the case of healthy men, under different conditions as to food and work of mind and body. I therefore determined its absolute amount per day with much care; and, in my later experiments, ascertained also the proportion in which it occurred combined with alkalies, as well as that in which it was found in combination with lime and magnesia.

The following Tables are divided, as before, into the flesh-fed and vegetarian subjects:—

Table E.—*Discharge of Phosphoric Acid per Day* (*Beef-eaters*).

No.	Phosphoric Acid.	Weight.
	Grains.	lbs.
1	47·14	126
2	43·28	126
3	40·78	126
4	38·10	174
5	23·72	189
6	29·43	145
Mean,	**37.07**	

I have placed the weights of the several subjects in connexion with their respective discharges of phosphoric acid, and I think it will be admitted that there is no direct proportion

between them, such as would be the case, were the discharge of phosphoric acid dependent simply on a waste of albuminous or other tissues proportional to the mass of the individual considered.

Table F.—*Discharge of Phosphoric Acid per Day* (*Vegetarians*).

No.	Total Phosphoric Acid.	In combination with Earths.	In combination with Alkalies.
	Grains.	Grains.	Grains.
1	30·00		
2	32·47	5·92	26·55
3	22·78		
4	27·54	6·30	21·24
5	20·70	3·46	17·24
Mean,	**26.70**	**5.23**	**21.67**

I have reason to believe that the proportion of phosphoric acid, in combination with alkalies, to that in combination with earths, given above, viz., 4 to 1, is the same in well-fed men as in vegetarians, and that it may be confidently used in drawing conclusions with respect to this subject.

To show how ill founded the idea is, that the waste of nervous matter in the system gives rise exclusively to the discharge of phosphoric acid, the following calculations may prove of some interest. Adopting Vauquelin's determination of the phosphorus in the brain at 1·5 per cent, and taking the weight of the brain and spinal cord from the most recent experiments, at 51 ounces, we find, the following chain to determine the total phosphoric acid existing in these nervous centres at any given moment:—

Term of demand, 1 man.
1 ⟵ 51 ℥ nervous matter.
1000 ⟵ 15 ℥ phosphorus.
32 ⟵ 72 ℥ phosphoric acid.
16 ⟵ 7000 grs. phosphoric acid.

= 753·04 grs.

This quantity of phosphoric acid in the nervous system of a well-fed man, discharging 37 grains per day, *per vesicam*, would only last for twenty days; and although the brain is better supplied with blood than any other portion of the body, it is quite

incredible that the process of renovation can proceed at such a rate as to replace all its phosphorus in twenty days.

On the other hand, we may fairly conclude that only the phosphoric acid, in combination with lime and magnesia, can be considered as having its origin in the wear and tear of the bony tissue; and, assuming the average weight of bone-earth in a man of 150 lbs. to be 10 lbs., we find the following chain to determine the number of years in which his bones can be removed *per vesicam*, and replaced by suitable food:—

Term of demand, 1 man.
1 — 150 lbs.
150 — 10 lbs. bone earth.
156 — 72 lbs., PO_5.
1 — 7000 grs. PO_5.
5 — 1 day.
365 — 1 year.

= 17·70 years.

Whatever may be thought of the idea that our bones are replaced in the manner here supposed, the result obtained is certainly less improbable than the former. I give them both, without attaching weight to them, as examples of the uses to which the analyses of healthy urine may be turned.

The following summary contains the results of my experiments on the eleven specimens of urine of twenty-four hours, examined by me. I made several careful determinations of the relation between specific gravity and the total quantity of solid matter per fluid ℥, and I found my experiments agree so closely with the Table published in Dr. Golding Bird's work, and deduced from Dr. Christison's formula, that I adopted the results of that Table in all my subsequent experiments.

Table G.—*Well-fed Beef-eaters.*

No. 1.

	Grains per Day.
1. Urea,	465·09
2. Uric acid,	1·02
3. Fixed salts, earthy,	23·46
,, alkaline,	214·54
4. Difference,	113·14
Total,	817·25
A. Chlorine,	26·30
B. Phosphoric acid,	47·14

Table G—*continued.*

No. 2.

	Grains per Day.
1. Urea,	677·25
2. Uric acid,	11·88
3. Fixed salts, earthy,	22·68
,, alkaline,	221·94
4. Difference,	131·94
Total,	1065·69
A. Chlorine,	49·42
B. Phosphoric acid,	43·28

No. 3.

1. Urea,	644·62
2. Uric acid,	1·04
3. Fixed salts, earthy,	11·96
,, alkaline,	222·04
4. Difference,	118·89
Total,	998·55
A. Chlorine,	12·70
B. Phosphoric acid,	40·78

No. 4.

1. Urea,	554·00
2. Uric acid,	7·40
3. Fixed salts, earthy,	15·00
,, alkaline,	145·00
4. Difference,	96·60
Total,	818·00
A. Chlorine,	40·00
B. Phosphoric acid,	38·10

No. 5.

1. Urea,	630·00
2. Uric acid,	5·29
3. Fixed salts,	405·00
4. Difference,	289·91
Total,	1330·20
A. Chlorine,	79·20
B. Phosphoric acid,	23·72

Table G—*continued.*

No. 6.

	Grains per Day.
1. Urea,	484·30
2. Uric acid,	0·71
3. Fixed salts,	297·25
4. Difference,	268·99
Total,	1051·25
A. Chlorine,	36·08
B. Phosphoric acid,	29·43

Table H.—*Well-fed Vegetarians.*

No. 1.

1. Urea,	367·50
2. Uric acid,	0·50
3. Fixed salts, earthy,	20·30
,, alkaline,	364·00
4. Difference,	260·60
Total,	1012·90
A. Chlorine,	115·90
B. Phosphoric acid,	30·00

No. 2.

1. Urea,	578·81
2. Uric acid,	0·71
3. Fixed salts,	421·20
4. Difference,	236·15
Total,	1236·87
A. Sulphuric acid,	40·65
B. Phosphoric acid, with earths,	5·92
,, with alkalies,	26·55

No. 3.

1. Urea,	315·00
2. Uric acid,	1·69
3. Fixed salts,	261·00
4. Difference,	134·81
Total,	712·50
A. Sulphuric acid,	18·88
B. Phosphoric acid,	22·78

Table H—*continued.*

No. 4.

	Grains per Day.
1. Urea,	366·12
2. Uric acid,	2·48
3. Fixed salts,	249·20
4. Difference,	99·28
Total,	717·08
A. Sulphuric acid,	23·50
B. Phosphoric acid, with earths,	6·30
,, with alkalies,	21·24

No. 5.

1. Urea,	342·55
2. Uric acid [and Hippuric],	3·04
3. Fixed salts,	252·30
4. Difference,	177·71
Total,	775·60
A. Sulphuric acid,	21·00
B. Phosphoric acid, with earths,	3·46
,, with alkalies,	17·34

Part IV.—*Comparison of the foregoing with the daily work, bodily and mental, and with the daily food of Man.*

Before deducing any inferences from the excretion of urea, uric acid, and phosphoric acid, established in the three preceding parts of this paper, a preliminary inquiry suggests itself, which, if decided in the negative, would go far to render any inferences deduced from the urine of but secondary value.

I take for granted that the substances excreted by the urine of a healthy man result from the wear and tear of tissue of every kind in the body; and that, in a state of health, this excretion is exactly compensated for by the assimilation of an equal amount of the same substances ingested.

This hypothesis would be readily granted, if it included all the *excreta*—per cutem, per halitum, per anum, per vesicam, provided the *ingesta* also included all received by the stomach, lungs (and skin?)—but, in restricting my hypothesis to the urine, I was aware that I differed from many physiologists, and that the *onus probandi* rested upon me. I, accordingly,

undertook a number of observations on the food taken by the persons experimented on in the preceding part of this paper, particularly with reference to the nitrogen received in their food compared with the nitrogen excreted by the urine. The results of these observations I shall now detail, previously to deducing any inferences from the excretions of the urine. I determined the water lost by drying each article of food at 212° F., and afterwards analyzed the dried product for nitrogen, by burning in a tube with soda lime, and collecting the ammonia in muriatic acid. The quantity of nitrogen was found by adding bichloride of platinum, and afterwards weighing the double chloride of platinum and ammonium on a weighed filter; or by the weight of platinum after ignition; or by both methods. I always found, when the ignition of the double chloride was conducted slowly, that the results were identical with those given by the direct weighing of the ammoniaco-platino-chloride.

Experiments on Food.

1. *Lean roasted Mutton;* leg; 5 days dead before cooking, 2 days cooked—
   114 grs., dried at 212° F., gave 48·10 grs.
   Solids = 48·19 per cent.

2. *Lean roasted Mutton;* leg, nearer the shank end; more fat and fascia than in No. 1; 5 days killed, 1 day cooked—
   89 grs., dried at 212°, gave 28·10 grs.
   18·10 grs. of the latter, burned with soda lime, gave of platinum 12·70 grs.
   Solids = 31·57 per cent.
   Nitrogen = 10·02 „

3. *Lean roasted Mutton;* leg, nearer the loin; very little fat or fascia; 5 days killed, 1 day cooked.
   123·75 grs., dried at 212° F., gave 62·80 grs.
   13·30 grs. of the latter, burned in tube, gave of platinum 11·70 grs.
   Solids = 50·75 per cent.
   Nitrogen = 12·56 „

4. *Lean raw Mutton;* loin; 5 days killed.
   118·75 grs., dried at 212° F., gave 38·90 grs.
   14·65 grs. of the latter gave of platinum 11·53 grs.
   Solids = 32·76 per cent.
   Nitrogen = 11·21 „

5. *Lean roasted Beef;* sirloin; 5 days killed, 1 day roasted.
   98·12 grs., dried at 212° F., gave 37·45 grs.
   14·97 grs. of the latter gave of am. plat. chloride, 30·15 grs.
   Solids = 38·16 per cent.
   Nitrogen = 12·64 „

6. *Lean raw Beef;* sirloin; 7 days killed.
   164·93 grs., dried at 212° F., gave 50·75 grs.
   13·80 grs. of the latter gave of am. plat. chloride, 28·13 grs.
   Solids = 30·77 per cent.
   Nitrogen = 12·80 „

[The following experiments are useful, as showing the proportion of the water and fat of lean beef and mutton lost by roasting.

A. *Lean raw Beef;* lap; 5 days killed, weighing 2339 grs. became by roasting (1 day cold) 1573·75 grs., and on the second day 1551·45 grs.
   Roasted solids (1st day) = 67·28 per cent.
   „ (2nd day) = 66·33 „

B. *Lean raw Mutton;* loin; 5 days killed, and weighing 2287·3 grs., became by roasting (one day cold) 1368·3 grs., and on second day 1345·0 grs.
   Roasted solids (1st day) = 59·82 per cent.
   „ (2nd day) = 51·83 „]

7. *Best white Bread;* 2 days old; white wheat.
   106 grs., dried at 212° F., gave 65·20 grs.
   20·90 grs. of the latter gave of platinum 3·10 grs.
   Solids = 61·51 per cent.
   Nitrogen = 2·12 „

8. *Prison white Bread;* second quality, probably made from red wheat; 2 days old.
   52·25 grs., dried at 212°, gave 32·05 grs.
   18·10 grs. of the latter gave of am. plat. chloride, 6·40 grs., and of platinum 2·80 grs.
   Solids = 61·34 per cent.
   Nitrogen = 2·21 „

9. *Prison Oatmeal.*
   94·01 grs., dried at 212° F., gave 86·53 grs.
   16·85 grs. of the latter gave of am. plat. chloride 7·30 grs., and of platinum 3·30 grs.
   Solids = 92·03 per cent.
   Nitrogen = 3·04 „

10. *Prison Indian Meal.*
    96·22 grs., dried at 212° F., gave 82·55 grs.
    15·07 grs. of the latter gave of platinum 1·65 grs.
    Solids = 85·20 per cent.
    Nitrogen = 1·56 „

11. *Boiled Cauliflower;* 1 day cooked.
203·18 grs., dried at 212° F., gave 22·25 grs.
20·33 grs. of the latter gave of am. plat. chloride 11·40 grs.
Solids = 10·95 per cent.
Nitrogen = 3·50 „

12. *Prison Milk.* Dr. Apjohn favoured me with the results of his own experiments, made by direct analysis on two specimens of the milk supplied usually to the Dublin market, and on a specimen procured direct from the cow. From these carefully conducted experiments it would appear that, if the adulteration of milk be merely water, the specific gravity affords a direct measure of the nitrogen present. The nitrogen was determined by him in the same manner as by myself, viz., by the corresponding weight of platinum procured.

No. 1, Sp. gr. 1025; Platinum per 1000 grs. 21·80 grs.
No. 2, „ 1027; „ 23·62 „
No. 3 (pure) „ 1035; „ 32·48 „

The prison milk examined by me had a specific gravity of 1024·9, and also contained 177 grs. (equivalent to glucose) of sugar per pint.

Collecting together the preceding data, the following Tables may be constructed, showing respectively the quantity of water and of nitrogen in the several articles of food experimented upon.

Table J.—*Quantity of Water and Solid Matter in Articles of Food.*

No.	Food.	Solids per cent.	Water per cent.
1.	Mutton (roast), . .	42·19	57.81
„	„ „	31·57	68·43
„	„ „	50·75	49·25
2.	Mutton (raw), . . .	32·76	67·24
3.	Beef (roast), . . .	38·16	61·84
4.	Beef (raw), ...	30·77	69·23
5.	Bread (1st quality), .	61·51	38·49
6.	Bread (2nd quality) .	61·34	38·66
7.	Oatmeal, ...	92·03	7·97
8.	Indian Meal, . . .	85·20	14·80
9.	Cauliflower, . . .	10·95	89·05

The following remarks may be made on this Table:—

The per-centage of water in roast mutton varies much more than it does in roast beef, probably on account of the more variable proportion of fat distributed in the cellular matter between the muscular fibres.

The average of the three specimens of roast mutton [solids, 41·50 per cent.; water, 58·50] comes very near that found for roast beef.

The close agreement between the two specimens of bread is what might be expected from the facts recorded by chemists respecting the "water of panification."

Table K.—*Per-centage of Nitrogen found in Articles of Food, previously dried at* 212° F. (except the milk).

No.	Food.	Nitrogen per cent.	No.	Food.	Nitrogen per cent.
1.	Mutton (roast), .	10·02	7.	Bread (2nd quality),	2·21
2.	„ „	12·56	8.	Oatmeal, . . .	3·04
3.	Mutton (raw), . .	11·21	9.	Indian Meal, . .	1·56
4.	Beef (roast), . .	12·64	10.	Cauliflower, . .	3·50
					per 1000 grs.
5.	Beef (raw), . . .	12·80	11.	Milk (sp. gr. 1035),	4·64
6.	Bread (1st quality),	2·12	12.	Milk (sp. gr. 1025),	3·11

The specimens of mutton (Nos. 1, 2) gave of solids 31·57 and 50·75 per cent., respectively; from which, and other similar facts, I infer that the mutton that contains least water and fat is not only the best for that reason, but that also its muscular fibre contains more nitrogen when dried. The poor fatty mutton, 2 or 2½ years old, with pale muscle, that abounds in the Dublin markets, is the result of our so-called improved breeding of sheep, possessed of high fattening qualities, but is very inferior to the dark-fibred 3 or 3½ year old mutton, now, alas! almost extinct; this inferiority depends on a double cause, the excess of water, and the deficiency of nitrogen. The difference in the nitrogen contained in the two qualities of bread, though slight, is, I believe, real, as it is probable that there is a greater quantity of gluten in our red than in our white wheats.

It is now necessary, by the aid of the preceding Tables, to calculate the quantity of nitrogen, or of its equivalent urea, taken as food by the subjects of Tables A and B; I am able to do so in the case of No. 1 of Table A, and of Nos. 2, 3, 4, 5, of Table B. The food used daily by No. 1, Table A, during some time preceding, and during the experiments, was as follows:—

I. Roast beef (cold), . . 8 oz.
II. White bread, . . . 8 oz.
III. Boiled cauliflowers, . 10 oz.
IV. Milk (sp. gr. 1025), . 1 pint.

By the aid of the chain rule, which is admirably adapted to such calculations, I find the equivalent of these four articles of diet in grains of urea, as follows:—

I.
8 oz. cold roast beef.
16 : 7000 grs. „ „
10000 : 3816 grs. dried at 212°.
10000 : 1264 grs. nitrogen.
28 : 60 grs. urea.
362 grs.

II.
8 oz. white bread.
16 : 7000 grs. „
10000 : 6151 grs. dried at 212°.
10000 : 212 grs. nitrogen.
28 : 60 grs. urea.
98 grs.

III.
10 oz. boiled cauliflower.
16 : 7000 grs. „
10000 : 1095 grs. at 212° F.
1000 : 35 grs. nitrogen.
28 : 60 grs. urea.
36 grs.

IV.
1 pint milk.
8 : 70000 grs.
10000 : 218 grs. platinum.
28 : 14 grs. nitrogen.
28 : 60 grs. urea.
58 grs.

Adding together the preceding results, we find—
I. Cold roast beef, 8 oz., equivalent to 362 grs. urea.
II. White bread, 8 oz., „ 98 „ „
III. Boiled cauliflower, 10 oz., „ 36 „ „
IV. One pint of milk, „ 58 „ „

Total equivalent of urea ingested, **554** grs.

Let us now compare this quantity of urea with that excreted *per vesicam* and *per anum*.

The fæces passed per day amounted to 5 oz., the analysis of which gave the following results:—

305·45 grs., dried at 212° F., gave 80·05 grs.
28·15 grs. of the latter gave of am. plat. chloride, 30·75 grs.
From these data I find—

Solids = 26·21 per cent.
Nitrogen = 6·86 „

From this we obtain the total urea equivalent to the nitrogen thus excreted.

5 oz. fæces.
16 : 7000 grs.
10000 : 2621 grs. at 212° F.
10000 : 686 grs. nitrogen.
28 : 60 grs. urea.
84·28 grs.

Combining this result with that given in Table A, we find—

1. Urea, excreted *per vesicam* = 465·09 grs.
2. Equivalent of urea, excreted *per anum*, . . = 84·28 grs.

Total, ... **549·37**

The close agreement of this result with that obtained from an analysis of the food entitles me to assume that the nitrogen used in the body is naturally excreted by the kidneys, and that the surplus taken in food, and not used or required by the body is thrown out as a mere excretion *per anum*.

I am aware that this result differs widely from the statements made in Carpenter's Physiology, and other books, on the authority of Barral and other experimenters, who divide the nitrogen excreted from the body into two nearly equal portions, one of which is discharged *per vesicam et anum*, and the other *per cutem et halitum*.

M. Barral's results are as follow:—

Subject.	Nitrogen in food.	Nitrogen excreted.		
		Per vesicam.	Per anum.	Per cutem et halitum.
M. Barral, æt. 29, in summer wt. 105 lbs.	432·3 grs.	168·3 grs.	42·2 grs.	220·8 grs.
M. Barral, in winter,	327·3 „	151·3 „	20·1 „	155·9 „
Boy (æt. 6, wt. 33 lbs.)	121·9 „	47·8 „	27·8 „	46·3 „
Man (æt. 59, weight, 129 lbs., ...	421·5 „	234·6 „	38·6 „	148·3 „
Unmarried Woman, æt. 32, wt. 135 lbs.,	345·8 „	154·4 „	12·3 „	179·1 „

I believe that the results of this Table are in error, both by underrating the nitrogen passed in the urine, a mistake commonly committed in estimating urea, and also by overrating

the nitrogen taken in food, as I certainly find some difficulty in understanding how M. Barral, only weighing 105 lbs., could possibly consume the equivalent of 926 grs. of urea per day. If any doubt rest on the actual facts as to the nitrogen in the food and urine, of course the result as to the skin and lungs becomes worthless, as it is found simply by difference. I now turn to the case of Nos. 2, 3, 4, 5, of Table B, for which I have accurate analyses of the food and urine, but not of the fæces.

These subjects of experiment, being prisoners in the Military Prison in Dublin, of course could have no access to any food except that allowed by the regulations. I am indebted to my friend, Mr. Tufnell, to whom I take this opportunity of acknowledging my obligations, for the facilities which I experienced in making my observations on this class of subjects.

The food allowed by the prison regulations is the following:—

I. Oatmeal, . . . 8 oz.
II. Indian Meal, . . 9 oz.
III. Bread, ... 8 oz.
IV. Milk, ... 1½ pints.

The preceding quantities of food, converted, as before, into their equivalents of urea, give the following results:—

I.
8 oz. oatmeal.
16 — 7000 grs. „
10000 — 9203 grs. at 212° F.
10000 — 304 grs. nitrogen.
28 — 60 grs. urea.

210·5 grs.

II.
9 oz. Indian meal.
16 — 7000 grs. „
1000 — 852 grs. at 212° F.
10000 — 156 grs. nitrogen.
28 — 60 grs. urea.

112 grs.

III.
8 oz. prison bread.
16 — 7000 grs. „
10000 — 6124 grs. at 212° F.
10000 — 221 grs. nitrogen.
28 — 60 grs. urea.

101·6 grs.

The milk used in the prison had a specific gravity of 1024·9, and contained 177 grs. of sugar equivalent to glucose per pint. I therefore adopt for it the same result as that before used, viz., one pint equivalent to 58 grs. of urea.

Combining the preceding results, I find the total food used per day to be equivalent to the following quantity of urea:—

I. Oatmeal, 8 oz., . . 210·5 grs. urea.
II. Indian meal, 9 oz., . 112·0 „ „
III. Bread, 8 oz., . . . 101·6 „ „
IV. Milk, 1½ pints, . . 87·0 „ „

Total, . . . **511·1** grs. urea.

From Table B, it appears that the excreta, estimated as urea, were as follows:—

I. Excreta *per vesicam*, . 400·62 grs. urea.
II. Excreta *aliter*, . . . 110·48 „ „

Total, . . . 511·10

I have no doubt but that the 110 grs., equivalent to 51·6 grs. of nitrogen, were discharged as surplus *per anum*, and that the lungs and skin had nothing to do with their elimination.

Having re-established the fact, that the kidneys are the sole excreting organ intended to be used in health for the elimination of nitrogen from the body, and that the nitrogen so excreted has done its work, it remains for me to show the connexion between the work done and the nitrogen so excreted.

I have already divided the work done into four different heads:—

a. Opus vitale.
b. Opus calorificum.
c. Opus mechanicum.
d. Opus mentale.

Of these, the first two, properly speaking, ought to be considered together, as they are both necessary to the life and health of the individual; but as the second is fully accounted for by the excretion of carbonic acid from the lungs, and in no way relates to the present subject, I shall omit its consideration for the present, and confine myself to the three other descriptions of work. Before doing so, it is necessary to estimate, in the usual manner, the amount of mechanical work done, and then to determine the number of grains of urea to which it is equivalent.

Let us turn first to Table B.

The habits of the subjects of experiment are as follows:—

No. 1, wt. 173 lbs., walks 3 miles per day.
No. 2, 3, 4, 5. Average weight 141 lbs.

1. Shot drill, . . . 3 hours.
2. Ordinary drill, . 1½ hours.
3. Oakum-picking, . 3½ hours.

1. To estimate the work done by a person of a given weight walking, it is only necessary to remember that the effort of walking is equivalent to that of lifting one-twentieth part of the weight of the body through the distance walked; this fact I have established by many observations. We therefore obtain the following formula—

$$\left.\begin{matrix}\text{Work done in walking estimated in tons}\\ \text{lifted through a foot,}\end{matrix}\right\} = \frac{5280 \times w \times n}{20 \times 2240} \qquad (4)$$

w and n denoting the weight in lbs., and the distance walked in miles.

2. The work done in shot drill is somewhat more difficult of estimation. From careful observation of the prisoners engaged in this highly uninteresting occupation, I obtained the following results:—

Each man lifts a 32 lb. shot to his breast (3 ft.) from a tressel, carries it through 9 ft. by measurement (4 paces by drill); and lays it down on a similar support; he then returns unloaded, and takes up another shot, and so repeats the double journey; of course, after a certain time, all the 32 lb. shots are transferred from one side to the other of the working gang, and they must then reverse the order of proceeding, and carry back the shots. Six of these double journeys occupy one minute.

Now, as it can be proved that equal work is done in lifting and laying down the shot, we readily obtain the following expression for the work done in the three hours—

$$\text{Work in tons} = \left(\frac{(2w + 32)\,a}{20 \times 2240} + \frac{32 \times 2h}{2240}\right) \times n \times 180^m, \qquad (5)$$

where

w = weight of man in pounds.
a = distance in feet to which the 32 lb. shot is carried.
h = height in feet to which it is lifted.
n = number of double journeys per minute.

Substituting for these their values, we find—

Work of three hours' shot drill in tons lifted through one foot

$$= \left(\frac{(282\text{lbs.} + 32\text{lbs.})\,9\text{ft.}}{20 \times 2240} + \frac{32\text{lbs.} \times 6\text{ft.}}{2240}\right) \times 6 \times 180 = \mathbf{160{\cdot}7}\text{ tons.}$$

3. *Oakum-picking.*—If we estimate the mechanical labour of this occupation as less than half that of walking, it will be safe to consider $3\frac{1}{2}$ hours of oakum-picking as equivalent to 5 miles' walking; this estimate I adopt for the present.

From the preceding considerations, the following estimate of the work done in actual mechanical labour by the subjects of experiment of Table B is easily deduced.

No. 1. Work done in walking = 61·1 tons lifted through one foot.

This result is obtained from equation (4).

Nos. 2, 3, 4, 5. (I assume common drill to be the same labour as walking.)

Work done:—

1.	Shot drill, ...	160·7	tons lifted one foot.
2.	Common drill (4 miles),	66·5	,, ,,
3.	Oakum-picking (5 miles),	83·1	,, ,,
		310·3	

All the subjects of Table B may be considered as simply expending force in the act of living, and in working or walking about. From these considerations, I find the following equations:—

$$\begin{aligned} 400 &= Aw + B. \\ 367 &= Aw' + B. \end{aligned} \qquad (6)$$

In these equations,

400 grs. of urea represent the total work of the Military Prisoners, Nos. 2, 3, 4, 5.

367 grs. of urea represent the total work of No. 1.

w, w', are the weights of the subjects respectively, viz., 141 lbs. and 173 lbs., referred to 150 lbs., as the weight of the standard man.

A denotes the daily discharge of urea in grains requisite to keep the standard man (150 lbs.) alive.

B, B', denote the urea in grains that are equivalent to the mechanical work done by Nos. 2, 3, 4, 5, and No. 1, respectively.

From the estimate of the work already given, we obtain the following additional equation—

$$61\,B = 310\,B'. \qquad (7)$$

Combining equations (6) and (7), we find—

$$\begin{aligned} 400 \times 150 &= A \times 141 + B. \\ 367 \times 150 &= A \times 173 + B. \\ 61\,B &= 310\,B'. \end{aligned} \qquad (8)$$

Solving for A,

$$A = \frac{310 \times 367 - 61 \times 400}{310 \times 173 - 61 \times 141} \times 150$$

$$= \mathbf{297{\cdot}7}\text{ grs. urea.}$$

This is the excretion of urea requisite to keep 150 lbs. wt. of man alive for four and twenty hours.

In round numbers, it may be called 300 grs.; from which it follows that each pound weight of living man requires an expenditure of work, represented by 2 grs. of urea, per day to keep it alive, and prevent it from becoming subject to the ordinary chemical laws of inert matter.

Substituting in equations (8) for A its value just found, we obtain—

$$400 = 297{\cdot}7 \times \frac{141}{150} + B,$$

$$367{\cdot}5 = 297{\cdot}7 \times \frac{173}{150} + B.$$

From which it is easy to deduce

$$B = 120 \text{ grs.}$$
$$B = 24{\cdot}2 \text{ grs.}$$

These numbers express the grains of urea, equivalent to the mechanical work done by Nos. 2, 3, &c., and No. 1, respectively; but the work done is 61 and 310 tons lifted through one foot, respectively; from which it is easy to calculate β, the number of grains of urea requisite to lift 100 tons through one foot, viz.:—

$$\beta = \frac{120 \times 100}{310} = 38{\cdot}71 \text{ grs.},$$

and

$$\beta = \frac{24{\cdot}2 \times 100}{61} = 38{\cdot}67 \text{ grs.}$$

Taking the mean of which, I find—

$$\beta = 38{\cdot}69 \text{ grs. of urea.}$$

I have already found that it requires 297·7 grs. of urea to keep 150 lbs. of man alive for 24 hours; the actual value of this vital work may now be calculated, by dividing 297·7 by β, the coefficient in urea of mechanical work—

$$\text{Opus vitale} = \frac{297{\cdot}7 \times 100 \text{ tons}}{38{\cdot}69} = 769{\cdot}45 \text{ tons lifted one foot.}$$

This enormous force is requisite to keep 150 lbs. of man living during 24 hours, and yet it is not all that is required; for, in addition, there is the *Opus calorificum*, or work necessary to keep the skin at a constant temperature of 90° F. The amount of this latter work will vary with climate and season, and appears to depend altogether on the excretion of carbonic acid, and not on that of urea.

No chemist who reflects on the number and power of the chemical affinities that are controlled and kept from acting by the vital force during life, will feel disposed to question the enormous energy which my results tend to attribute to it.

Let us examine these results from another point of view. I have estimated the work done by the prisoners at 310 tons lifted one foot. This is a large amount of work, perhaps as large as can ever be obtained in forced labour, but it falls below what is observed in free labour. I take as an example the paviours, Welsh and Irish, employed in this city, on whom I made careful observations last October, and found the following results:—

Weight of rammer, . . .	5 st. 9 lbs.
Blows of rammer, ...	78 in 2^m 45^s.
Rest, ...	3^m 30^s.
Height lifted, ...	16 in.
Hours of labour, ...	10 hours.

From these data, it is easy to calculate that the work is equivalent to 352 tons lifted one foot per day; to which must be added the work done in walking to and from the work, and while at work; this is equivalent to one-twentieth of the distance walked, multiplied by the weight of the labourer. The whole mechanical work is, in all probability, less than 400 tons lifted one foot per day. According to the observations of Coulomb, Lamandé, and others, French workmen are not usually capable of this amount of labour, although it is sometimes approached. I select the following instances:—

1. Coulomb estimates the work done per day in pile-driving at 75240 kilogram-metres = 242 tons lifted one foot.

2. Lamandé found the work done in pile-driving, at the building of the bridge of Jena, to be
80635 kilogram-metres = 260 tons lifted one foot.

3. Coulomb determined the daily work of men employed turning a lever at 115920 kilogram-metres
= 374 tons lifted one foot.

4. Coulomb found, on questioning a number of pedlars, that they stated that on an average they carried 44 kilos, 19,000 metres per day. Supposing each pedlar to weigh 55 kilos, this would give 1881000 kilogram-metres horizontally. The one-twentieth part of this is the weight lifted
= 94050 kilogram-metres.
= 303 tons lifted one foot.

The low amount of work done in the case of pile-driving

appears to be partly due to the rapid character of the work, and its consequent exhausting effect.

Let us take the work done by the paviours as the average work of well-fed and well-paid labourers, working freely; and, supposing each man to weigh 150 lbs., and calculate how high this work would lift his body in the 24 hours. We find, calling x this height in miles—

$$\frac{x \times 150 \text{ lbs.}}{2240} = \frac{352 \text{ tons}}{5280 \text{ ft.}};$$

or,

$$x = 1{\cdot}005 \text{ miles.}$$

This result is confirmed by the consideration that 20 or 21 miles per day, with rest on Sundays, is the utmost that a skilled pedestrian would undertake as a constant performance, carrying no weights except his clothes. Since the coefficient of traction is 1-20th or 1-21th, this would be equivalent to lifting his body through one mile per day.

This mode of considering the result is independent of the weight of the agent, and will be found useful in almost every kind of labour, as the work done is generally proportional to the weight of the labourer; and it is easily remembered that a man could lift his own body through one mile by the utmost daily labour he is capable of. Let us now consider the height to which the work that keeps a man's body alive for 24 hours is capable of lifting that same body. The vital work on 150 lbs. is measured by 297·7 grs. urea, and 38·69 grs. of urea are equal to 100 tons lifted one foot, from which I find the following chain:—

	1 lb. living man.
150	297·7 grs. urea.
38·69	100 ton-feet.
1	2240 lb. feet.
5280	1 lb. mile.
	2·18 miles.

Therefore, the vital force that keeps a man of any weight alive for 24 hours, is capable of lifting his body through 2·18 miles in the same time.

I may add, as a confirmation of the estimate of vital work (297·7 grs. urea to 150 lbs.), that I tried some experiments on convalescent hospital patients, who took little or no exercise, and were well fed, and that I found 300 grs. urea to be the daily excretion of men weighing 11 stone, or 154 lbs.

It remains now to determine, if possible, the *Opus mentale*, and to give its coefficient in waste of tissue, or discharge of urea. For this purpose, I constructed the following Table:—

Table L.—*Work done, expressed in Urea Grains, and divided into its component parts.*

No.	Total Urea.	Opus vitale.	Opus mechanicum.	Opus mentale, &c. &c.
	grs.	grs.	grs.	grs.
Table B, No, 1, . . .	367·5	343·3	24·2	–
,, Nos. 2, 3, 4, 5,	400·6	279·8	120·8	–
Table A, No. 1, . . .	465·0	250·1	28·7	186·2
,, No. 2, . . .	677·2	250·1	57·4	369·7
,, No. 3, . . .	644·6	250·1	57·4	337·1
,, No. 4, . . .	554·0	345·3	15·8	192·9
,, No. 5, . . .	630·0	375·1	34·5	220·4
,, No. 6, . . .	484·3	287·8	33·0	163·5

In this Table I have calculated the grains of urea corresponding to the vital work from the expression—

$$\textit{Opus vitale} = \frac{297{\cdot}7 \times w}{150}, \qquad (9)$$

where w denotes the weight in pounds.

I have calculated the mechanical work done by the following equation deduced from (5),

$$\textit{Opus mechanicum} = \frac{5280 \times w \times n \times 38{\cdot}69 \text{ grs.}}{20 \times 2240 \times 100}, \qquad (10)$$

n denoting the number of miles walked.

On making this calculation, I have assumed the partridge-shooting of No. 2, and athletic games of No. 3, as each equivalent to 10 miles' walking. The column headed *Opus mentale*, &c., is found by difference, and has now to be discussed.

In the first place, I take Nos. 1, 5, 6, whose mental occupation was almost identical, consisting of high class teaching, and severe study preparatory to it (correcting the statement appended to No. 6, in Table A, as I find on inquiry, that the two hours there mentioned only include the time of preparation, and not the time of teaching, which was also two hours).

If we suppose that mental work, like other work, causes a waste of tissue proportional to the weight of the body, then the coefficient of mental work, or the number of grains of urea that represent one hour's mental work of a man 150 lbs. weight, will be—

$$\textit{Opus mentale} = \frac{150 \text{ lbs.} \times x \text{ grs.}}{n \times w} \qquad (11)$$

where x is the number of grains of urea taken from Table K, n the number of hours' work, and w the weight of the subject in pounds.

From this formula I find—

Coefficient of No. 1 = 44·3 grs. of urea.
" 5 = 43·7 "
" 6 = 42·3 "

Mean, . . 43·4 grs.

The near approximation of these numbers to each other is very remarkable when we consider that the residual grains of urea, from which they are calculated, are 186·2, 220·4, and 163·5, respectively. It certainly proves that the hypothesis I have made, that the mental work sets up a waste of tissue proportional to the mass of the body, is very near the truth.

The kind of mental work in which Nos. 1, 5, and 6, were engaged, was much more severe than that known by the title of "office-work," the coefficient of which in grains of urea may be computed from No. 4, by dividing 192·9 grs. of urea by 6, the number of hours of occupation, and reducing the result in the proportion of 174 lbs. to 150 lbs.

Coefficient of routine mental work for man of 150 lbs. weight } = 27·71 grs. of urea per hour.

Whether we consider this kind of mental labour, or that already discussed, it is plain that they both indicate a waste of tissue of a higher order than that involved in the common *Opus mechanicum*, as may be seen from the following comparison of a day's labour in each.

1. *Opus mechanicum*, or 150 lbs. lifted one mile, } = 136·5 grs. of urea.
2. *Opus mentale*, or 5 hours' study, ... } = 217·0 grs. of urea.
3. *Opus mentale*, or 8 hours' office work, ... } = 221·7 grs. of urea.

It remains to consider the cases Nos. 2 and 3, which are exceptional, and do not fall under the preceding investigation. This is shown by the following Table, which distributes the work of Nos. 2 and 3 under the heads already recognised, and shows a residuum not accounted for.

Table M.—*Distribution of work done, after deducting the Opera Vitalia et Mechanica.*

	Opus mentale.	Residuum.	Total.
Table A, No. 1, .	182·3 grs.	+ 3·9 grs.	186·2 grs.
" No. 2, .	69·5 "	+ 300·2 "	369·7 "
" No. 3, .	182·3 "	+ 154·8 "	337·1 "
" No. 4, .	192·9 "	. . . . .	192·9 "
" No. 5, .	218·7 "	+ 1·7 "	220·4 "
" No. 6, .	167·8 "	− 4·3 "	163·5 "

The large residuum of urea not accounted for in the cases of Nos. 2 and 3, must be attributed to disease or to some state of mind equivalent to disease. I have ascertained, since completing my observations, that No. 2 had passed a small mulberry calculus some months before I experimented on him, a circumstance which excludes his case from a discussion of the constants of healthy urine; yet I believe the main cause of the excessive discharge of urea in his case, and that of No. 3, is to be attributed to a cause which did not escape the observation of the sagacious Prout[a], viz., *anxiety of mind*.

No. 2 was anxious about business matters at the time of my experiments; and No. 3 was at the same time preparing for a competitive examination, the requirements of which exceeded the efforts of the mental constitution with which nature had endowed him; in other words, his mind was overtaxed by the work which he had undertaken, and the consciousness of this caused the anxiety to which I attribute his surplus discharge of urea.

Conclusions deduced from the preceding discussion.

1. The quantity of urea passed per day by men in health varies with their food and occupation, the latter being the principal cause, and regulating the other.

2. Men employed only in manual or routine bodily labour, are sufficiently well fed on vegetable diet, and discharge on an average 400 grs. of urea per day, of which 300 grs. are spent in vital, and 100 grs. in mechanical work. This conclusion is in conformity with the experience of the mass of mankind employed in manual labour in all ages and countries.

[a] Stomach and Urinary Diseases, p. 95. Third Edition. 1840.

3. When the work is of a higher order, a better quality of food must be supplied, sufficient to allow of a discharge of 533 grs. per day of urea, of which 300 grs., as before, are spent in vital work, and 233 grs. in mental work and the mechanical work necessary to keep the body in health.

4. The quantity of urea discharged per day varies also with the weight of the individual, which influences the vital and mental work.

5. The habits, weight, and occupation of the individual enable us to account for a range of the diurnal quantity of urea, varying from 300 to 630 grs. per day; and this discharge may be confidently predicted, when the habits and weight are known.

When, in any case, the discharge of urea exceeds that calculable from the preceding data, it must be attributed to ill health, and most generally to that most fatal of all diseases to which man is liable—anxiety of mind—a vague and unscientific expression, which, however, denotes a most real disease.

This fact alone would render the preceding investigation of importance to the physician, as it enables him, in a given case, to pronounce whether there is an excess of urea or not, and a consequent waste of the system. I have shown that the mere quantity will not decide this question, as from 300 to 630 grs. may be discharged by persons in perfect health, according to their peculiar work and physical conditions.

Note.—The following Table has been published by Dr. William D. Moore, of this city, since the first part of my paper was read, in the Dublin Medical Press of the 27th July, 1859, and reached me too late for insertion in my own paper.

Quantity of Urea eliminated during 24 hours, according to Dr. Warneke.

1.	Adult man (mixed diet), . .	33·7 grms	520 grs.
2.	,, (vegetable diet), .	25·3 ,,	390 ,,
3.	Adult woman (mixed diet), .	26·8 ,,	413 ,,
4.	,, (vegetable diet),	20·1 ,,	310 ,,
5.	Boy (17 years), ...	19·8 ,,	305 ,,
6.	Girl (17 ,,), ...	18·0 ,,	277 ,,

These results, which are the averages of 7 experiments on each individual, are perfectly in accordance with my own results, contained in Tables A and B.

To Professor Parkes
with the Author's Compliments.

THE

HISTOLOGY

OF THE

SUPRA-RENAL CAPSULES.

By GEORGE HARLEY, M.D., F.C.S.
LECTURER ON PRACTICAL PHYSIOLOGY AND HISTOLOGY IN UNIVERSITY COLLEGE, LONDON, ETC.

(*Reprinted from* THE LANCET *of June 5th and 12th*, 1858.)

LONDON:
PRINTED FOR THE AUTHOR, BY
J. KENNY, 414, STRAND.
1858.

THE HISTOLOGY OF THE SUPRA-RENAL CAPSULES.

At the present moment, when the connexion between disease of the supra-renal capsules and bronzed skin is attracting so much attention, I think one need scarcely apologise for bringing under the notice of the profession a contribution to their histology.

Within the last few months several physiologists have attempted to discover the function of the supra-renal capsules, and the relation which might exist between them and the chromatogenous function of the skin, by direct experiment, but without success. The results of my own experiments,* as well as of those that have been published by other observers,† have convinced me that the problem is one more likely to be solved in the dead-house than in the physiological laboratory; and if such be in reality the case, a thorough knowledge of the healthy organs, both as regards their appearance to the naked eye and under the microscope, becomes of primary importance. So little attention has been hitherto bestowed upon the supra-renal capsules, either in the dissecting-room or at post-mortem examinations, that it is much to be feared that occasionally healthy organs are mistaken for diseased, while others but slightly affected are passed by as healthy. Any remarks, therefore, that may tend to remedy this evil can scarcely be looked upon as unimportant.

* British and Foreign Quarterly Review, January and February, 1858.

† Brown-Séquard, Philipeaux, Gratiolet, Vulpain, Martin-Magron, and Virchow.

Supra-renal capsules are found in the bodies of all vertebrated animals. They are developed simultaneously with, but independently of, the kidneys. In the human fœtus at the second month, both organs are of equal volume. From the third month onwards an increasing difference is observed in their relative size.* It has generally been believed that after birth the supra-renal capsules diminish in proportionate bulk and activity of function with advancing years, and consequently they have been denominated fœtal organs. This opinion, however, is now losing ground, since it has been found that they do not become atrophied after birth, and that their growth is not so much even as arrested. In some animals they continue of the same relative size as the kidneys throughout life. In the cat I found that they increased in a greater ratio than the latter organs. In man, however, although they go on increasing in volume after birth, it would appear from the observations of Meckel that the ratio is much smaller than in the case of the kidneys; but it can scarcely be said that even in old age they become proportionally more atrophied than other organs. From these facts we may conclude that the supra-renal capsules have a certain function to perform in adult as well as in fœtal life.

The supra-renal capsules are usually two in number; occasionally, however, supernumerary ones are met with. Very recently I found four in a sheep. The supernumerary were considerably less than half the size of the normal capsules. It has been also mentioned by several authors that they are sometimes altogether absent. Their presence or absence being quite independent of the presence or absence of the kidneys, they would seem to have no direct connexion with the latter organs, for they do not even follow the displacement of them. In brainless monsters they are generally found deficient (Frey).

In the human subject they have a triangular form, which has been very pertinently compared to a cocked-hat.† They are sometimes, however, of an oblong form, or even flattened, and

* Vide Kolliker, Frey, Leydig, &c.

† The left is more triangular in shape than the right capsule; the latter being often crescent-shaped.

spread over the upper and anterior surface of the kidney, like a piece of paper. Dr. Ogle showed a good specimen of the latter kind at a recent meeting of the Pathological Society. Their shape is very various in different species of animals. In some they are almost quite circular, as in the rat; in others they are very much elongated, as in snakes; occasionally they are lobular, and resemble a compressed bunch of grapes, as is seen in the case of the frog.

In man the upper margin of the supra-renal capsule is convex, and about one-eighth of an inch in thickness; the lower margin is concave, and usually about a quarter of an inch thick. The left is generally narrower at its base, longer from above downwards, and larger, than the right. They are on an average about two inches by one in diameter, and, according to Huschke, weigh from 80 to 180 grains. By comparing the relative size of the human supra-renal capsules, as given by Huschke, and of the kidneys by Clendinning,* I find that the weight of the former is to that of the latter as 1 to 18, which I think is much nearer the truth than that given by Meckel—namely, 1 to 28. The relative size of the capsules and kidneys varies very much in different animals. In the common guinea-pig, Frey† gives it as only 1 to 4; while in the seal, Cuvier‡ found it to be no less than 1 to 150.§

Externally the supra-renal capsules have a dirty-yellowish colour, not unlike that of powdered scammony. When cut into, they are seen to be composed of two different coloured portions. The central portion, which constitutes about one-third of the whole thickness of the organ, is of a dark-reddish hue when full of blood; when washed and freed from the latter, of a pale slate colour. In the centre of this, the medullary substance, are several round or oval openings—the mouths of venous sinuses. The cavity, which some authors have described as existing in them, is produced by rupture of the

* Quain's Anatomy, edited by Sharpey and Ellis, p. 316.

† Cyclopædia of Anatomy and Physiology, Part xxxvii. ‡ Ibid.

§ In a specimen kindly sent me from Dublin by Dr. Macdonnell, the relation was as 1 to 188. In the adult mole I find the capsules are to the kidneys as 1 to 48.

tissue between the sinuses, either accidentally during the examination, or as the effect of disease. The medullary substance is bounded on either side by a margin of yellow substance, the internal border of which is of a pale straw, the external of an orange, colour. This, the cortical substance, has a somewhat fibrous appearance, and, although less firm to the touch than the internal medullary part, can scarcely be called friable. (These remarks, of course, only apply to quite fresh and perfectly healthy human capsules.)

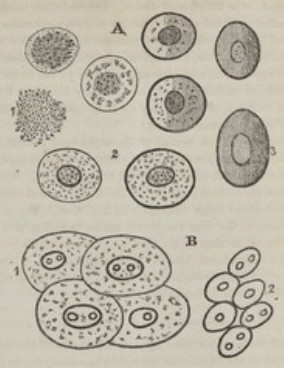
When a transverse section of the supra-renal capsule is examined with a pocket-lens, the medullary substance appears of a somewhat bluish colour, becoming tinged with red as it approaches the cortical portion. In the centre, several openings which were not before visible, come into view. The cortical substance, on the other hand, still retains its yellow hue; but, in addition, is seen to be streaked perpendicularly with red lines; an appearance due, as I shall afterwards have occasion to show, to bloodvessels. The fibrous nature of the cortical substance is readily recognised with the lens.

Microscopical Appearances.

Until within a few years ago, our knowledge of the minute structure of the supra-renal capsules was entirely derived from the results obtained by a microscopical examination of their juice and *débris*. In consequence of this limited method of investigation, the real arrangement of their tissues remained long unknown. And it was not until within a recent date that an observer (Ecker), more zealous than his predecessors, by examining thin sections, discovered that they possessed a minute structure scarcely excelled in beauty by any other organ of the body.

Before describing the appearance of their tissues, I shall first allude to the microscopical characters of their juice. For notwithstanding that the examination of the latter yields no clue to the arrangement of the component parts of the supra-renal body, it nevertheless affords us some interesting information.

Fig. .



From the supra-renal capsule of the frog.—A, 1, Granules; 2, nucleated cells; 3, blood corpuscles. B, 1, Large nucleated cells; 2, free nuclei.

On placing a drop of the expressed juice in the field of the microscope, it is seen to contain an immense number of round bodies of every imaginable size, between that of a large oil globule and the minutest visible speck. At first sight the appearance reminds one of that presented by a drop of new milk. On attentive examination, the smaller of the granules are observed to be in active movement; repelling and attracting each other in rapid succession. Amongst these molecules are seen ordinary oil globules and blood corpuscles. But besides these are a number of round pale-coloured cells, with granular contents. They look not unlike lymph corpuscles, but are

somewhat smaller. They average, in the human subject, $\frac{1}{1\,2\,5}$ mm. ($\frac{1}{5\,0\,0\,0}$ inch) in diameter. In the juice of the supra-renal capsule are also found nucleated cells, filled with molecular particles. These cells occasionally, although not often, contain two nuclei. Besides these, there are nuclei that appear to be surrounded by molecular particles unenclosed in a cell-wall; and others where indistinct marks of a cell-wall are visible; which leads one to suppose that the cell-wall is either in the course of formation, or has been partly destroyed. The fully-developed nucleated cells, as well as the aggregation of granules round nuclei, measure about $\frac{1}{2\,5}$ mm. ($\frac{1}{1\,0\,0\,0}$ inch) in diameter. In some the granules are pale; in others, very dark coloured. The cells, which were previously spoken of as resembling small lymph corpuscles, may possibly be nothing more than free nuclei.

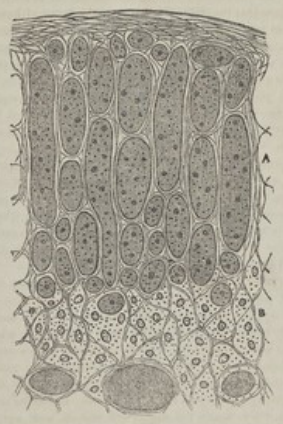
Mr. Gulliver mentioned having found, in many of the ruminants, "the minute molecules less plentiful, their place being supplied by corpuscles somewhat resembling lymph globules in size, but often of a reddish colour, and occasionally of an oval figure."* The small granules and active molecules are particularly abundant in the supra-renal capsule of the rat. The peculiarity of these molecules is that they undergo, as pointed out by Gulliver, no change, when treated with acids or alkalies, until after a considerable lapse of time.

In order to study the structural arrangement of the component parts of the supra-renal capsules, very thin sections of the organs must be examined. Although healthy and perfectly fresh supra-renal capsules admit of being cut into thin slices with the knife, yet it rarely happens that the sections are sufficiently delicate to allow of their structure being well seen. On operating on fresh capsules, I usually obtain the best sections by cutting them with a razor under water. But by far the best method is to harden the capsule with chromic acid, and afterwards render the section transparent with glycerine. Those who have had to contend with the difficulties attending the microscopical examination of supra-renal capsules, will scarcely, I think, regard these remarks as superfluous.

* Gerber's General Anatomy, translated by Gulliver, Appendix, p. 904.

When a transverse section of the human supra-renal capsule is examined with a low power of about eighty diameters, the cortical substance is seen to be composed of numerous cells, arranged in somewhat irregularly-sized rows; the rows of cells appear like a number of dark-yellow columns, placed perpendicularly to the surface. (Fig. 2, A.) The columns terminate

Fig. 2.



Transverse section of a human supra-renal capsule.—A, Cortical. B, Medullary substance. C C, Sinuses.

somewhat abruptly at the margin of the medullary substance. (B.) The latter, when viewed with a low power, such as that

just mentioned, presents no definite structure. Running along the centre of the medullary substance is a line of round or oval openings of various sizes, the mouths of the sinuses. (c c.)

If the cortical substance be examined with a high power of about 400 diameters, the cells constituting the rows will be found to resemble in size and general appearance those lining the tabuli urinifери of the kidney. They differ from the latter very strikingly, however, in their peculiar colour. Each individual cell in the human subject has an average diameter of about $\frac{1}{75}$ mm. ($\frac{1}{1875}$ inch.) Their size varies but slightly. The minimum diameter being about $\frac{1}{75}$ mm. ($\frac{1}{1875}$ inch), the maximum, $\frac{1}{75}$ mm. ($\frac{1}{1875}$ inch.) They consist of a cell wall, granules, pigment, a nucleus, and a greater or less quantity of oil globules. Some authors speak of their peculiar yellow colour being dependent upon the amount of fat globules they contain. I think, however, that the fat globules only contribute to the colour. When floating free in the field of the microscope, the cells appear to be irregularly round; but when grouped together, they have a polygonal shape. Each cell possesses a nucleus, although it is not always visible without the employment of reagents.

The simplest and most beautiful mode of demonstrating the existence of a nucleus, is by colouring the cells with carmine. As this process of imbibition, as it is called, is quite new in England, indeed in any country, it being scarcely a year since it was employed for the first time in Germany,* I may briefly describe it.

"Imbibition" might be termed a process of natural injection. The principle upon which it is founded exists in the fact, that different animal tissues absorb and retain pigments with various degrees of avidity. Thus, for example, while granular tissues possess a special attraction for colouring matters, homogeneous substances display but a feeble affinity for them. And in the tissue composed of aggregated cells, different parts are

* As far as I am aware, Gerlach and Welcker were the first to employ it.—P.S. Since writing the above, I find that, in 1778, Gleichen describes a process of colouring infusoria by carmine.

coloured in various degrees of intensity. Thus the homogeneous cell wall retains its colourless appearance long after its molecular contents have become deeply tinged, and the nucleus, which is usually the most granular part, has assumed a prominent dark hue.

The process of "imbibition" is very simple. A small quantity of pure carmine is dissolved in liquor ammonia. The solution is diluted with water, and thin sections, ready for microscopic examination, are kept in it during twenty-four or forty-eight hours. At the end of this time, the substance will be found thoroughly dyed and fit for use. I may remark, that attention must be paid to avoid having any gritty particles in the colouring solution. I would therefore recommend it to be filtered before the tissues are added. The carmine solution unfortunately putrefies very rapidly, but I find this is easily prevented by adding a few drops of alcohol, which does not impair its efficacy, unless in excess.

On examining some coloured preparations of the brain and spinal cord kindly presented to me by Professors Gerlach and Welcker, I was struck, as everyone must be who examines them, with the remarkable distinctness of the nuclei of the ganglion corpuscles. Thinking that the affinity for colouring matters might be especially characteristic of ganglion cells, and being at the time very anxious to discover a test by which these might be distinguished from the large candate corpuscles occurring in the medullary substance of the supra-renal body, I tried the process of imbibition on a variety of tissues, and soon found that all cells with equally granular contents became tinged in a similar degree. While investigating this point, I was fortunate enough to find the process of imbibition a valuable aid in the examination of the supra-renal bodies. Through its means, it could be shown that all the cells in the cortical columns possessed a large nucleus. The nucleus appeared so very distinct in cells where before the colouring matter was employed not a trace of one could be detected, that I at first thought that the nucleus did not perhaps pre-exist in all the

cells, but was merely the result of the treatment, the ammoniacal solution of carmine having caused the granules distributed throughout the cell to become aggregated together, and thus give rise to the appearance of a nucleus. On examining the cells attentively, however, I was able to satisfy myself that such an explanation was incorrect. The cell still retained its granular appearance, irrespective of the nucleus. While the cell, as I said before, has an average diameter of about $\frac{1}{75}$ mm. ($\frac{1}{1\,9\,0}$ inch), the nucleus is of considerable size, measuring, on an average, $\frac{1}{1\,2\,5}$ mm. ($\frac{1}{3\,2\,5}$ inch) in diameter. The nuclei are generally round and granular.

Arrangement of the Cells in the Cortical Substance.

Considerable difference of opinion exists amongst histologists as to the manner in which the cells are arranged. Mr. Simon thinks that the columnar masses which they form are a series of closed tubes lying perpendicular to the surface of the organ; while Kölliker, Frey, Ecker, and most other observers, describe the cells as being grouped together in a number of oblong vesicles, lying in parallel rows, but having no communication with each other. Mr. Gray, on the other hand, believes that the adjoining walls of the vesicles are sometimes removed by absorption, and thus give rise to tubular cavities. My own opinion regarding the arrangement of the cells may be given in a very few words, as it will be more readily understood by a glance at the woodcuts than by pages of explanation.

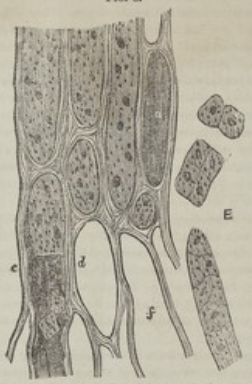
It appears to me that the contrary descriptions given by different histologists are more the result of a variety in language than a difference of opinion. To the same structures the name tubes, sacculi, and vesicles have been applied. This variety of interpretation is easily accounted for. For even in the same section the cells may be seen arranged in the form of tubes, of sacculi, and of round vesicles. Fig. 2 represents somewhat roughly this state of things.

In a good section the cells are seen to have a definite arrange-

ment. They are grouped together, as represented in Fig. 2, in a number of larger and smaller masses. These lie in parallel rows, and give rise to a columnar appearance. Some of the cell-masses are small, and round, not greater than the diameter of a single cell. These are chiefly found at the external and internal extremities of the columns. Others of them are oval, like vesicles, and some are oblong, not at all unlike blind sacculi. On the other hand, occasionally a cell-mass is found to extend almost throughout the whole thickness of the cortical substance, and to have very much the appearance of a tube with closed ends. Notwithstanding that the cell-masses vary so much in length, their breadth remains tolerably uniform, the diameter of the smallest being about $\frac{1}{75}$ mm., ($\frac{1}{1\,9\,0}$ inch,) and of the thickest about $\frac{1}{50}$ mm., ($\frac{1}{1\,2\,5}$ inch.) Each cell-mass, as well as each column is separated from the rest by intervening fibrous tissue. This is represented in Fig. 3. The fibrous tissue surrounding the mass of cells is most distinctly seen when the cells have chanced to fall out, as frequently happens in thin sections. (Fig. 3, *d*, *c*.) On tracing the fibres they are seen to arise from prolongations of the investing fibrous capsule of the organ.

As was already mentioned, the cell-mass may take on the appearance of a tube. In good sections of the human suprarenal capsule it is not uncommon to meet with a single straight row of cells extending throughout the whole breadth of the cortical substance, and it is difficult to divest oneself of the idea that it is a tube. This appearance is represented in Fig. 4. In the specimen from which the figure D was copied, there were two single rows of cells lying parallel to each other. The cells were of a quadrilateral form, and the whole structure reminded me forcibly of two rows of bricks. In the case in question, the intervening space between the rows was about half the breadth of the cells, and it looked exactly like the cavity of a tube bounded on either side by a row of epithelium cells, such as is seen in the tubuli uriniferi of the kidney. (See Fig. 4.)

FIG. 3.



Portion of a transverse section of the cortical substance of a human supra-renal capsule. *a*, Empty lobules, with basement membrane. *d*, The same, without basement membrane. *f*, Fibres. E, Cells fallen out from the loculi in masses.

On one occasion I found a single row of cells extending from the one margin of the cortical substance to the other. It contained upwards of thirty cells. It not unfrequently happens that one meets with a column, partly containing a single, and partly a double, row of cells. Such an one is represented in Fig. 4, E. The walls enclosing the single and the double row of cells are perfectly continuous with each other, showing that the appearance is not due to the accidental union of two cell-masses. Indeed, I have never met with any appearance which would lead me to believe that one cell-mass communicated with

FIG. 4.

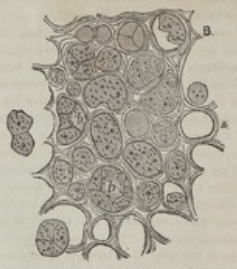


Columnar cell-masses having the appearance of tubes from the human capsule.

another, either directly or by a duct. It sometimes happens that the cells in the single rows are not closely packed together, but are separated by irregularly-sized intervals. This appearance may, however, be due to the process of preparation, as I have not seen it in sections of the fresh capsules. In order to ascertain if the columnar cell-masses, having a close resemblance to tubes, were not actually what they looked like, I made sections of the cortical substance at right angles to the direction of the columns. These transverse sections are very

characteristic. The ends of the columnar cell-masses appear as round or oval spaces filled with cells, as seen in Fig. 5. The

FIG. 5.



Section across the columnar cell-masses. *a*, Empty loculi. A, Loculi completely filled with cells, and having no central opening.

oval or round spaces containing the cells measure from $\frac{1}{175}$ to $\frac{1}{75}$ mm. ($\frac{1}{4450}$ to $\frac{1}{1910}$ inch) in diameter; they usually contain from one to five cells. They are in some parts very closely packed together; at others loosely scattered about, leaving interlocular spaces of various sizes and shapes. Sometimes in thin sections the cells have fallen out, and left empty cavities. (Fig. 5, *a*.) At other times they have only in part fallen out, and those remaining are seen partially detached from the walls of the cavity. In uninjured specimens it is easily seen that the cells completely fill the entire loculi, be they small or be they great, which, it will be remembered, is a very different state of things from what is found in true tubes. When, for example, a transverse section of the tubuli uriniferi is examined, the ends of the tubes are not found to vary greatly in diameter in the same part of the organ. They are never so small as to

contain but one cell, as in the case of the supra-renal capsules, neither are they ever completely filled with cells. There is invariably a free space in the interior of each tube, and around this central cavity are arranged the epithelial cells. Further, the cells are only one row deep, and regularly ranged in a circle upon the membrana propria. As the columns of the cortical substance of the supra-renal capsules, when seen in transverse section, present none of these characters, I do not think that they deserve the name of tubes. (See Fig. 5.)

In describing the cavities containing the cell-masses, Kölliker speaks of them as mere loculi in the stroma of the organ, possessing no limitary membrane. With all due deference to the opinion of my former teacher, which I have much cause highly to appreciate, I must acknowledge that I think Ecker and Frey are correct in stating that the loculi are lined with a homogeneous membrane. I have been able to demonstrate it in the longitudinal section of the cortical substance by carefully washing away the cells. The basement membrane, although exceedingly delicate, is, however, distinctly visible, especially at those points where, when accidentally torn, it is thrown into folds. After the preparation has been kept during some weeks in glycerine, the membrane, which at first appeared homogeneous, becomes granular. The basement membrane is represented as lining the loculi in Fig. 3, *a* and *c*, which contrasts strongly with some of the other loculi from which the membrane has been removed in making the section, and which appear as empty holes. This condition is seen at *d* in Fig. 3.

The cells sometimes fall out of the loculi all in a mass, and they then appear as if they were held together by an invisible investing substance. There is no special connecting substance, however, for they are readily separated by the needles. (Fig. 3, x.)

Medullary Substance of the Supra-renal Capsules.

The dark, slate-coloured, medullary substance, when examined with the microscope, is seen to possess a very different structural arrangement from that of the cortical portion of the

organ. When viewed with transmitted light, it looks much paler in colour than the latter, and is quite free from any columnar cell-masses. The numerous openings of its sinuses give to it a very characteristic appearance. With a low magnifying power, no very definite idea of the arrangement of its component parts can be obtained. It looks merely like a uniform mass of cells, bounded on either side by the cortical columns. With a high power of about 400 diameters, it is seen to be composed of a reticulated fibrous structure, in the meshes of which lie a number of large, pale-coloured cells with round nuclei. The cells measure, on an average, $\frac{1}{77}$ mm. ($\frac{1}{1777}$ inch), the nuclei $\frac{1}{215}$ mm. ($\frac{1}{7777}$ inch), in diameter. Although at first sight these large cells appear to be irregularly distributed throughout the network of fibres, on the close inspection of a favourable specimen they are seen to follow a somewhat symmetrical arrangement, being singly or in small groups, surrounded by fibres. No basement membrane is here visible, nor can the cells be easily detached from the fibres. (Fig. 2, *b*.)

The cells of the medullary substance have been described by Kölliker and Brown-Séquard as resembling ganglion corpuscles; and certainly, when seen lying in clusters amongst the fibres, they have some similarity to them. For example, they have about the same diameter, they have a well-marked nucleus, and they appear in some cases caudate. When seen singly, however, they look much more like secreting cells. I have never seen a detached one have a caudate appearance, and I am inclined to believe it is the peculiar arrangement of the reticulated fibres which gives the cells that appearance. In the sheep, these ganglion-corpuscle-shaped cells are remarkably plain. Some have two nuclei, and also nucleoli. Both the nuclei and the cells are filled with granular contents. This is best seen in the coloured specimens. Ecker, in describing the medullary substance, speaks of the nuclei as being surrounded by granular matter, but omits all mention of a distinct cell-wall.*

* Ecker on the Supra-Renal Capsules. Translation in the *Medical Gazette*, 1848.

While examining fresh human supra-renal capsules, I frequently scratched out some of the nuclei from the reticulated fibres, and they had in most cases exactly the appearance spoken of by Ecker. The nucleus lay in the midst of granular flocculi, without any vestige of an enclosing envelope. When the meshes of the fibres were slowly torn across, however, the margin of the granular flocculi in some cases was so distinct, that one could scarcely doubt the existence of a cell-wall. This point requires further investigation. The general appearance of the medullary fibres is given in fig. 2, *b*.

Virchow mentions having seen true ganglion corpuscles in the human capsules; but adds that he does not look upon them as composing the medullary substance of the organ, but merely attached to the nerves.* In describing the supra-renal capsules of the land salamander, Leydig states that one part of them is almost entirely composed of dirty-yellow ganglion corpuscles; and that these, through a gradual change in their contents, go directly over into the fatty granular cells proper to the supra-renal body.

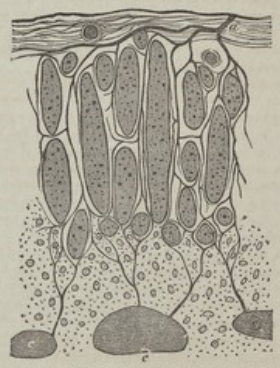
Vessels.—The supra-renal capsules are very freely supplied by bloodvessels. Nagel,† who described the arrangement of them very minutely, says that the greatest part of the capsules consist of capillaries, and that there are two sets of bloodvessels very differently distributed. One set pierce the fibrous sheath of the organ and penetrate directly into its substance, where part of them immediately divide themselves into a capillary network, which surrounds the columnar cell masses. Another portion pass through the cortical substance, and divide into branches in the medullary part. Some of these latter branches here form a capillary network, while others return to the cortical substance, and there, amongst the cell-masses, break up into capillaries.

Another portion of the arterial trunks, before entering into the substance of the supra-renal body, to follow the ultimate

* Archiv. f. Pathol. Anat., band xii., heft 4, w. 5, p. 483.
† Müller's Archiv. f. Physiol., 1836, p. 366.

distribution just spoken of, course along for some distance in the fibrous capsule. I have been able to confirm this description, although not entirely. I never saw, for example, any of the branches returning from the medullary portion of the organ break up into capillaries in the cortical substance, which they had already traversed in the form of twigs. My idea of the distribution of the vessels may be given in a very few words. When the arteries arrive in the fibrous capsule of the supra-renal body, they divide into a number of branches, which spread themselves in the fibrous sheath over the organ. These branches penetrate at various points into the cortical substance, in which

FIG. 6.



Transverse section of a human supra-renal capsule, showing the distribution of the bloodvessels. *a*, artery; *b*, veins; *c*, sinuses.

they subdivide into smaller branches. The greater part of the latter form a capillary network amongst the columnar cell-masses. While a few pass onwards into the medullary substance to supply its tissue with blood. The veins resulting from the reunion of the capillaries commence in the cortical part of the organ, and pass on towards the sinuses, into which they empty themselves. The arrangement of the bloodvessels is represented in Fig. 6.

Nerves.—The supra-renal capsules are supplied by almost more nerves than any other organ of the body. Nearly every section placed under the microscope contains some twigs. They run in such irregular order that I have not yet been able to satisfy myself with their true distribution. According to Ecker, however, the bundles of nerves surrounded by cellular tissue, which penetrate the cortical, give off no fibres until they have reached the medullary substance. They then break up into twigs, and form a network of nervous tissue.

Upon histological grounds, Kölliker says that he would regard the function of the cortical and medullary substances as distinct from each other. The former portion of the organ, he thinks, is probably in some way connected with the blood glands; while the latter substance, in consequence of its richness in nerves, and its containing cells resembling ganglion corpuscles, he regards as probably an apparatus connected with the nervous system. Leydig and Bergmann go even further, and say that the supra-renal bodies stand in so close a relationship with the nervous system, that they are entitled to be considered as a part of it. I cannot agree with either of these conclusions; because, although in man and some of the higher mammalia the cortical and medullary substances appear to be made up of perfectly different elements, this is not the case in all. In the horse and in birds, as Frey has pointed out, the whole supra-renal capsule seems to have a tolerably uniform structure, and even in man I have occasionally seen the medullary substance filled to such an extent with the cell-masses proper to the cortical portion, that scarcely any difference

could be detected between the two parts. Moreover, from my observations, chiefly those upon sheep, I am not prepared to admit that the large cells of the medullary substance are ganglion corpuscles; and further, the columnar cell-masses are so closely allied to some of the gland vesicles found in the digestive canal, especially to those of the Peyerian patches, as Frey pointed out, that I incline to the opinion that the function of the former is like that of the latter, secretory. As the aim of this communication is not to attempt an explanation of the function, but merely to describe the structure of the supra-renal capsules, I shall not at present enter more fully into the question.* I shall merely add that in the supra-renal capsules of the frog I have been able to trace the development of blood corpuscles. I have seen, as represented in Fig. 1, first, a collection of granules without any cell-wall, or the slightest appearance of a nucleus, (Fig. 1, A); secondly, a mass of granules surrounded by a delicate envelope; thirdly, a gradual aggregation of the granules in the centre of the cell; fourthly, the increased aggregation of the granules, giving rise to the appearance of a nucleus, (Fig. 1, A, 2); fifthly, a perfectly-formed granular nucleus, and a great decrease of the granular matter throughout the rest of the cell; sixthly, a homogeneous nucleus, and a total absence of granular matter, the cell having at the same time a general yellow hue; seventhly and lastly, a perfectly-formed blood corpuscle. (Fig. 1, A, 3.) Besides, there are a number of much larger cells in the supra-renal capsule of the frog; they are pale, slightly granular, have a very delicate cell-wall, a well-marked nucleus, and occasionally one or two nucleoli. (Fig. 1, B, 1.) Sometimes a cluster of nuclei is observed, which present no appearance of being enclosed in cells. (Fig. 1, B, 2.) This observation tends to support the opinion that the supra-renal capsules serve for the higher organization of the blood.

Somerset-street, Portman-square, May, 1858.

* Vide paper entitled "An Experimental Inquiry into the Function of the Supra-Renal Capsules, and their supposed connexion with Bronzed Skin," in the British and Foreign Quarterly Review for January and April, 1858.

ILLUSTRATIONS

OF A

PECULIAR BLEEDING TUMOUR OF THE RECTUM.

BY

RICHARD QUAIN, F.R.S.,

Professor of Clinical Surgery in University College, London;
Surgeon to University College Hospital.

LONDON:
WALTON AND MABERLY,
28, UPPER GOWER STREET, & 27, IVY LANE, PATERNOSTER ROW.
1859.

LONDON:
BRADBURY AND EVANS, PRINTERS, WHITEFRIARS.

ILLUSTRATIONS,

&c.

THE morbid growth which it is my purpose to illustrate here, must be of very rare occurrence, since I have not been able to find any recorded instance of a strictly analogous formation. Two examples have fallen under my own observation in practice. In the history of the case to be first referred to, which I saw in consultation with Dr. Rogers, these were the leading facts:—

Mrs. R., a lady in good health and with every appearance of a sound constitution, at middle age, gave the following account of her malady. Ten years ago she began to suffer "irritation" of her bowels. She had frequent calls to evacuate their contents—as often as ten or twelve in a day, passing mostly nothing but mucus.

Four years since she became conscious of the protrusion of some "substance" or tumour at the anus in company with every fæcal evacuation; and since then the protrusion has continued under the

same circumstances. When I first saw this lady, she still had the same frequent needs to evacuate. The frequency varied. Any food that disagreed with the stomach, pastry for instance, increased it. The discharge has lately been for the most part a thin fluid reddened more or less with blood. At night, while lying a-bed, Mrs. R. was free from uneasiness; but when she left her bed, there arose at once a sense of fulness in front of the abdomen as well as behind, together with a feeling of inability "to draw herself up." At the same time the nisus for evacuation became urgent, insomuch that while engaged with her toilette, she was obliged to empty the bowel commonly three times. The protrusion of a tumour from within the gut into the grasp of the sphincter often occurred without fæcal evacuation.

This lady had been told from time to time, during several years, that she suffered from piles. From the various methods of treatment that had been resorted to for her relief, she derived no advantage. Recently she had taken the acid tincture of iron (tinctura ferri sesquichloridi) prescribed by a physician. While she used that medicine, the general health was improved; but the sense of a lump being in the bowel became more evident, and at the same time the loss of blood was greater.

I found the tumour fixed to the bowel about two inches from its orifice. It was of deep red colour; and it bled freely. It was connected with the mucous membrane upon which it was sessile. In other

words, there was no connection with the muscular coat of the gut, and there was no narrow part in the form of a neck. The bowel around the diseased out-growth, was found to be free from hardness, and also from any other indication of disease.

Assisted by Dr. Rogers, Mr. Cardell and Mr. Vincent Jackson, I removed the tumour. After the operation, which was performed in July 1857, the patient did well in all respects. Dr. Rogers has informed me that some months ago his patient had a degree of discomfort from a costive state of the bowels; but that the trouble on that score being removed, she felt not the least inconvenience in the bowel. I have ascertained that now, August 1859, Mrs. R. has not the slightest suffering from a return of the former or from any other affection of the bowel.

The size and form of the tumour are well represented in the figure, No. 1, which has been taken from Mr. Ford's accurate drawing of the recent preparation. The colour was that of arterial blood. To the touch the mass was soft and pulpy, giving the notion of a comparatively small amount of solid matter. Short, thick, soft processes projected over the whole surface; they were more or less club-shaped; some were undivided, others were notched. Under the microscope each process was found to consist of a thick coating of epithelium, large vessels, and basement membrane, with a support of fibrous structure: fig. 2. No appearance was discovered to

indicate a malignant or cancerous growth, though careful search was made by Professor Quekett, of the College of Surgeons.

The tumour in the second case was in a great measure similar in appearance and in kind to that above described. The same rounded projecting processes were seen on it. The size, however, was much greater, and the processes differed more one from the other in shape. The vascularity was the same, even more developed—the whole tumour and its parts being, as it were, more developed. The drawing has been taken from the preserved preparation of the full size. When first removed, however, the mass was considerably larger. It has been shrunk by the strong spirit in which it was immersed for preservation, and has, at the same time, become more solid to the feel. See figure 3.

Before I saw the patient from whom I removed this growth (a lady, then aged 68 years) she had been suffering inconvenience in the bowel for seven years; and during two of those years she was in almost continual distress occasioned by the presence of the large mass and its constant protrusion each time she had an evacuation, and even with the escape of flatus. It was replaced by a servant after each descent; in fact, a servant was kept exclusively to attend on this lady. There was much loss of blood.

Five years have elapsed since the operation by which the tumour was removed, and there has been

no indication of any return of the disease in the same place, or of any evil elsewhere arising from it. In a former publication ("The Diseases of the Rectum," 2nd edition, page 295), I have made reference to this case.

It will be observed that the processes, which constitute the characteristic of the appearance of the disease, vary much in thickness and in length in the example now under observation: figures 3 and 4. They are much larger than in the preceding case, though, like the whole mass, they have lost much of their natural size under the influence of the preserving fluid. The processes are generally enlarged at the ends—club-shaped; and that shape is much more marked in some than their neighbours. In the smaller figure (4) some of the processes are seen to be single, but others are joined at their origin, springing, several from the same stem; while again, slender processes arise from thick ones near their bases. In the middle of the same (smaller) figure, No. 4, one process with a narrow root swells suddenly into a little mass, shorter and much thicker than those around. Many such will be recognised in the larger figure, and, indeed, the few more completely shown in the small drawing may be regarded as typical of the whole growth.

The result of microscopical examination was in this case similar to that in the former one: vascularity was the most prominent character, the vessels being covered only with a very delicate membrane,

and a layer of epithelium. No character of malignant disease was observed, though carefully sought for. The tumour was examined in its recent state by my colleague, Dr. Jenner, the professor of pathological anatomy in University College.

I do not hesitate to regard the morbid outgrowths in the preceding cases as being the same in kind. The principal characteristic of both was vascularity; the principal evil they occasioned was the loss of blood. There was in both, when recent, as well as around them in the bowel, the same absence of hardness, with the same freedom from taint of the constitution. The naked eye appearances of the surface did not differ materially; and the microscopical characters were substantially the same.

In a publication before referred to, I applied the term "Villous" to one of these tumours, on account of the projections on its surface. But that designation has been abandoned, for the reason that it might lead to error, or at least to confusion, since it has been already assigned by German observers of high authority to growths described by them as a form of cancer. To the nature of the class of growths so named (villous cancer), it becomes necessary to make some reference, since the tumours described in this paper might be assimilated to them. The reference will be such only as may be enough to serve for comparison.

Under the name "villous cancer," (in German, Zottenkrebs), and as a variety of medullary cancer,

Rokitansky* has ranged the arborescent vascular tumours which form in the urinary bladder, and (besides others) the arborescent growths in cysts, "as chiefly on the inner wall of the cysts of cysto-carcinoma of the uterus." The following is his definition:—"The villous cancer is a medullary carcinoma, whose stroma is represented by the arborescent form of growth," p. 266. Further he states, "that the only difference between villous cancer, and the common form of medullary carcinoma is in the form, which, moreover, depends on the stroma," p. 269. The arborescent form, which is thus regarded as the distinguishing character of villous cancer, is well illustrated in the tufted vascular growth of the urinary bladder. But that form cannot be said to characterise the "peculiar bleeding tumour" of this essay.†

Another German writer, a practical surgeon,‡ has described, as examples of "medullary villous cancer of the rectum," two cases in which he operated, for extirpation of the disease. The nature of the tumours in these cases will be sufficiently shown by a short extract. "The medullary villous cancer of the rectum," says Dr. Schuh, "is not, to my knowledge, mentioned by any writers. I have published

* Lehrbuch der pathologischen Anatomie, von Karl Rokitansky, Band 1, dritte Auflage, 1855.

† See Note, page 11.

‡ Pathologie und Therapie der Pseudoplasmen, von Dr. Franz Schuh, Professor der Chirurgie an der Universität in Wien. 1854.

two such cases. The likeness to epithelial cancer is much calculated to mislead, and it may be because of its being confounded with this latter, that no one makes mention of medullary villous cancer of the rectum."

"First case.—A woman, 32 years old, states that two years ago she began to have difficulty in voiding fæcal matter, which was often mixed with blood. Six months since a lump appeared at the anus, which gradually grew. The appearance on admission was as follows: outwardly, the degeneration formed a light-red, hard, everted, annular tumour, which continued inwards, and extended about three inches upwards. There were no prominences within, but all appeared everywhere pretty even," p. 432.

Of the second case it is said, in Schuh's report, that "a lump appeared at the anus five months before:" "that at the time of the patient's reception the anus was nearly encircled by a bluish-red excrescence * * * * the consistence of which was solid, in parts remarkably hard, and the skin and the mucous membrane were involved." * * * "The patient was a female, aged 31 years, who had been suffering in the rectum for two years," p. 433. The diseased parts were excised in both cases; and in the first, indurated glands were removed from the cellular membrane, high above the disease.

It can be but little necessary to point out that these hard deposits, growing quickly, spreading into the surrounding structures, infiltrating them, and

affecting lymphatic glands, are entirely unlike the soft vascular outgrowths which it has been the object of this paper to describe. If Professor Schuh's cases are examples of medullary villous cancer, it is obvious that mine are not; and the two ought not, in my judgment, to be connected by any community of name.

Note.—It is no part of my present object to discuss the questions:—If, indeed, the arborescent tumour of the bladder belongs to the same class of growths as the "vegetations in cysts," and the "cauliflower formations" elsewhere; and if it is really a form of cancer. Nevertheless, I think it right to say here, that in the examples of that vesical growth which have come under my observation in practice—and the same may be said of others which have been recorded, in so far as I am acquainted with them—the evidence of a malignant type, which the history of a case would afford, was wholly wanting. But, with the most willing acknowledgment of the great assistance microscopical observation affords in skilful and experienced hands, I do not think it too much to say, that that kind of evidence is indispensable in order to affirm the truly cancerous nature of any morbid growth rarely met with. I would add, that notwithstanding some differences of shape, the Bleeding

tumour of the rectum seems to me to have in its nature, as well as in its effects, much analogy with a form of the vascular tumour of the bladder—more with it than with any other growth I am acquainted with.

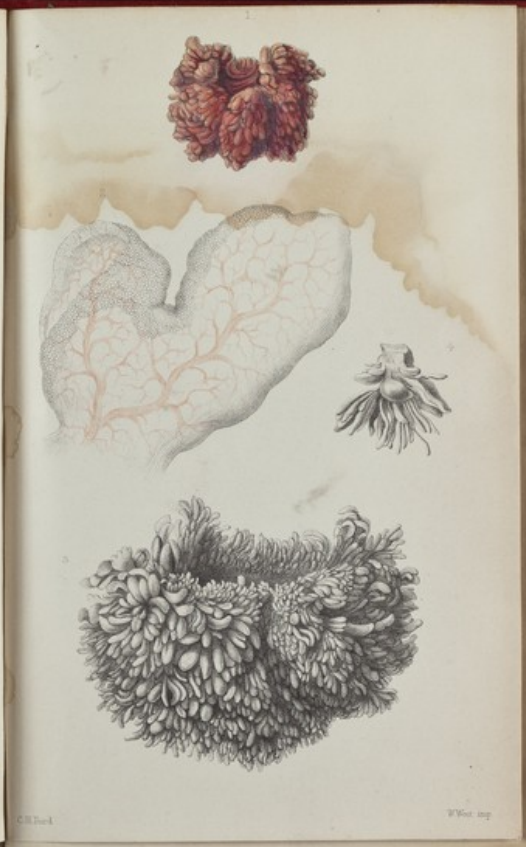
EXPLANATION OF THE DRAWINGS.

FIGURE I.—The tumour removed in Case 1, is here represented of the natural size. It was drawn from the fresh preparation.

A single process is shown in FIGURE II., enlarged to 100 diameters. The epithelial covering is here partially displayed at one side. The blood-vessels are shown.

FIGURE III.—The whole tumour of the second Case is here shown. Before this drawing was made the preparation had been for some time in a strong spirit for preservation; and it was thereby deprived of its natural colour, which was that of the arterial blood it was loaded with. From the same cause the mass had lost much of its natural size, and so had its individual parts or processes.

FIGURE IV. represents a small piece of the tumour (Figure III.), to exhibit the various shapes of some of the processes.



ON THE

TRANSMISSION OF DISEASES

BETWEEN

MAN AND THE LOWER ANIMALS.

BY

W. LAUDER LINDSAY, M.D., F.L.S.,

PERTH.

FROM THE

Edinburgh Veterinary Review and Annals of Comparative Pathology.

JULY 1858.

ON THE

TRANSMISSION OF DISEASES

BETWEEN

MAN AND THE LOWER ANIMALS.

BY

W. LAUDER LINDSAY, M.D., F.L.S.,

PERTH.

FROM THE

Edinburgh Veterinary Review and Annals of Comparative Pathology.

JULY 1858.

ON THE

TRANSMISSION OF DISEASES

BETWEEN

MAN AND THE LOWER ANIMALS.

When, in 1854, I announced—as the result of a series of experiments to which I had been led by an inductive process of reasoning, founded on a study of cholera in man, of the diseases of the lower animals, and of the habits and structure of these animals—that I had succeeded in transmitting cholera artificially from man to the dog and cat, the statement was scoffed at as the "baseless fabric of a vision." Such a thing "had never been done," and "had never been heard of," and therefore could not be! The announcement, indeed, only met with the reception common to all innovations in science or art at the hands of the sceptical, the prejudiced, and the ignorant. I ventured farther, in opposition to the dicta of "constituted authority," the general belief of the profession, and the "*vox populi*," to state my conviction that full investigation would not only bear out all the results arrived at by me, but would establish beyond a doubt, by numerous instances, the fact that certain diseases of the human subject are propagable in, or transmissible to, certain of the lower animals; and that diseases, so produced in the latter, might in them be studied with such effect and facility as to throw much light on certain obscure human diseases. I pointed out the important results likely to be attained by experimentation on the lower animals as to the transmissibility of disease both to and from man, and showed that this branch of comparative pathology will probably prove a necessary key to a correct knowledge of certain epidemic diseases of man. I propounded a full scheme of investigation, to be undertaken partly by professional, partly by non-professional persons, the details whereof may be found in the under-noted journals.[1]

---

[1] 1. "Suggestions for Observations on the Influence of Cholera and other Epidemic Poisons on the Lower Animals," *Edinburgh Medical Journal*, July 1857. P. 33.

2. "Experiments on the Communicability of Cholera to the Lower Animals," *Edinburgh Med. and Surgical Journal*, April and Oct. 1854; *Gazette Hebdom. de Méd. et de Chirurgie*, Oct. 13, and Nov. 24, 1854.

3. "Natural Influence of Cholera on Plants and the Lower Animals," *Association Med. Journal*, Dec. 15, 1854.

4. "Facts for Contagionists, relative to the Artificial and Accidental Communicability of Cholera," *Ibid.*, Sept. 15, 1854.

5. "The Cattle Murrain in some of its aspects," *Lancet*, May 16, 1857. P. 496.

In answer, and in opposition, to my statements, it was argued, that the transmissibility of human diseases to the lower animals, being contrary to analogy, is beyond the bounds of probability; that cholera is essentially a human disease; and that, though cramps, purging, vomiting, and other symptoms might have existed in the animals which died under, and as the result of, my experiments, such symptoms were insufficient singly, or in the aggregate, to constitute *cholera*. Many, indeed, were the ingenious theories and objections raised to explain away the *facts* of my experiments. But it held good in this, as in every other, instance, that

"Facts are chiels that winna ding,
And daurna be disputed."

After the lapse of four years, the facts remain unscathed by the storm of adverse criticism to which they were exposed. Nay, the views and results at which I arrived, in 1854, have been fully borne out by the history of comparative pathology subsequently to that period—chiefly by the ingenious and painstaking researches of various French and German experimentalists; and they are still daily being corroborated and supported. My observations and conclusions have now, therefore, stood the test of theoretical criticism and practical research for four years, and the facts I brought forward have now, I think, been tacitly admitted as established. The veteran physiologist and pathologist, Professor Alison, remarks, "That the cholera can be communicated to animals by inoculation with this matter of the peculiar rice-water stools, has been sufficiently proved by the experiments of Dr Lindsay in this country."[1] The worthy professor has, however, erred as to the mode in which, and the material wherewith, I succeeded in transmitting human cholera to the dog and cat; and, as the publication of this inadvertent error may have given rise, and may still give rise, to misapprehension, I think it right here to correct it. Among other modes and materials of experiment, I certainly used extensively the "rice-water" evacuations of human cholera patients. But though I thus succeeded in producing disease in the animals experimented upon, the disease was not the specific affection —cholera. The latter was produced by exposing the animals to the cutaneous exhalations from the bodies of cholera patients, as contained in fomites. And this, it appears to me, is a most important distinction to be borne in mind, as throwing light, or, perhaps, in some cases, darkness, on various theories that have from time to time been advanced of the essential nature or the *materies morbi* of cholera, and of the mode of its propagation. A reviewer in the *British and Foreign Medico-Chirurgical Review*, referring to my experiments, says: "It would seem probable that this gentleman has *solved the question of the contagion of cholera* by communicating the disease to animals."[2] And lastly, the editor of the *Gazette Hebdomadaire* remarks:—"C'est par des récherches de ce genre qu'on pourrait espérer d'acquérir quelques notions vraiment neuves et élévées sur l'étiologie et la pathologie du choléra."[1] Had it not been that glanders and hydrophobia are familiar examples to the contrary, and beyond all disproof, there is little doubt that the same individuals or parties, who denounced the transmissibility of disease from man to the inferior animals as a doctrine absurd and illogical, and as a fact impossible and untrue, would also have denied the transmissibility of disease from the lower animals to man!

[1] "Application of Statistics to Questions in Medical Science," *Edinburgh Med. Journal*, Nov. 1855.
[2] July 1854: Review of Works on Cholera.

I did not advance theories unsupported by facts; nor did I denominate facts what were mere suppositions or speculations. I laid before the profession fully, and I trust frankly, the whole history of my experiments—their results and the conclusions to which they had led me—having neither bias nor prejudice, but actuated solely by the desire that the truth should prevail. Nor did I trust to my own experiments nor my own studies alone; I was too inexperienced as a comparative zoologist and pathologist to do this. I had the advantage of the opinion of experts in human pathology, who saw the appearances on dissection in the animals experimented upon, and who, I think, agreed with me unreservedly as to the interpretation to be put upon the symptoms and pathological appearances. And further, in order that I might not be led into error by mere resemblances, I requested the assistance of one of the most distinguished and accurate veterinarians and comparative pathologists then in Scotland—the late Professor Barlow of the Edinburgh Veterinary College—who expressed no doubt as to the conclusion to which we were shut up—from a consideration of the whole circumstances of the experiments conducted by me—viz., that the cause of the death of the animals experimented on was cholera, and that that cholera had been transmitted to them from the human subject. For a long period I stood alone and unsupported in my views on the transmission of disease between man and the lower animals—views which, since the date of my original experiments, I have repeatedly brought before the public in various ways, in order to stimulate a spirit of inquiry. Gradually opposition has awakened investigation; sympathy has been evinced, and co-operation secured. The cause has been not a little strengthened by the assistance of such men as Dr Richardson, the accomplished editor of the *Sanitary Review*, and one of the foremost of our living experimental physiologists, who has recently laid before the Epidemiological Society of London, a most suggestive paper "On the Investigation of Epidemics by Experiment."[2] A certain amount of impetus has been given to the study of this, and of kindred subjects, by the institution, some years ago, of the *Sanitary Review*, originally the *Quarterly Journal of Public Health*, the prospectus

[1] In a Note introductory to a paper by me, entitled "Transmission du Choléra aux Animaux; nouveaux détails," Novem. 24, 1854. P. 1044.
[2] Read March 1, 1858: Abstract given in *Medical Times*, Mar. 20, 1858. P. 805.

of which contains the following very catholic announcement:—"As there is evidently a connexion between certain unhealthy conditions of the human family and unhealthy conditions in inferior animals and plants, care will be taken to investigate and record the spreading diseases of these last-named divisions of the living creation, and to point out the best measures which science may suggest for the prevention or removal of such diseases." And a still further impetus, I trust, will now be given by the establishment of the *Edinburgh Veterinary Review and Annals of Comparative Pathology*. Such encouragement is very opportune; for even yet there are few bold enough to advocate the doctrines I have from time to time enunciated regarding the relations of disease in the various tribes of animals. The study of the habits and structure of the lower animals is too much left to the zoologist or comparative anatomist; comparative pathology is handed over almost exclusively to the province of the veterinarian; the placing of veterinary medicine at all on a par with human medicine is regarded as unjustifiable presumption; and the diseases of the lower animals are conceived to be peculiar and not comparable with those of man. It is full time that all such absurd ideas were exploded, and replaced by views more in the spirit of modern philosophy and enlightenment—more consonant with the achievements of modern science—more worthy of the age we live in. Now, this can only be done, I think, by members of the medical and veterinary professions zealously co-operating toward one great end—the discovery of truth and the promotion of science. It has ever, probably, been granted, that veterinary science is deeply indebted to medical science—to human anatomy and surgery, our materia medica, &c.; but it has yet, in great measure, to be proved—though I have no doubt of the fact, as I shall immediately attempt to show—that human medicine may become largely indebted to veterinary medicine. Undoubtedly, the studies of the diseases of man and the lower animals borrow and reflect light mutually on each other. I am bound in justice to avow, however, that veterinary practitioners are not, generally speaking, sufficiently scientific; they have not hitherto, as a body, in this country, done that service to human medicine—to comparative pathology—for which their opportunities so well qualify them; and the main object of the present remarks is to show veterinarians how they may so avail themselves of their opportunities as to render true service to science. My paper, however, is more suggestive than informational; and I propose, therefore, only to give a few illustrations to indicate the kind of harvest that may be gleaned by willing and qualified labourers. For details, as well as for bibliographical notes, which may assist the student, I beg to refer to my various publications in comparative pathology, which will be found enumerated at the foot of page 3 of this Brochure. These publications contain, *inter alia*, 1. Notes of the best authenticated instances—selected from Foreign and British journals—of cholera-epizootics in different parts of the world; 2. Illustrations of the transmission of cholera from

man to the inferior animals; and 3. Illustrations of the influence of a cholera-atmosphere on animal life in different countries.

The discussion stirred up by my views, arrived at inductively, and by the results of experiments to which these views led, has induced me to study, during the last four years, the whole subject of the relations of diseases of the lower animals to those of man. Though the points which at first mainly arrested my attention were, 1. The transmission of human diseases to the lower animals; and, 2. The communication of the diseases of those animals to man, with the laws that regulate such transmission; yet the subject gradually expanded itself into a vast field of inquiry or investigation, embracing a great variety of departments of research—each apparently distinct, yet all more or less intimately related. As a result of my inquiry, I am prepared to lay down the following fundamental propositions; and I select this mode primarily of explaining my views, in order that I may place the subject of the relation of disease in man and the inferior animals, in its chief bearings, clearly before my readers:—

1. That certain human diseases occur, under certain modifications—which are, however, often comparatively insignificant—in various of the lower animals, both wild and domesticated. Such diseases are cholera, yellow-fever, typhus and typhoid fevers, plague, small-pox, measles, croup, pleuro-pneumonia, influenza.

2. That certain "epidemic constitutions" of the atmosphere affect equally, or, at least, in marked degree, man and the lower animals; or, in other words, that a relation frequently subsists between co-incident epidemics and epizootics, or epizootics preceding or following epidemics and such epidemics.

3. That certain human diseases are transmissible, by contagion or otherwise, to various of the lower animals.

4. That certain diseases of the lower animals are transmissible to different individuals, species, and genera of these animals.

5. That certain diseases of the lower animals are transmissible, by contagion or otherwise, to man.

6. That certain diseases, communicated to man from the lower animals, are propagable from man to man in the same manner as ordinary contagious human diseases are.

7. That there are certain modifications in type, character, and degree in transmitted diseases, depending on the habits and structure of the animals affected; and that such modifications must be carefully studied, in order to a correct appreciation of comparative pathology.

As subsidiary or collateral propositions, I would further state my conviction:—

8. That the study of the diseases of the lower animals is calculated to throw much light on our knowledge of various obscure human diseases, and specially such as possess an epidemic character.

9. That the same study has an important bearing on the health of our flocks and herds, the integrity of our agricultural interests at

home and in the colonies, and on the use of the flesh of the domestic animals as food for man.

10. That the pathology, especially, of the diseases of the lower animals, requires an extended and more accurate study in this country.

11. That experimentation on the lower animals, as to the transmissibility of diseases to or from them and man, is one of the most promising means of arriving at sound conclusions as to the nature of various wide-spread diseases, both in them and in man.

12. That, with a view to the proper conducting of experiments, it is necessary to make a selection of the animals to be operated upon, according to the nature of such experiments, or of the diseases to be studied.

I have illustrated, as fully as the literature of the various subjects will allow, most, or all, of the above propositions in my published papers already referred to: I shall not, therefore, occupy space by going over trodden ground. The few additional citations I have to offer here are chiefly such as have been published subsequently to the date of my last paper.[1] Few though they be in amount, and insignificant in value, they may still stimulate some active and liberal minds to inquiry; and should they do so, the aim of my present remarks shall have been fully gained.

Proposition 1. Dr M'Donald describes a "virulent attack of cholera amongst pigs and cattle," in the neighbourhood of Glasgow, in the spring of 1857.[2] He mentions one of the affected pigs as "lying on its side in a state of collapse, eyes dull, ears hanging back, vagina hanging out, pale and flabby. . . . The belly was of a beautiful pink colour, shaded with blue; on some parts of the body the blue colour approached to a black. . . . At intervals it was much cramped, and the soft fæces were jerked to a little distance. It had also vomited, and its mouth was filled with a grumous fluid." It died after six hours' illness. The *post-mortem* appearances were, a flaccidity of body; surface a dark blue, with a pinkish tint on belly; stomach moderately distended with half-digested food, surface studded with patches of inflammation, varying in size from that of a shilling to that of a half-crown; intervals natural in colour; small intestines, at intervals of about eight to twelve inches, inflamed, the length of the inflamed patches being about one inch; large intestines healthy; liver and spleen natural; heart natural. The lungs alone presented features which specially arrested the eye: the surface, and every part cut into, had an intense scarlet colour. One gentleman lost forty pigs from this disease within a short time. At the onset of the epidemic, the disease had the characters of influenza or intestinal fever; there were present prostration, constipation, and loss of power in the limbs, and the animals lingered for a long time. Latterly the disease became more

[1] *Lancet*, May 16, 1857. P. 496. *Ol. citat.*

[2] "Cholera amongst Cattle." By Wm. M'Donald, M.R.C.S., Medical Officer for Cadder Parish, Chryston, near Glasgow. *Lancet*, June 20, 1857.

acute in its character and short in its duration, the animals dying after a few hours' illness. With the supposed causes and the suggested remedy for this disease, as well as with its bearings on the quality of our food-supply, we have, under this proposition, strictly nothing to do. But I shall nevertheless venture to add, in regard to the character of the homes of the affected pigs and cattle, that "the long ranges of building are badly ventilated and not drained. I felt suffocated," says Dr M'Donald, "while walking through them, and there was a depth of six inches of putrid water under the plank on which I walked." He also mentions that he saw two cattle dead of choleroid disease, marked by cramp, after a few hours' illness; and he winds up by asserting—greatly to the comfort of the Glasgow public, no doubt—that the meat of all animals, dying as above described, finds its way regularly into market!

It would appear, from the researches chiefly of Continental pathologists and veterinarians, that that heterogeneous disease known under the general or collective term of Murrain, is chiefly one or other of cholera, contagious typhus, bronchitis, or pleuro-pneumonia. Dr Radcliffe of Bromley, near Leeds, says, that a murrain which he saw in Asia Minor, while serving under Omer Pacha in the autumn and winter of 1855, "was characterized by an intestinal affection. It was a choleraic murrain, and it was regarded by the natives as being intensely contagious." This disease was very fatal. The same gentleman describes a pulmonary murrain or disease of cattle, as common in Yorkshire and other parts of England—which is also very deadly and contagious—the pathological appearances wherein are congested and friable lungs, and distension of veins at the root of the heart, with fibrinous clots.[1] The cattle murrain in Germany "is thought to be a typhoid fever, spreading perhaps by contagion, perhaps by atmospherical causes; and in fact more resembling the march of cholera over the world, than an ordinary epidemic engendered by local causes, and propagated by contagion."[2] The *Times'* Vienna correspondent, again, says that there are three forms of the "Vieh-Seuche," or cattle-plague of the continent: 1. A catarrhal affection of lungs; 2. A pulmonary complaint, with typhoid symptoms; and 3. A highly contagious typhus; the last being the real cattle-plague—the "Löser-Dürre"—of Austria. Mr Jos. S. Gamgee has also distinctly stated that the cattle-murrain of Moravia and Poland, the importation of which was so much dreaded in this country in the beginning of last year, is contagious typhus,—which opinion is also that of the most distinguished veterinary pathologists on the continent, including Professor Spinola in Poland, Professor Bochdalek in Bohemia, and MM. Rénault, Yvart, and Imlin, who were members of a commission instituted by the French Emperor, with a view to investigate the murrain question. The chief pathological lesion is ulceration of the

[1] Letter on subject of "Cattle Murrain," in *Times*, April 2, 1857.

[2] *Spectator*, April 1857.

intestinal glands, the lungs being mostly exempt from disease. The same gentleman shows that it is rather the pleuro-pneumonia of cattle which we have to dread in this country—a disease which has repeatedly committed sad ravages among our flocks and herds, and which would appear to be always more or less prevalent in our midst.[1]

There are now many instances on record of cutaneous eruptions, either identical with, or similar to, those of man, occurring in the lower animals.[2] Alibert has found *Herpes circinatus* in horses.[3] Additional illustrations under this proposition are the occurrence of croup, small-pox, measles, and plague, in the pig, as mentioned by Dr Richardson; of cholera in fowls in the Crimea, by Dr Blenkins; of cholera in cattle, pigs, the horse, dog, cat, racoon, camel, zebra, monkey, &c.: of typhoid fever in hares about Paris; of small-pox in the monkey; of influenza in the lower animals in Jamaica, as mentioned by Dr Milroy; of the grouse disease; and of the hog distemper in America. Many of these illustrations refer equally to the second proposition. Special exception would appear to be taken to cholera as a disease of the lower animals, by those who maintain that it is essentially human. But if animals are subject to such diseases as typhus and typhoid fevers, pleuro-pneumonia, croup, variola and rubeola, what *à priori* reasoning can there be hostile to the idea, that they may be also subject to cholera? The above diseases have been attended by the same symptoms and the same pathological appearances; have even, in many cases at least, apparently been produced by the same causes as in man; and the evidence on which their occurrence, in a variety of the lower animals, has been established, is abundantly trustworthy.

Prop. 2 is sufficiently proved by the published histories of epizootics in this and other countries; by the admirable Report to the Faculty of Medicine of Vienna, on the influence of the epidemic constitution of 1832 on the lower animals—including all tribes thereof—in Austria; and by Dr La Roche's account of the yellow fever of 1853 in New Orleans,—where also all kinds of animals were affected.[4]

[1] Letter in *Times* on subject of "Cattle Murrain," April 3, 1857.

[2] Instances will be found in:—

Hering's *Repertorium der Thier-Heilkunde.* Band. 1, 1840.

Gurlt and Hertwig's *Magazin für die Gesammte Thier-Heilkunde.* Band. 7, 1841.

Letteneur, *Réflexions sur l'Herpes Tonsurant*, 1852.

[3] "On the Existence of Herpes in Domestic Animals and its Communication to Man." By Dr Von Bärensprung. *Annalen des Charité Krankenhauses.* 8. *Jahrgang Heft.* 1: or quoted in *Brit. and For. Medico-Chirurg. Review*, July 1857. P. 263.

[4] For the information of the student I beg to offer a few bibliographical citations on Epizootics:

Census of Ireland for 1851, vol. v. p. 358-9. There is here given an analysis of Epizootics from 1837, in which all tribes and kinds of animals seem to have been affected.—Quoted in Letter by Dr Radcliffe, in *Times*, April 7, 1857.

It does not follow, if we may judge from analogy, that every atmospheric poison which is deleterious to man should be equally so to all or any of the lower animals. This is not likely, when we consider that many mineral and vegetable substances, which are poisonous to the human subject, are altogether, or comparatively, inoperative in certain of the lower animals. I found, several years ago, while experimenting in the lower animals on the antidotes to certain poisons, that the dog is extremely insusceptible of the poisonous action of opium, while it is peculiarly susceptible of that of strychnia. I have given twenty grains of muriate of morphia, and large quantities of opium and belladonna, to dogs, without poisonous effects.[1] Such results demonstrate the inutility or fallacy of employing the dog in experiments on these and certain other poisons; or, in other words, it shows the necessity of selecting the animal according to the nature of the particular experiment, or series of experiments. Other poisons, again, are inoperative, or nearly so, in the rabbit. We know that some animals eat with impunity plants, such as *Œnanthe crocata*, which are poisonous to man; and we know, further, that what is poisonous to one animal is not necessarily so to another.

Prop. 3. My transmission of human cholera to the dog and cat is an instance in point. Experiments of a similar kind, and with similar results, are detailed by Marshall of London, Thiersch and Meyer in Germany, Namias of Venice, Bassani, Feschi, Novati, Calderini, Semmola, and others.[2] Most of these experimenters have

Ramazzini, *Constit. epid. Hasniae*, ann. 1691; *Hist. Feb. Catarrh*, ann. 1730; *Tract. de Contagione Pleuroz: Sager Libellus de Aphthis Pecorinis*, ann. 1764; *Ens. Disquisitio Anat. Pathol. de Morbo Boum*; also *Schreeck, Herder, Valentinus, Gohrliep, Beckrens, Rayger, Stegman, Schelhamer, Hoyer, Grobezius*, in *De Re Rustica*, vol. ii. p. 182. Layard's *Essay on ye Nature, Causes, and Cure of the Contagious Dist. among ye Horned Cattle*, 8vo. 1757; His *Letter* also in *Phil. Trans.* vol. lxx., pt. ii., p. 536; *Dossie Mem. of Ag.*, vol. ii. p. 364.—Quoted in Letter by W. St Croix, in *Times*, April 7, 1857.

Prof. Sauvage's *Account of the Epizootic of* 1730 *and* 1731 *in France.* Quoted in *Illustrated Times*, April 11, 1857.

Reports on the relation of Food and Disease. No. I.: "On the Connexions and Relations of Epidemic Diseases in Man and the Lower Animals," *Med. Times*, April 18, 1857.

Letter on the "Rinder-pest" or Steppe Murrain, from Dr Greenhow, Lecturer on Public Health at St Thomas' Hospital, to the President of the Board of Health, dated 11th April 1857.

Kreutzer's *Gesammte Veterinär. Medicin für Ärzte.*

Herina's *Pathologie und Therapie für Thierärzte.*

Spinola, *Handbuch der Speciellen Pathologie und Therapie für Thierärzte.*

Lancisi, *Dissertatio Historica de bovilla peste a* 1711 *et* 1768.

Ramazzini, *De Contagione Boum Epidemica.* Ed. Lond. 1717.

Haupt, *Mag. für die Gesammte Thierheilkunde*, 1854.

Renault, *Recueil de Médecine Vétérinaire*, 1855.

[1] "On the Non-Susceptibility of the Dog to the Action of certain Poisons," in a paper on "Belladonna," *Assoc. Med. Journal*, June 9, 1854. P. 509.

[2] See an admirable *resumé* of these experiments in a Paper by Mr Marshall in *Brit. and For. Medico-Chirurgical Review*, April 1853.

used either the blood or evacuations of cholera patients, which they have either introduced into the alimentary canal of the animals, or inoculated in the cellular tissue of different parts of the body. The symptoms and *post-mortem* appearances, in many cases, were identical with those of human cholera; in others very similar. Marshall states that Meyer, "with whom," he says, "our own observations would lead us so far to coincide," found that cholera stools given to dogs produce certain symptoms, which "are sometimes followed by death with asphyxia, and with *post-mortem* appearances very much like those observed in the cold stage of cholera among men." Meyer also describes the case of a dog, "in which the symptoms and *post-mortem* appearances were highly characteristic of true cholera"—the disease having been produced by its licking the evacuations of cholera patients. In reference to the presence of a *peculiar* or specific poison in cholera evacuations, Meyer, "perhaps too sanguinely," says Marshall, "maintains, that from the entire evidence this is probably the case."

"In some experiments I have made," says Dr Fletcher, "I have transmitted the human *small-pox* through the horse to the cow, and so to the child in the form of cow-pox."[1]

Prop. 4 has been proved by experiments and observations made by the professors of the French Veterinary Schools of Alfort and Lyons—schools which have gained and deserve a proud pre-eminence by and for their achievements in veterinary pathology.

Prop. 5. Various memoirs have of late years been published in Germany, and especially France, to prove the transmission of the *Herpes* of animals to man. One of the most recent of these, is the Report by M. Devergie to the Academy of Medicine of Paris, in regard to *Herpes tonsurans*. It has long been *suspected* that transmissibility by contagion existed; but it has been *proved* only recently. He seems to proceed on the experiments and observations of M. Reynal, which go to prove that the Herpes of horses and oxen, at least, is transmissible to man. M. Devergie believes that the *Herpes tonsurans* of the lower animals may give rise either to the same form of *Herpes* in man, or to the *Herpes circinatus;* and he ascribes both these species of herpes to the growth of a parasitic fungus—the Tricophyton.[2] Dr Von Bärensprung essentially agrees with M. Devergie. He thinks that the herpes of animals resembles most closely, if it is not, the *Herpes tonsurans* of man; and he ascribes it to a *conferroid (?)* growth. He and other observers probably use the term *conferroid* in a popular sense, and withal an erroneous one. The *confervæ* belong to the *algæ*—salt or fresh water plants mostly; while growths of this character are always *fungoid*. He rubbed some of the scales of this herpes from one of the lower

[1] "Letter on Vaccination," by J. S. Fletcher, Surgeon to the Bromsgrove Union House and District. *Lancet*, Oct. 31, 1857.
[2] Quoted in *Medical Times*, Feb. 13, 1858. P. 176.

animals, containing necessarily abundance of the sporules and mycelium of this fungus—Tricophyton, or by whatever other name we may call it—on his left fore-arm. No effect was produced for several days; but after a longer interval considerable itching caused his attention to be directed to the spot, which he found occupied by a well-formed patch of *Herpes circinatus*, about the size of a sixpence. This patch increased in three weeks to the extent of the size of a crown-piece.[1] Again, Dr Fehr has noticed, in Switzerland, a peculiar herpetic eruption in certain of the lower animals, which is communicable to man. Similar observations have frequently been made on the Continent in regard to itch.[2] A further illustration is the disease in France called *Anders*, which appears recently to have been described for the first time. M. Lemaistre gives an account of it as occurring among horned cattle, in the provinces of Limousin and Auvergne; in the former province it goes under the name of *endai*, and in the latter under that of *anders*. It is transmissible to man; but the characters of the disease are not quite the same in man as in animals. This is what we should *à priori* be prepared to expect. In cattle, it is a cutaneous eruption, beginning as pimples, which discharge a reddish-yellow fluid, and harden ultimately into crusts or scabs like Impetigo. These pimples grow in the form of ring-like patches. It is popularly believed to be contagious among cattle. In man, it is more local and circumscribed than in animals, being confined chiefly to the face, fore-arms, and back of hands. In some cases the disease in man was traced to contact with affected animals; while in other cases it appeared to have been communicated from man to man.[3]

The latter circumstance, which does not appear to be clearly proved, illustrates also Proposition 6, which, it must be confessed, is as yet but meagerly supported by substantial evidence. It is note-worthy, that in the cases of herpes, scabies, and anders, the material or medium of communicating the disease is something tangible and visible—something more substantial and satisfactory than an undefined volatile poison, as in cholera. In herpes we have the sporules of a fungus, in scabies the ova of an insect, and in anders, probably, as in glanders and hydrophobia, contact with diseased secretions or excretions—the pus of the pimples or pustules, which, in a sense, constitute the disease. This class of cases is, therefore, distinctly separable from such affections as cholera, where the germ of the disease is intangible, invisible, inappreciable, except in its effects.

Prop. 7 is in a great measure self-evident; and I have in former

[1] *Brit. and For. Medico-Chir. Review*, July 1857. P. 263. *Ol. cit.*
[2] *On the Animalcules of the Itch of Man and Animals, and on the Transmission of Itch from Animals to Man*, by M. Bourguignon and Delafond, laid before the Academy of Medicine in Paris, in Nov. or Decem. 1857.
[3] "Anders—a Disease communicable from the Bovine Species to Man, and probably from one Human Being to another," by M. Lemaistre of Limoges. *L'Union Médicale*, 26th Jan. 1858; and *Brit. and For. Medico-Chir. Review*, April 1858. P. 536.

publications sufficiently illustrated it. When we consider to what an extent in man the same disease varies in individuals according to circumstances of idiosyncrasy and habit, sex and age, &c., we shall cease to be surprised that the same disease should present many and varied modifications in animals, differing so much from man, and from each other, as cattle, horses, pigs, dogs, rabbits, fowls, and fishes.

Prop. 8 remains, in great measure, to be proved. The light to which the proposition refers is as yet only glimmering in the far distance; still it is a distinct glimmering, and it is for the veterinarians of our age to enlarge this glimmer into a brilliant light. It seems to me that the veterinary pathologists of this country are, as a body, behind their French and German confrères; and that, to place themselves on an equal footing, veterinary science must be studied more philosophically in this country than hitherto. It is a great pity that the veterinary practitioner should, in too many cases, degenerate into the mere horse or cow doctor, whose ignorance leads him to be frequently regarded as a quack, or an "old wife." Why should he, with all the facilities of modern education, not be as fully informed of chemistry, histology, pathology, zoology, and botany, as his brethren of the medical profession? Why should he throw away his glorious opportunities of contributing, in his own way, and in his own sphere, to the progress of knowledge—to the advancement of his science and art? Never, in the history of veterinary medicine, in this or any other country, was the young veterinary student in a more favourable position for entering upon the philosophic study of the etiology, symptomatology, and pathology of the diseases of the lower animals. Veterinary colleges abound, whose chairs are occupied by distinguished professors; there are ample opportunities for the study of all the sciences bearing upon veterinary medicine; there are not a few magazines devoted to veterinary science; and the scientific veterinarian would here unquestionably secure the sympathy and co-operation of the medical profession. The veterinary student should aim at higher education; his talents cannot be too highly cultivated. It is quite a mistake to suppose that any sort of education is good enough for a "horse doctor!" Undoubtedly, the student who, in addition to a knowledge of veterinary science, possesses some acquirements in human medicine, will be better qualified for advancing his profession than his less accomplished companion. But let not these remarks dishearten nor discourage any observer or student, however humble his qualifications. I have taken pains elsewhere to show that all persons concerned in any way with the management or use of the lower animals may contribute towards the elucidation and proof of such propositions as I have stated above. The essential qualifications of the observer are shrewd common sense, patience and perseverance, and a willingness to labour in behalf of science.

Prop. 9 is, again, in great measure, self-evident. Its bearings are amply illustrated by the recent efforts of Messrs Gamgee, Gant,

and others, in regard to the better supplying of our markets with healthy butcher meat. These gentlemen have shown that the flesh of animals, which have died of all manner of diseases, finds its way regularly into our markets—partly, no doubt, from ignorance of veterinary pathology on the part of the inspectors of these markets; and Mr Gant has pointed out how fatty degeneration, *inter alia*, results from the absurd prize fattening system.

Prop. 10. The department of pathology is, perhaps, that in which our veterinary practitioners are most deficient. The records of many epizootics and rare sporadic diseases have frequently been rendered worthless by the absence of pathological details. The Registrar-General of Deaths, &c., in a quarterly report about the beginning of 1857, remarks: "The pathology of domestic animals is exceedingly imperfect; their diseases are badly characterised; and the effect of epizootic causes on the human race is little understood."[1] This is a most true and most suggestive sentence. There are few more important studies to the young veterinarian than this; few likely to be more pregnant of advantage to science; few more certain to gather laurels for himself.

Prop. 11. I am daily more and more convinced of the important results likely to accrue from experimentation on the lower animals, in regard to our knowledge of such diseases as cholera, syphilis, small-pox, erysipelas, dysentery, &c.; and it is not a little encouraging to find my views shared in by Dr Richardson of London, who has recently earned for himself an early, but solid and enviable fame, by his admirable series of experiments on the "Cause of the Coagulation of the Blood." In his paper at the Epidemiological Society, he specially recommends experiments in scarlatina, small-pox, and typhus; he narrates experiments of his own, in which he succeeded in producing all the characters of typhus by introducing alkalies into the blood; and he shows also that a typhus state may be produced by the injection into the system of putrid animal matters. He deplores, as deeply as I do, the present unsatisfactory and unscientific character of the study of epidemiology; and he believes experimentation on the lower animals to be one of the most likely means of improving our knowledge of epidemics. He suggests, as subjects for investigation, the following points, which he puts in the form of propositions:—

1. "That, by experiment, it might be ascertained in what excreta the poisons of certain of the epidemic diseases are located."

2. "By what surfaces of the body such poisons may be absorbed, so as to produce their specific effects."

3. "Whether the virus of disease, in reproducing its disease in a healthy body, acts in the development of the phenomena by which the disease is typified, primarily or secondarily, *i. e.*, by its own re-

[1] Quoted in a Letter from Mr Joseph S. Gamgee on "Quarantine and the Contagious Typhus of Cattle," in *Lancet*, May 16, 1857.

production and presence, or by the evolution of another principle or product."

4. "Whether climate, season, or other external influences, modify the course of epidemics, by producing modification of the epidemic poisons, or modifications in the system of persons exposed to the poisons."[1]

Every one versant either with human or veterinary medicine is fully aware of the importance of experimentation on the lower animals in questions of physiology or toxicology; but it has not been resorted to, to the extent that is desirable, in questions of comparative pathology, or of epidemiology.

Prop. 12 is only consonant with our knowledge of the variations in structure and habits of the lower animals, and of the phenomena of disease as modified by such variations. Dr Richardson remarks, the pig is specially suitable in experiments on small-pox, scarlatina, and typhus, inasmuch as it appears to be more susceptible of the influence of these diseases than any other of the lower animals. Again, I selected the dog and cat in my experiments, as having a structure and habits closely resembling those of man; while other experimentalists have chosen the rabbit, mice, fowls, &c.

In conclusion, I would earnestly call upon veterinary practitioners throughout Scotland to investigate, so far as lies in their power, some of the propositions given above, or any of the collateral subjects or points which they may suggest; and further, to avail themselves of the opportunity which the *Edinburgh Veterinary Review* affords of recording their observations, however isolated and apparently insignificant. I think I can venture to assure the reader of the favourable reception of all reliable observations in comparative pathology by the editor of this journal, whose efforts to place veterinary science more on a par with human medicine deserve to be crowned with success. The reader will find a vast field of inquiry embraced in the propositions I have laid down, and in my various papers on comparative pathology: he may select for himself a path of research suitable to his tastes or opportunities. Due advantage has certainly not, I think, been taken of that section of the *Sanitary Review* which is devoted to a record of epizootics and their relation to epidemics. But let us hope for better things now, when both the medical and veterinary professions have journals opening their pages for the reception of this species of information, and inviting, nay urging, contributors to avail themselves thereof!

[1] *Medical Times*, Mar. 20, 1858. P. 306. *Ol. cit.*

PRINTED BY ANDREW JACK, CLYDE STREET, EDINBURGH.

ON THE

ACTION

OF

HARD WATERS UPON LEAD.

BY

W. LAUDER LINDSAY, M.D., F.L.S.

From the Edinburgh New Philosophical Journal, New Series, for April 1859.

EDINBURGH:
PRINTED BY NEILL AND COMPANY.
MDCCCLIX.

On the Action of Hard Waters upon Lead.* By W. Lauder Lindsay, M.D., F.L.S.†

It is, and has long been, currently believed—1. That where there is free access of atmospheric air, pure or soft waters—that is waters absolutely or comparatively free from saline ingredients, readily corrode lead and become impregnated—sometimes to a poisonous degree, with some of the salts thereof. 2. That the rapidity and extent of this solvent or corrosive action are proportionate to the purity of the water—that is, its freedom from neutral salts. 3. That impure or hard waters, that is waters containing a considerable amount of neutral salts, do not so affect, or become impregnated with, lead. 4. That such waters are prevented from acting on lead by, or in virtue of, their saline constituents, which exert a sort of protective or preserva-

* I use the term *hard waters* to indicate waters containing an easily appreciable amount of neutral salts, especially of the carbonates, sulphates, and chlorides of lime and magnesia. Under this head a large proportion of spring waters is to be classed. The term is employed in contradistinction to that of *pure* or *soft* waters, which either contain no saline ingredients, or a very small proportion: such are rain, snow, and many river waters. I think it necessary thus to define my meaning, because I have found that there is frequently great difference of opinion between professional and non-professional persons as to what is, or constitutes, *hard or soft* water. A water, in reference to which I will immediately have occasion to make some statements, was reported upon by the analyst, an English chemist of repute, as "*very soft*," while the user of the water, a lady of great intelligence and experience, described it to me as decidedly *hard*.

Professor Christison considers a water *soft* which contains less than $\frac{1}{2500}$ part of its weight of saline ingredients; *hard* if it contains more than $\frac{1}{1000}$; *mineral* if more than $\frac{1}{250}$. *Soft* water has the property of forming a lather with soap, and it is suitable for washing purposes, neither of which properties does *hard* water possess.

† Read before the Chemical section of the British Association, Leeds Meeting, 24th September 1858.

tive power in regard to the lead. 5. That, if a given water does not, within a short period, cause a white coating on freshly burnished lead plates or rods, it may be regarded as destitute of any corrosive action, and may therefore be safely allowed to be kept in leaden cisterns, and transmitted through leaden pipes.

Observation, experiment, and inquiry have led me to the following somewhat opposite conclusions:—1. That certain pure or soft waters *do not* act upon lead. 2. That certain impure or hard waters, in some cases containing abundance of the very salts, which are generally regarded as most protective or preservative, *do act* upon lead. 3. That the rationale of the action in these anomalous or exceptional cases is very imperfectly understood. 4. That experimentation on the small scale, and for short periods, is most fallacious, and frequently dangerous, in regard to the conclusions thence to be drawn. 5. That water may, under certain circumstances and to certain extents, contain lead without necessarily being possessed of appreciable poisonous action on the human system. 6. That water contaminated with lead may deleteriously affect certain members or individuals only of a community, family, or house-hold. 7. That the use of water so contaminated is the obscure cause of many anomalous colicky and paralytic affections.

The remarks which follow are intended to be purely suggestive; they are meant chiefly to introduce to the notice of the chemical section of the British Association the fact that the rationale of the non-action of certain soft waters, and of the action of certain hard waters, on lead is very imperfectly understood. The subject of the action of waters upon lead is not at all in a satisfactory state; and, in these days of sanitary legislation, when we are boasting of our acquirements in sanitary science, this is an *opprobrium scientiæ* and an *opprobrium medicinæ* which should no longer be permitted to exist Chemists are totally at variance; when they agree they chiefly agree in what I believe to be error; their analyses are most contradictory, and their theories still more so. In order to place this fact prominently before the reader, I need make but a few citations.

"Numerous attempts," says Dr Medlock, "have been made by some of the most able chemists to arrive at a correct solution of this important question; but it will be admitted that all their attempts have hitherto failed, and that the conclusions arrived at are unsatisfactory in the extreme. Dr Noad examined three waters, which were known to act strongly upon lead. The first was taken from a deep well in the neighbourhood of Highgate churchyard, and was found to contain 100 grains of solid matter in a gallon, of which 57 grains consisted of the *nitrates of lime and magnesia*. The second water, from a spring at Clapham, contained 77·74 grains of solid matter in a gallon, consisting of salts of lime, magnesia, potash, and soda, with 4·10 grains of *organic matter*. The third water, which was found to act upon lead, was that from the deep wells of London. This water contains about 68 grains of solid matter in a gallon, consisting chiefly of the salts of potash and soda, with very little carbonate of lime or organic matter. These waters differ widely in their chemical composition. In the first *nitrates* prevail, in the second *organic matter*, and in the third *alkaline carbonates*. The action of the first on lead he attributes to *nitrates;* that of the second to *organic matter;* and that of the third to *free alkali!* Dr Smith, who investigated the action of the waters of the Dee and Don on lead, found that the quantity of lead dissolved increased with the time the water remained in contact with the metal. He considers the action of these waters on lead to be due to the quantity of *air* dissolved in them."*

It may admit of a doubt whether the opinion of Dr Lambe of Warwick, that *all* natural waters must be held to act upon lead,† which therefore is at all times, and in all circumstances, a dangerous metal to have in contact with drinking water, is not safer, so far as the public health is concerned, than the more modern idea that a small quantity of certain neutral salts in water acts invariably as a preventive to its action on lead.

* "On the Action of certain Waters upon Lead:" Record of Pharmacy and Therapeutics of General Apothecaries' Company, London. Part 2, p. 33. 1857.

† Dr Lambe of Warwick: Researches into the Properties of Spring Waters, 1803, quoted in Dr Christison's Treatise on Poisons, 1835.

I have no desire to create unnecessary alarm; but I have a desire that chemistry should be more creditably represented in its relation to sanitary science than it at present is in reference to the action of drinking waters on lead.

In regard to the desirableness of a thorough knowledge of the chemistry of the phenomena in question, I need here say nothing. The action of drinking and culinary waters on lead is a subject of too great moment to the national health to be lightly considered. Few of our standard writers on Chemistry, Materia Medica, or Public Hygiene, omit reference to this subject. But unfortunately many errors have crept into their works; and these errors have been the means of disseminating false views, not only among the public, but also among the medical profession. Statements of the most opposite kinds will be found in our most familiar text-books on Chemistry and Materia Medica; and this is the more curious and extraordinary, inasmuch as they all profess to borrow or quote from the admirable researches entered upon and published by Professor Christison of Edinburgh, now many years ago. It is true that this gentleman lays down, as a general rule, that pure waters corrode, and become contaminated with, lead; a certain proportion of some saline matters, however, exercising a protective influence, and preventing such contamination. But, as I read and understand the narrative of his experiments and the statements of his conclusions, he appears to me to admit fully and freely that there are exceptional cases of the nature mentioned in my propositions.* These exceptional

* I append a few quotations or statements from Professor Christison's publications on the subject:—"The action is *impeded* by the coexistence of *any* neutral salts in the water; and some salts, whose acids form very insoluble compounds with oxide of lead, *prevent the action entirely in very minute quantity.*"*

Water, which contains less than about $\frac{1}{2\,0\,0\,0}$ of salts in solution, cannot be safely conducted in lead pipes without certain precautions.

Even this proportion will prove insufficient to prevent corrosion, unless a considerable part of the saline matter consist of *carbonates* and *sulphates*, especially the former.

So large a proportion as $\frac{1}{2\,0\,0\,0}$, probably even a considerably larger proportion, will be insufficient if the salts in solution be in a great measure *muriates*.

* Dispensatory, second edition. 1846. Art. *Plumbum*.

cases are not brought so prominently under notice in his various publications on the subject as they deserve to be, nor are they in general satisfactorily explained. But I think that Professor Christison has been very unfairly quoted, nay, frequently misquoted, by compilers, who have never taken the trouble to investigate the matter for themselves. In some text-books the statements made are absolutely erroneous; in others they are too dogmatic and positive,—general rules only being laid down, and exceptions, which are of the utmost importance, being omitted. In order to illustrate the errors of omission or commission to which I have just adverted, I will cite a very few quotations from well-known works on chemistry or materia medica. Professor Graham says*—"Rain or soft water cannot be preserved with safety in lead cisterns, owing to the rapid formation of a white hydrated oxide at the line where the metal is exposed to both air and water: the oxide formed is soluble in pure water, and highly poisonous. But a small quantity of *carbonic acid*,

"It is, I conceive, right to add that in all cases, even though the composition of the water seems to bring it within the conditions of safety now stated, an attentive examination should be made of the water after it has been running for a few days through the pipes. *For it is not improbable that other circumstances besides those hitherto ascertained may regulate the preventive influence of the neutral salts.*"*

"When lead has been exposed for a few weeks to a solution of a protecting salt, and has acquired a thin film over its surface, it not only is not acted on by the solution, but is even also rendered incapable of being acted on by distilled water."†

"Most spring waters, unlike rain or snow water, have little or no action on lead, because they generally contain a considerable proportion of *muriates* [?] and sulphates."‡

"The general result of these experiments appears to be that neutral salts, in various, and for the most part minute, proportions, *retard or prevent* the corrosive action of water on lead, allowing the carbonate to deposit itself slowly, and to adhere with such firmness to the lead as not to be afterwards removable by moderate agitation, adding subsequently to this crust other insoluble salts of lead, the acids of which are derived from the neutral salts in solution; and thus, at length, forming a permanent and impermeable screen, through which the action of the water cannot any longer be carried on."§

* Elements of Chemistry, vol. ii., p. 111. London. 1858.

* Trans. Royal Society of Edinburgh, vol. xv., part 2, p. 271.
† Treatise on Poisons, 1835, p. 481. ‡ Ibid., p. 486. § Ibid., p. 483.

which spring and well water usually contain, arrests the corrosion of the lead by converting the oxide of lead into an insoluble salt, and prevents the contamination of the water." Dr Graham professes to quote from the conclusions of Professor Christison; but the above two sentences contain several statements at variance with the results arrived at by the latter gentleman, and by the majority of experimenters on this subject. The crust or film produced by the action of pure water on lead is neither a simple hydrated oxide nor a simple carbonate, but a permanent compound or mixture of both,—in the proportion, according to Professor Christison, of two equivalents of the latter to one of the former. Where the water contains saline matters, in the deposit are generally found, in addition, the sulphates and chlorides and other salts of lead. Carbonic acid in the water, so far from preventing contamination, assists it, according to Professor Daniell and others, by dissolving a portion of the precipitated carbonate of lead. The carbonic acid, which is the source of the metamorphosis of the hydrated oxide of lead into carbonate, is generally derivable from the atmosphere, not from the water, and this agent operates more powerfully in pure than in hard water. Professor Fyfe of Aberdeen, in a somewhat extraordinary paragraph,* remarks—"Water, when *pure*, has *no* action with lead; but if it be admitted, the metal is slowly oxidated and dissolved. This action goes on *more quickly* when spring water is employed, the *presence of the minute quantity of saline matter* in it *favouring* the action. *Hence* the corrosion of leaden cisterns and pipes conveying water!" It is needless here to point out how completely at variance with the truth these statements are. So entirely are matters reversed, that it would almost appear as if there were some typographical error! Again, Sir Robert Kane† says—"*No danger* is, therefore, to be apprehended from the supply of water to a city being conveyed through leaden pipes and preserved in leaden cisterns; for all water of mineral origin dissolves, in filtering through the layers of rocks in its pass-

* Elements of Chemistry. Second ed., p. 529. Edinburgh, 1830.
† Elements of Chemistry. Second ed., p. 558. Dublin, 1849.

age to the surface, a sufficiency of saline matter to serve for its protection." Other authors follow Professor Christison more closely and correctly, and make use of more cautious expressions. But here, again, I must prefer the complaint, that the exceptional cases, to which I have already referred, are mentioned in so subordinate a way and place that they are apt to be entirely overlooked by the casual reader. Hence the propagation of errors.

In reference to my first proposition, that certain comparatively pure, or what are usually considered soft, waters, *do not* act on lead, it may be mentioned that Professor Christison* found rain water collected on his own house in Edinburgh to be devoid of any corrosive action—a circumstance which he attributes to the presence of alkaline sulphates and chlorides. Again, Professors Graham, Hoffman, and Miller, who sometime ago conducted a Government investigation into the action of waters on lead, with a view to discover "whether any comparative inconvenience would arise from a supply of soft water to the metropolis," contrary to what might have been expected, assert that, "with one exception, neither the soft waters of the Surrey Hills, which have a hardness of only two degrees, nor spring water artificially softened to three degrees of hardness, have any perceptible action on lead. The idea that soft waters invariably act upon lead seems to have its origin in the fact that certain specimens of distilled water, placed in contact with a large surface of bright sheet lead, dissolve as much as six or eight grains of the metal to the gallon."† Dr Medlock‡ asserts that "perfectly neutral and pure" distilled water, from which nitrite of ammonia has been expelled, has no action upon lead; for, in a gallon of this water, allowed to remain in contact with 560 square inches of lead for forty-eight hours, no trace of lead could be discovered.

* Treatise on Poisons, 1835, p. 484, "On the Action of Natural Waters on Lead."
† "On the Action of certain Waters upon Lead," by Dr Medlock; Record of Pharmacy and Therapeutics, Part II., p. 34; General Apothecaries' Company. London, 1857.
‡ Ibid., p. 35.

My second proposition I can illustrate more easily and fully. The illustrations which have presented themselves to my notice have been chiefly of the two following kinds:—1. Corrosion, or erosion, to such an extent as to cause leakage, of cisterns by waters of various degrees and kinds of hardness, or containing various kinds and amounts of neutral salts. 2. The poisonous action of hard or hardish waters impregnated with lead on the human body.

It is needless to multiply instances: two or three must here suffice; but they will probably serve to recall to the memory of many of my readers parallel illustrations. My attention was specially called to the erosion of lead cisterns by spring or well waters about two years ago, by my being requested to examine some cisterns, the bottom-lining of which had been repeatedly eroded to the extent of causing leakage, and which had been as repeatedly repaired. These cisterns contained the water supply of a large public Institution of which I am the physician. The Institution is supplied with water from three different sources, viz.—1. Rain water from the roof; 2. Spring water; and 3. Surface water. With the first I have here nothing to do: it is in reference to the second especially, and also subordinately to the third, that my remarks apply. The spring water is from a deep well on the northern declivity of Kinnoull Hill, penetrating the Old Red Sandstone near where the trap protrudes through it. The water rises in the well to the height of twenty-eight feet. It is good, hard, drinking water, and is used in the Institution for drinking, as well as for culinary purposes. The surface water is chiefly rain water, which percolates through the soil, and is collected in a large tank holding 95,156 gallons. This water is used chiefly for baths, water-closets, and general cleansing processes. The cisterns have no covers; they are contained in the attics of the building, but are not exposed directly to the external air. My attention was first directed to the cistern containing the spring water. I found the leaden bottom of this cistern scooped out here and there into a series of cavities or holes, some of which were minute perforations, allowing of an escape of the water. Moreover, the bottom was covered by a pretty thick layer or coating of a heavy

cream-yellow, putty-like matter, which also filled the cavities or holes above referred to. The cistern was comparatively new. I was told it had been repeatedly thus eroded, and as frequently repaired. But it would appear that the mode of repair had been mere soldering; and this, it is of great importance to bear in mind, inasmuch as I believe this mode of repair to have been the cause of the subsequent more rapid erosion. It has now been abundantly proved that galvanic action occurs in a leaden cistern holding water at the point of contact of the lead with other metals, whether these be in the form of solder or of iron bars, &c.; that this galvanic action is extremely favourable to corrosion; and that it is greatest in waters containing saline matters,—that is, precisely in circumstances in which, were there no galvanic action, the corrosive effect of the water would be least. Similar phenomena had been repeatedly observed in cisterns containing the surface water; and the leakage of these cisterns became a matter of some moment, not only from the expense of constant repairs in comparatively new cisterns, but also from the damage done to roofs and walls of apartments situated below the attics by the escaped water. Facts bear out what theory would lead us to conclude, that the newer the cistern, the more rapid and energetic is the corrosive action of the water on it.

I was naturally led to make an analysis (a rough and qualitative one only, however), with a view to discover precisely the circumstances under which this corrosive action took place. This analysis embraced,—1. The deposit on the bottom of the cistern; 2. The supernatant water in the cistern; and, 3. The spring water, as drawn from the well before it had traversed iron pipes, or been contained in leaden cisterns. The results were as follows:—The deposit contained carbonates, sulphates, and chlorides of lead, lime, and magnesia; iron in abundance; and faint traces of soda. The supernatant water contained the sulphates, carbonates, and chlorides of lime and magnesia, with traces of soda. The carbonates and chlorides were most abundant—the sulphates less so; lime was plentiful; magnesia was in small quantity. There was no lead; and this is of importance to bear in mind; for it frequently happens that, though lead is found abundantly in the deposit on the bottom

of such a cistern, it cannot be detected readily, or at all, in the supernatant fluid. Nor did this water contain distinct traces of iron. The spring-water, as drawn directly from the well, contained the same salts of lime, magnesia, and soda, in similar proportion, without lead or iron. The iron found in the cistern-deposit was probably dissolved as oxide by the water as it passed through the iron pipes which convey it from the well to the Institution; but it may also partly have been derived from the soil. A more careful analysis would probably, however, have detected it in the water of the cistern, and possibly in the water drawn directly from the well, being, in the latter case, derived from the iron apparatus of the pump. Distrustful of my own analysis, I had it repeated more carefully by a friend in Edinburgh.* His results were entirely corroborative of my own more rough and hasty essays. The lead in the deposit was by him converted into sulphate, with a view to ascertain its quantity. Calculating from the weight of the sulphate thus obtained, the deposit was found to contain no less than 43·18 per cent. of lead,—a large amount, to account for which it is right to mention that, in scraping the deposit from the bottom of the cistern, minute portions of metallic lead had been probably included.

Now, the water in question curdles soap with great rapidity and ease, and is therefore decidedly *hard*. But, in considering the action of water on lead, it is important to remark the nature or kind, as well as the amount, of the neutral salts present in it. The carbonates and chlorides were much more abundant than the sulphates, while lime was plentiful, and magnesia and soda occurred only in small quantity. The water of Airthrey Well, Bridge of Allan, for instance, is said to possess no action on lead; yet it contains no less than $\frac{1}{7}$ part of its weight of salts, which are chiefly sulphates and chlorides.† The experiments of Professors Christison and Taylor, and others, have established that the sulphates and carbonates are among the most protective or preservative salts, while the chlorides are among the least so. There is a difference of opinion among observers as to whether the carbonates or sulphates of lime and magnesia are most strongly

* Made in June 1857. † Christison on Poisons. 1835. P. 486.

protective; but there is no doubt that the sulphate of lime is a powerfully protective salt, while it is also one of the most common ingredients of hard waters. According to Professor Taylor, sulphate of lime is the salt occurring in hard waters, which chiefly prevents their corrosive action on lead; and he describes the coating deposited on the lead as consisting of the sulphate of lead. The results of Professor Christison, as well as a consideration of the chemical theory of the action, would lead to the suspicion that the latter statement is not quite accurate; for it is extremely probable, that not only sulphate, but also the carbonate and hydrated oxide of lead, will at least be found in such deposits. Professor Taylor deduces from his investigations, that a water containing sulphates and lime is not likely to corrode, or become contaminated with, lead, and may therefore be safely used for drinking and cooking.

I made a series of comparative analyses, with a view to ascertain especially the condition as to hardness, or the nature and amount of the saline constituents, as well as the action upon lead, of various waters used for drinking and culinary purposes in and around Perth. The following are briefly the results at which I arrived,—the analyses or experiments in question being only tentative or qualitative,—rough and approximative in character, and not aiming at quantitative accuracy:—

I. *Well waters; decidedly hard.*—The wells, the waters of which were examined, are apparently chiefly sunk in the Old Red Sandstone, near where it is penetrated by the trap mass of Kinnoull Hill.

1. Rosemount, Bridgend, Perth; well 16 feet deep.—*a*. Water drawn from lead pipes in the house, but before entering cisterns; carbonates, sulphates, and chlorides abundant; lime and magnesia also abundant, especially the former; sediment or solid matter, on evaporating to dryness, comparatively large; no lead nor iron.—*b*. Water drawn from cisterns lined with lead, but covered with wood, placed in garrets, and not exposed directly to external air. Had precisely the characters of that described under head *a*.

2. Murray's Royal Institution, Kinnoull Hill, Perth.—West well, at a considerably higher level than the well at Rosemount. Water, after passing through lead and iron pipes, taken from a lead cistern in the Institution, having no cover; sulphates and chlorides abundant, particularly former; lime abundant; magnesia in small quantity; neither lead nor iron; burnished lead rods immersed in the water which was exposed to the air became tarnished or streaked white with carbonate in four or five days, but the water was not rendered opalescent nor muddy till the end of one month's immersion.

3. Pitcullen House, Bridgend, Perth.—The elevation of the well is intermediate between that of the wells at Rosemount and Murray's Royal Institution; all three being situated on the slope of Kinnoull Hill. The water had precisely the characters of that of the other two wells above described. Burnished lead rods were tarnished in five days, but were not further affected at the end of one month's immersion, at which time, also, the water in which they were immersed was unaffected as to its transparency.

II. *Spring waters; mostly hard.*—The springs referred to appear to rise from the trap of Kinnoull Hill (consisting of basalts, amygdaloids, and tufas, chiefly) at or near where it bursts through the old red sandstone of lower Perthshire. Like the wells above described, these springs are all situated on the declivity of Kinnoull Hill.

1. Bowerswell, Bridgend, Perth. — Carbonates, chlorides, and sulphates abundant, particularly the two former; lime abundant; magnesia in small quantity; sediment or solid matter, on evaporating to dryness, considerable; burnished lead rods tarnished after three days' immersion; no change of transparency of water after one month's immersion.

2. Muirhall Quarry, Kinnoull Hill.—This is an old basalt quarry, at a higher elevation on Kinnoull Hill than any of the springs, wells, or other sources of water supply, which I have described, or am about to describe. There is a perennial spring at the bottom of the quarry, now covered by a mass of water, which has accumulated for years. But this mass of water consists, in great measure, of rain water and of surface

drainage water from the neighbouring fields. It is a very soft, comparatively pure water. Carbonates plentiful; chlorides and sulphates in small quantity; lime and magnesia also in small quantity; considerable sediment on evaporation; burnished lead rods tarnished by five days' immersion; but transparency of water unaffected at end of a month.

3. Bridgend Water Company's supply.—Water taken from the stream immediately before it enters the reservoir at New Scone. The stream in question flows from, and constitutes the overflow of, the water in the Quarry immediately before mentioned. The composition of the water was identical with that of the quarry water, except that there was a small sediment on evaporation. Its action on lead was also precisely similar.

III. *Surface or Drainage Water; hard.*—This is chiefly rain water, percolating through the soil; but it is also partly made up of spring water of the characters already described.

1. Murray's Royal Institution, east tank.—Carbonates, sulphates, and chlorides in considerable quantity, especially latter; lime and magnesia also in notable amount, especially former; sediment, on evaporation, considerable; lead began to be slightly tarnished after one night's immersion; but the water remained clear after one month.

IV. *River water; decidedly soft.*

1. Tay River water, taken directly from the river as it flows past Perth. This is the source of supply of the Perth Water Company. It is comparatively pure; and in this respect, resembles the water of Muirhall Quarry and the Bridgend Water Company. Carbonates, chlorides, and sulphates, in small quantity, especially latter; lime and magnesia also in small quantity, especially latter; considerable sediment, chiefly mechanical impurity, on evaporation; lead began to be tarnished after a single night's immersion; it was quite tarnished in four days, but the water was unaffected in its transparency after one month's immersion.

2. Tay River water, drawn from the cistern of the water works, South Inch, after filtration. Water had precisely the characters described under head IV., No. 1.

V. *Rain waters; very soft.*

1. Pitcullen House; rain water barrel.—No carbonates; almost no chlorides; a trace of lime and sulphates; sediment, on evaporating down, very small, and chiefly mechanical impurity; lead tarnished, and water, in which it was immersed, opalescent after one night. After three nights' immersion the water was full of white pearly crystals of carbonate, which rapidly accumulated by longer exposure.

2. Murray's Royal Institution; rain water cisterns.—No appreciable amount of chlorides, carbonates, lime, nor magnesia; faint traces of sulphates; sediment, on evaporating down, very small, and chiefly mechanical impurity; lead tarnished and water opalescent after a single night's immersion. After five days, lead rod copiously covered with flocculent masses of crystals of carbonate, which also formed a plentiful sediment; they formed more rapidly, and in greater abundance, than in the rain water from Pitcullen House.

It will be observed that all the waters examined possessed some action on lead; though only in the cases of the rain waters was this action apparently dangerous in amount or degree.

It has sometimes been ignorantly believed that deposits in lead cisterns, such as I have described, are the precipitated salts of hard waters, which are themselves the means of corroding the metal; and, under this impression, great care has been taken to scrape away the deposit so as to expose a fresh surface of lead. This procedure, of course, has only had the effect of aggravating the mischief! Again, it has been imagined by those who regard such deposit as consisting wholly or partly of carbonate or other insoluble salts of lead, that the supernatant water cannot possibly contain salts of lead, and cannot therefore possess any poisonous properties. Now, even the carbonate, which is undoubtedly insoluble in pure water, may be held in solution, it would appear, in minute quantity—sufficient, however, to possess poisonous properties—if the water is surcharged, as is frequently the case, with carbonic acid. Further, it is not at all necessary that it be held in *solution* in order to the development of its poisonous properties, for it

may be simply held in *suspension* in a state of very minute division, in which case there is no doubt of its possessing a poisonous action on the human system. Besides, the hydrated oxide, as well as the chlorides and nitrates, which are frequently, if not always, found in such deposits, are all sparingly soluble in water, and may be, individually or severally, the source of poisonous action. It is sufficiently established, that the chlorides of lime and magnesia are common constituents of spring waters; but it is not perhaps so generally known that the nitrate—according to some chemists, the *nitrite*—of ammonia or other nitrates or nitrites, occur frequently in rain, as well as in river and spring, waters. The mere transparency of a water cannot be regarded as a proof that no lead exists in it. It may appear perfectly pure, and yet contain dissolved hydrated oxide, or other salts of lead, which are slowly thrown down as white and insoluble carbonate on exposure to the air.

A friend in London has furnished me with the particulars of the erosion of a new cistern in his new house at Brompton; and similar cases are common in London. The cistern was roofed with wood; no rain-water entered it; the water supplied to the house was Thames water, from the Chelsea water-works,—a water which he describes to be regarded by laundresses as a soft water, compared with London pump or well water. This water, however, contains nearly 20 grains per gallon of solid matter, 16½ grains consisting of carbonate of lime, and 3 of sulphate of lime and chloride of sodium; while oxide of iron, silica, magnesia, and carbonaceous matter, exist only in minute proportions. There soon appeared, and there continued to be, a copious white sediment on the bottom of the cistern. He made a practice of cleaning out the cistern once a-year; and in about five or six years he found the lead fast becoming eroded over the spots most copiously covered by the white deposit. The remedy he resorted to was plugging the erosions or leakages with gutta percha.

The case related by Dr Wall of Worcester*, and quoted by Dr Christison,† is worthy of record here. A family, consisting

* Transactions of London College of Physicians, ii. 400.

† Treatise on Poisons, 1835, p. 488.

of parents and 21 children, were constantly liable to stomach and bowel complaints; 8 children and both parents died in consequence. The house was sold after their death, and the purchaser found it necessary to repair the pump, the cylinder and cistern of which were riddled with holes, and as thin as a sieve. The plumber employed informed Dr Wall that he had repaired it several times previously. Unfortunately, no analysis was made of the water, and we are left to infer that it was *hard*, from the fact that most of the water about Worcester is hard. This, however, does the reverse of account for its action on lead: hence the cause in this case was supposed to be one of the three following:—1. An unusually small proportion of salts; 2. An excess of carbonic acid; or, 3, "Which is perhaps fully more probable, the corrosion was the effect of *some obscure galvanic action!*" Now it is doubtless very convenient to ascribe or attribute cases, otherwise inexplicable, to "obscure galvanic action;" but it is just to the rationale of this, and similar *obscure* actions of water on lead, that I am desirous of directing the attention of chemists. Many cases similar to that given by Dr Wall are on record; but they are comparatively valueless, from the want of an analysis of the waters concerned. It is extremely desirable that, in all future records of cases, there should be given some key to the character of the water. If nothing further can be stated or ascertained regarding it, the narrator would do service by mentioning whether it is hard or soft—whether it curdles soap, or forms a lather therewith. Dr Christison says,—"Of spring waters, which act with inconvenient or dangerous rapidity on lead, *several instances* might be quoted. But it is hardly worth while mentioning more than one or two by way of illustration: because the nature of the water has been very seldom described so carefully as to supply the means of explaining their operation."*

It may be asked, in the case of cisterns so eroded, and which must continue to contain the same kind of water, How is the tendency to erosion to be obviated? In the cisterns mentioned at p. 10, it was obviated, after repeated solderings and patchings, by coating the lead with a composition of rosin and tallow,

* Treatise on Poisons, 1835, p. 488.

which has answered the desired purpose admirably. This is the common mode of rectifying the mishap in this quarter (Perth). Plumbers, then, use a very suitable *mechanical* means of protecting the lead; but a very ingenious *chemical* means has also been recommended by several distinguished authorities. It has been found that different neutral salts protect lead from the corrosive action of water with very different degrees of efficiency—chloride of sodium in the proportion of $\frac{1}{10}$th, and sulphate of lime in that of $\frac{1}{100}$th. Of these, one of the most powerful is phosphate of soda, a very small quantity of which added to a water, prevents its action on lead. Hence it has been recommended to add to a water which is, or is supposed to be, corrosive of lead, a given proportion of this salt (about $\frac{1}{1000}$th part). Professor Christison has pointed out that this so-called "preservative power" depends on, or resides in, the *acid*, and not the base, of the salt. For instance, the *sulphates* of soda, magnesia, and lime, as well as the triple sulphate of alumina and potash, preserve, as nearly as can be determined, in the same proportion. In regard to their relative preserving power, it is found that salts, whose acids form with lead a soluble salt, are least energetic, and *vice versa*.* Other mechanical means might be suggested to remedy the evil complained of,—such as the substitution of cisterns of other metals, or of wood, earthenware, glass, or gutta-percha. Or the lead may be lined with gutta-percha, india-rubber, or other materials. But my object at present is merely to indicate fully the evil, not to suggest a remedy.† For an illustra-

* Treatise on Poisons, 1835.

† From communications received in relation to the above paper, as it was laid before the late meeting of the British Association, I find that my ideas regarding the coating of the interior of lead pipes and cisterns with substances on which water exerts no action, or, at least, no deleterious action, have been anticipated by the patents of Mr E. K. Davis, of 42 Tenison Street, York Road, Lambeth, London. The lead he manufactures and lines he guarantees to resist the action of waters of all sorts, as well as of beer and similar fluids; and his patents extend to England, France, and Austria. The essential principle of his patents is simply the lining or coating of the lead with substances not acted upon by water, beer, and other fluids, and which cannot, therefore, confer upon such fluids poisonous or deleterious properties. The materials he uses are various, specimens of which I have had an opportunity of examining for myself through the kindness of the patentee. I have accordingly every confidence in recom-

tion of the deleterious effects on the human system of hard water contaminated with lead, I am indebted to the kindness

mending Mr Davis's patents to the notice of all who are charged with, or concerned in, the conveyance or storing up of water, beer, &c., for culinary or drinking purposes. The materials Mr Davis uses are chiefly of two classes, viz.:—

1. Pure tin. A special machinery is applied; the lead pipe or sheet is operated on in a solid state, but at a heat above the melting-point of tin. The fused tin and lead are firmly united, it being easy to make the tin coating of any desired or desirable thickness. If the coating be comparatively thick, then there is virtually a double cylinder or tube,—the outer or thicker being of lead, the thinner and inner of tin. It is perhaps necessary to explain here that such pipes are quite different from what are known to plumbers as "tin-coated pipes." In the latter case, the coating is very thin, and consists of what is termed "fusible metal,"—a mixture of lead, bismuth, and tin.

2. Compositions of various gums, resins, gutta-percha, caoutchouc, bitumen, and similar materials, selecting such only as form an adhesive and flexible film, which will not be ruptured or abraded by the processes of bending and otherwise manipulating the lead. The specimens of pipes I have examined are first coated internally with a composition of several of the materials just named, and subsequently lined by a film of gutta-percha. Gutta-percha or caoutchouc cannot be directly applied as coatings, from their non-adhesiveness to metal when precipitated from their solutions. But they adhere firmly to a surface of any of the compositions prepared by Mr Davis.

In reference to Mr Davis's patents, the *Builder* remarks:—"The most hopeful and efficient remedy, however, now practically available, appears to be that provided under a patent secured by Mr E. K. Davis of Lambeth, for the coating (amongst other processes) of the interior of leaden water-pipes with a thin film or inner tube of tin, or of compositions of gum-lacs and india-rubber or gutta-percha, in a soft or fluid state, by means of improvements in the usual machinery for making leaden pipes themselves. The coating appears (in pieces of the various tubes which we have purposely examined) to be perfectly adherent, of quite sufficient thickness, and otherwise efficient in its formation for the purpose in view."

"The mechanism by means of which these and other processes are effected is ingenious; and the patent of Mr Davis comprehends various improvements, —such as a double-action hydraulic press, the casing of soft metal pipes with block-tin and other metals, the forcing of soft metals by compressed air, the plating of sheets in a way described, the making of dies for coating lead and other pipes with other metals, a mode of making still-worms, the coating of soft metal pipes with gutta-percha, india-rubber, &c., and various other improvements. It is with special reference to a remedy for the contamination of water by lead that we have examined the pipes coated under this patent; and we think it well deserves the public patronage, as tending towards the realization of that very desirable end."

The London plumbers, who are familiar with the fact that new cisterns in London houses are frequently eroded to the extent of leakage in the course of one or two years, usually apply as a remedy a coating composed of rosin

of a lady in Derbyshire, in whose household the following incident occurred.*

"Mr B——, the gentleman, who acted as tutor to our children last year, came to us soon after Christmas. He had previously been in perfect health, so far as regarded the digestive organs, although the localities in which he had been residing—Edinburgh during the College session, and Glasgow as his home—did not offer any peculiar advantages in this respect. Soon after his arrival he complained of dyspepsia in no ordinary degree. The usual remedies had no effect. A strict system of diet proved equally inefficacious. After exhausting the skill of our country practitioners, he went to Manchester to seek for relief there. Mr ——, a surgeon of large practice there, after a careful investigation of his mode of life, his symptoms, &c., told him that he could not account for the attack except on the supposition of lead being held in solution by the water. Mr B—— objected that he was the only invalid in a family of nineteen, and that he believed the water was from a spring, and conveyed to the house by iron pipes. Mr S—— rejoined that the conditions of his ordinary life were so favourable to health, the hours of the meals so appropriate, and the remedies which had been used so fitted to benefit a case of pure indigestion, had there been no irritating cause, continued that the presence of lead in the water offered the only solution of the difficulty. On his return, we drew four bottles of water from four different taps within the house, and sent them to Professor —— for investigation.

"The origin of the water with which we are supplied is rather singular. Far up on a high hill two springs, within ten or twelve feet of each other, rise, and mingle their water a few yards lower down. One of them is *excessively hard;* the other is *equally soft.* Both flow together into a reservoir; thence filtering through gravel, they pass into *iron* pipes,

white lead, and grease! This, it need hardly be observed, is a most objectionable remedy compared with the substances enumerated in Mr Davis's patents.

Dissolved india-rubber has, however, been applied to a similar purpose; and it has been suggested to me by an ingenious correspondent that a varnish of coal-tar, which dries hard and fast, and is deemed a most serviceable coating for exposed iron and other metallic work, might also be used.

* Narrative contained in letter dated 14th July 1838.

which, descending into a deep valley, and crossing it, rise again to the hill on which our house is situated. It is carried up to the very top of the building, and is there deposited in a *lead* cistern, from which some leaden pipes lead it down to the butler's pantry and the place from which we used to get our drinking water. The kitchens, &c., were supplied from the iron pipes before the water ascended to the top of the house. The house is very large, and the roof is flat. The cistern occupies a large space on the top, and is of course exposed in its full extent to the air.

"Professor —— detected lead in the two taps *leading from the cistern;* the other two he declared to be perfectly free from it. The bottles were simply labelled with initial letters. We were very incredulous, and, on expressing our disbelief, he asked us to send the water again differently labelled. The result was the same as before. We immediately removed the filter to one of the kitchen pipes [the water from] which had been declared free from lead. In a short time Mr B. was decidedly better: he looked very much better, his colour clearer, and his whole appearance improved; but he said he still felt occasionally some touches of his old complaint, although nothing in comparison to what he formerly endured. He has gone to Glasgow for the holidays, and wrote to me the other day that 'he ignores the very name of indigestion.'"

In a subsequent letter,* explanatory of some details, the same lady informs me:—"We consider the water *hard*, although, as I mentioned before, there is a *soft* spring mingled with the *hard* one. . . . . Lead was found in this *hard* water *after* it had been conveyed into a leaden cistern and distributed thence in leaden pipes through part of the house. *No trace of lead* was found in the same water, which was also in the house, but flowed through *iron* pipes only."

According to the analysis of Professor ——, the water drawn before it entered leaden pipes or cisterns contained of

Organic matter, per gallon, . .	3·50 grs.
Inorganic, " . . .	11·37
	14·87

* Of date 2d August 1858.

The latter consisted of the following salts:—

1. Potash.—*a*. Chloride—very small quantity.
   b. Sulphate—a trace.
2. Lime.—*a*. Sulphate—rather abundant.
   b. Carbonate—small quantity.
3. Magnesia.—*a*. Chloride—rather abundant.
   b. Sulphate—small quantity.
   c. Carbonate—small quantity.
4. Iron.—*a*. Carbonate—very small quantity.
5. Ammonia.—*a*. Nitrate—a trace.*

The analyst designates the water "very soft," and reports it as fit for all domestic purposes, in the absence of lead; while Mrs B., in her letters above quoted, repeatedly speaks of it as *hard!* It is now well known that the quality of hardness in water does not depend merely on the *amount* of its saline ingredients, but chiefly on their *kind* or *nature*. Professor Graham points out that some London waters are *soft*, though containing as much as $\frac{1}{17\frac{1}{2}0}$th of solid matter.†

* Date of analysis, October 10, 1857.

† At my request, two beer bottles full of the waters in question,—the one specimen (No. 1) drawn from lead pipes issuing from a lead cistern containing the water, and the other (No. 2) taken from iron pipes, the water not having in this case been at all contained in, and not having passed through, leaden vessels, were kindly sent to me for analysis in the beginning of August last; but I was unable to overtake their examination till 20th September. My results were as follows:—No. 1 had, on drawing the cork, a distinct smell of sulphuretted hydrogen; and the water contained a quantity of brown flocculent matter, apparently derived from the cork. A stream of sulphuretted hydrogen was passed through the water, both in its original state, as received, and after it had been greatly concentrated by evaporation. Not a trace of lead was discovered by this or any other process applied; neither was iron detected by the rough qualitative methods employed. There was a small proportion of sediment on evaporation. Carbonates were comparatively abundant; chlorides and sulphates in very small quantity: lime and magnesia were plentiful, the former especially.—No. 2 also had a slight smell of sulphuretted hydrogen. The water gave precisely the same reactions as No. 1, except that the sulphates and carbonates were rather more abundant.

It will be observed that there is some discrepancy between the analysis originally made by an English chemist and my own results as just given. This is not to be wondered at, when we consider the changes in the composition of waters produced by season, rainfall, drought, &c. A fall of rain, for instance, must greatly modify the composition of waters, especially if they are derived from the surface drainage of an agricultural district. The tables furnished by Dr R. D. Thomson of St Thomas' Hospital, London, and published in the Reports of the Registrar-General of Births, Deaths, &c., show distinctly

I was recently informed by a friend in London, who formerly lived in Brompton, that he constantly or repeatedly suffered from anomalous stomach complaints while he resided there, and which he attributed solely to the water with which his house was supplied being contaminated with lead; of this, however, he had no chemical proof. The water he described as hard: it was supplied by one of the London water companies,—the water furnished by all of which contains, in comparison with the waters of Edinburgh or Aberdeen, large quantities of solid or saline constituents. He only got rid of his symptoms by removal to Clapham, where the water supply is derived from a garden-well, sunk in the gravel of the London basin. This water also is described as hard; but it is not contained in leaden cisterns, nor does it pass through leaden pipes. The same gentleman, who is connected with the Metropolitan Board of Works in London, and who has an ample experience of such matters, informs me that such cases are very common in London,—so common, indeed, that he laughed at the idea of hard waters protecting lead.

I will now briefly review the various theories or explanations that have been, or that may be, advanced to account for the action of spring waters, which are generally more or less hard, upon lead. They may be tabulated as follows:—

1. Unusually small quantity of
   a. The neutral salts generally, or in the aggregate.
   b. Particular neutral salts, such as the carbonates and sulphates.
2. Unusually large proportion of carbonic acid in the water.
3. The presence of acids derivable from decaying vegetable or animal matter.
4. Presence of foreign bodies or accidental impurities.
5. Presence of nitrite of ammonia.
6. Presence of atmospheric air in the water, perhaps in unusual amount.
7. Use of leaden covers to cisterns.

the difference in the composition, from month to month, of the same waters, supplied by the same metropolitan water companies, and derived from the same sources. Dr Medlock remarks,—"It not unfrequently happens that a water which, at one season of the year, acts vigorously upon lead, has no action upon it at other seasons." (*Op. citat. p.* 33.)

8. Long exposure of still water to the same surface of metal.
9. Great extent of surface exposed.
10. Galvanic action from the contact of different metals, or of different qualities of the same metal, under water.
11. Chemical or electro-chemical causes, with which we are at present unacquainted.

With a knowledge of the statements of Professor Christison as to the power of the neutral salts generally to protect lead from the corrosive action of water, it is unnecessary to illustrate the first half of the first proposition or cause above tabuted, because a water deficient in such salts approaches the characters of rain or distilled water. And further, with a knowledge of what has been determined in regard to the different protective powers of particular neutral salts, it is easy to comprehend how a water containing abundance of chlorides, but a minimum of sulphates and carbonates, should act upon lead nearly as readily and rapidly as if there were an absolute deficiency of all neutral salts.

Professor Daniell says that *all* waters will corrode lead if they contain *excess of carbonic acid*. "My friend, Professor Daniell," says Dr Pareira, "informs me that he has found lead in the well-water obtained at Norwood. The water is *very hard* (that is, holds a large quantity of sulphate of lime in solution), and contains much free carbonic acid. It is the latter ingredient apparently which holds the lead in solution; for, by boiling, the whole of the lead is precipitated. The water is raised from the well by a leaden pump, to which is (?) attached a few feet of leaden pipe. Professor Daniell's attention was directed to the subject in consequence of the occurrence of several cases of lead colic in the neighbourhood of his residence at Norwood."* Professor Taylor opposes the statement, that carbonate of lead is at all soluble in water charged with carbonic acid. "It has been supposed," says he, "that carbonic acid contained in water would partially dissolve and suspend the carbonate in it; but in saturating water with carbonic acid, over finely-divided carbonate of lead, it

* A Treatise on Food and Diet. By Jonathan Pereira, M.D., &c. P. 96. London, 1843. See also Morson in Pharmaceutical Journal, November 1, 1842.

was not found, on filtration, that any perceptible portion had been dissolved."* "Natural water," says Professor Fownes, "highly charged with carbonic acid, *cannot under any circumstances*, be kept in lead or passed through lead pipes with safety; the carbonate, though very insoluble in pure water, being slightly soluble in water containing carbonic acid."†

It has been supposed, and with every prospect of the supposition being correct, that the presence of decaying leaves and similar organic matters, whether of vegetable or animal origin, may favour the corrosion of lead by the development of various *acids* due to the gradual decomposition of such organic matters. Pareira mentions a case in which a fragment of mortar appears to have aided or determined the corrosive action of the water.

"The presence of *nitrite* of ammonia in water," says Dr Medlock, "I have clearly established by the most conclusive experiments; and it is to the presence of this substance, both in distilled waters and waters selected for domestic use, that the action of such waters on lead is due. The nitrous acid attacks the lead and forms the soluble *nitrite* of lead, which, by exposure to air, combines with carbonic acid, and precipitates the oxide of lead in the form of insoluble carbonate of lead, setting free the nitrous acid again to attack and dissolve a fresh portion of lead."‡

It has been suggested that the presence of an unusual amount of atmospheric air *in water* would favour or facilitate its action on lead. Doubtless it would do so were it proved to exist, but this cannot be an important element or agent in the action in many cases. The presence of air *externally to* the water is as much an essential to the action as is that of the water itself; for, unless there is free exposure to the air, oxidation and the subsequent changes in the metal will not proceed. Water is well known to be capable of holding in solution a considerable proportion of air and other gases. Rain-water, remarks Professor Christison, generally contains 2½ per

* On Poisons. London, 1848. P. 450. "Water Poisoned by Lead."

† Manual of Elementary Chemistry. London, 1856. P. 333.

‡ On the Action of Certain Waters upon Lead. Record of Pharmacy and Therapeutics, Part II. London, 1857. P. 34.

cent. of its bulk of air, in which the proportion of oxygen gas is so high as 32 per cent. In water from freshly-melted snow the proportion of oxygen is 34·8 per cent., according to the observations of Gay-Lussac and Humboldt, while the oxygen in atmospheric air does not exceed 21 per cent.* Professor Christison has also pointed out that distilled water deprived of its gases by ebullition, and excluded from contact with the air, has no action on lead.†

The use of leaden covers to cisterns is fraught with great danger, and fortunately is comparatively seldom resorted to by plumbers. If cisterns are covered at all, probably the safest material is wood. However impure or hard a water is, however saturated with sulphates and carbonates, and however free from plumbeous impregnation, the moisture which results from its evaporation, and which condenses on the leaden cover, is pure or distilled water. Hydrated oxide and carbonate are produced in the ordinary way; these gradually encrust the lead, and by-and-by scales or fragments fall off and drop into the subnatant water, in which they are partly dissolved, partly held in a state of suspension.

Time is a most important element or condition in the action of water on lead. The mere flowing of water through leaden pipes, and the standing of water for a long period in leaden cisterns, are two very opposite conditions. Accordingly, it is found that the same water which will cause no perceptible change on lead from short contact, will produce a copious deposit of oxide and carbonate on standing for some weeks or months. Professor Christison remarks this in regard to Edinburgh water, which contracts no material impregnation after a few days' contact. A cistern in his laboratory in the University of Edinburgh was left undisturbed for some five or six months with a few inches of water in it. After the lapse of this period, he found "so large a quantity of pearly crystals lying loose on the cistern, and diffused through the water, that, when the whole was shaken up and transferred to a glass vessel, the water appeared quite opaque."‡ I believe

* Trans. Roy. Soc. Edin., Vol. 15, Part II., p. 271.

† Treatise on Poisons, 1835, p. 476.

‡ Treatise on Poisons, 3d edit. 1835, p. 491.

that such is the action of Edinburgh water,—a water containing about 12 grains per gallon of solid matter,* that in the course of ten days or a fortnight, while at rest in contact with lead, it will dissolve the metal to the extent of about $\frac{1}{10}$th of a grain at least per gallon. Professor Christison again remarks,—"Neither ought ordinary terrestrial waters to be *kept very long in leaden vessels;* because the same changes, though not appreciable in a few days or weeks, are nevertheless accomplished in the course of time."† The late Dr Thomson of Glasgow, says, when he lived in Edinburgh, he "could always detect a minute trace of lead suspended in the water, which at that time was brought six miles in leaden pipes."‡

An equally important element in the action is perhaps the *extent of surface exposed.* It not unfrequently happens that experiments on the small scale give erroneous, and consequently dangerous, results, merely because the surface exposed was too small and the time allowed too short. Professor Christison gives an apt illustration. A gentleman in Dumfriesshire had resolved to introduce into his house, through leaden pipes, the water of a spring some three-quarters of a mile distant. But, with a view to ascertain whether it was quite safe to do so, the usual experiment was made, of placing a few freshly-scraped or polished leaden rods in contact with the water for a few days. At the end of a fortnight, the result was imperceptible; from which it was forthwith concluded that this water might be conveyed through lead pipes and stand in lead cisterns with all safety.§ The works were immediately

* As a comparative statement, I may here mention that, in round numbers, the water of Glasgow,—supplied from Loch Katrine,—contains from 1 to 2 grains per gallon of solid matter; that of Aberdeen 2 to 4; and that of London from 15 to 30!

† Dispensatory, 2d edit. 1848. Art. *Plumbum.*

‡ Christison's Treatise on Poisons, 1835, p. 487.

§ "Water, which tarnishes polished lead when left at rest upon it in a glass vessel for a few hours cannot be safely transmitted through lead pipes without certain precautions. Conversely—*it is probable, though not yet proved,* that if polished lead remain untarnished, or nearly so, for twenty-four hours in a glass of water, the water may be safely conducted through lead pipes."* Taylor,

* Christison, Trans. Roy. Soc. Edinb., vol. xviii., part ii., p. 271.

carried out; but the results were as dangerous as they were unexpected. The water was soon found to be impregnated with lead to such a degree as to be opalescent. Analysis showed the water to have been extremely pure, containing not more than $\frac{1}{22\,000}$th of saline matters, which consisted mainly of sulphates, chlorides, and carbonates. Here the unintentional experiment with 784 square feet of lead produced very opposite results from that with a few inches!*

Galvanic action in connection with the corrosion of lead seems to me to be a most hopeful field of research. It is generally called into requisition to explain all anomalous cases of the action of hard waters on lead. But this is making too much of it. I have no doubt that it exerts a powerful influence in many cases, but it has yet to be distinctly shown in what cases, how and under what circumstances, it operates. The subject is by no means new. Dr Paris long ago drew attention to it, and it has also been taken up by Professors Christison and Pareira; but it is still, to say the least of it, very imperfectly understood. I would therefore strongly recommend it to the study of chemists and electricians. The contact of other metals with lead, the use of solder, the mere existence of impurities or inequalities in the lead, seem to determine galvanic action; in which event the lead "becomes more susceptible of, and exposed to, the agency of electro-positive elements, among which are alkalis and alkaline earths, and these exert considerable solvent power over it."† "Galvanism," says Professor Christison, "is a most important co-operating agent, or rather, perhaps, it should be considered a distinct power, for it acts with energy where water alone acts least,—viz., where there is saline matter in solution, because then a gal-

too, regards the freshly-scraped piece of lead as a good test of the absolute purity of distilled water; and, conversely, of the liability of water to poisonous impregnation with lead. If the fresh metallic surface, says he, remain bright after some days, or only acquire a faint incrustation of sulphate, and if sulphuretted hydrogen does not give a brown tint to the water, there is but little danger of its becoming poisoned with lead.*

* Treatise on Poisons, 3d edit. 1835, p. 489.

† Pareira on Food and Diet, p. 97, and Brande's Dict. of Mat. Med. and Pract. Pharmacy, p. 80. London, 1839.

* On Poisons, London, 1848, p. 450—"Water poisoned by lead."

vanic current of greater force is excited. . . . Even inequalities in the composition of the lead may have the same effect. Sheet-lead, long exposed to air or water, is sometimes corroded in particular spots; and these will always be found in the neighbourhood of parts of the metal differing in colour, hardness, or texture, from the general mass. This, however, is a *mere supposition;* but it affords ready explanation of the corrosion. Similar effects may arise simply from fragments of other metals lying long in contact with lead. . . . *I have no doubt* that many of the instances of unusually rapid corrosion of lead by water,—such as that mentioned by Dr Wall, are really owing not to the simple action of water, but to galvanic action, excited obscurely in one or other of the ways now mentioned."*

In the course of a lecture on "Public Health," delivered in the City Hall, Perth, on 4th January last, Dr Stirling of Perth made the following statement in regard to the influence of galvanic agency in aiding or determining the corrosive ac-

* Treatise on Poisons, pp. 494–5.

In the discussion following the reading of this paper at the British Association, several interesting illustrations were given of corrosive action on lead and copper, attributable, or supposed to be attributable, to galvanic action. For instance (I quote from a Leeds newspaper), "a member said that when copper sheets from different factories were joined together to cover the bottoms of ships, strong galvanic action was set up when they were immersed in sea-water. When all the sheets on the bottom of the ship were of the same kind of copper, the corrosion was very slight. As lead was affected in a similar manner, the sheets of that metal used for lining cisterns *ought always to be of one kind*."

"Dr Edwards attributed the different action of the same water on different lead cisterns to the fact that some sheets of that metal were made from old lead, containing an admixture of solder, which caused it to be corroded by galvanic action. It was shown that thirty or forty years ago, cisterns were not so much acted on by water as now; and it was probable that this was owing to the fact, that the silver in lead was now extracted, whereas formerly it was not worth while to do so. The long corroded streaks which were often observed in sheet-lead were caused by galvanic action set up by small portions of tin which had been attached to old lead."

"The Rev. W. V. Harcourt could not understand how the presence of silver in lead could affect it much, considering the electrical relations the two metals bore to each other. In his opinion, it was *advisable to use leaden vessels for holding water as little as possible; for it was clear that health was in constant danger from it.*"

tion of various waters, containing saline matters, on lead:—"Having compared the effect produced on lead rods immersed in solutions of various neutral salts, in distilled water, with that produced on similar rods in contact with copper wire, immersed in solutions of the same salts, and of the same strength, I am led to the following conclusions, viz.—1. *That galvanic action is a most powerful agent in promoting the corrosive action of certain waters upon lead;* 2. That, in water containing a small proportion of certain feebly protective salts, galvanic action is slightly, if at all, produced; 3. That, in water containing the same salts in so large quantity as to be considered protective to the metal, galvanic action is so powerfully induced as to render the water, instead of innocuous, nearly as corrosive as the very weak solution; 4. That, in water containing certain powerfully protective salts, galvanic action is sufficiently produced to render it doubtful whether any saline solution can altogether protect lead from corrosive action, when, from the nature of the containing vessel, or other cause, galvanic currents may be induced."

Lastly, there are perchance other chemical or electro-chemical causes, whose action is presently unknown or imperfectly understood. These I leave with confidence for investigation in the hands of the Chemical Section of the British Association; for on such causes or their action I am not prepared to throw any light.

I firmly believe that cases of lead poisoning on the small scale, or rather, I should say, in a minor degree, are constantly occurring in all our large towns from the plumbeous impregnation of drinking-waters. The medical man is often extremely puzzled to account for certain anomalous symptoms in his patients; and he puzzles himself in vain, until he at last bethinks himself of the assistance of the chemist, who discovers lead in the drinking-water used. Attacks of lead colic—isolated or endemic—are frequently the first circumstance to attract attention to the state of the water supply. On talking over the matter with medical men in London and elsewhere, I find the suspicion strong, though they are seldom in a position to prove indubitably the correctness of their suspicion, that many obscure cases of colic and other intes-

tinal affections, as well as of paralysis of the nature of lead palsy,—sometimes going on to a fatal issue,—are really due to plumbeous impregnation of drinking-waters; and under so many different circumstances do such cases occur, that, in a large proportion at least, the water so impregnated must be *hard* in the sense in which I have used that term. In all cases of obscure colicky or paralytic complaints,—especially if it be found that several persons are simultaneously affected,—the condition of the water supply should be diligently inquired into, with a special view to the presence in it of lead. Dr Roberton of Manchester remarks,—"What I would here advert to is the custom, so general in England, of using lead cisterns for storing water, especially where, as in many of our towns, the supply is intermittent. Such cistern water is *always tainted with lead*, not in quantity, it may be, to produce the distinguishing symptoms of lead poisoning, but enough to affect very injuriously the stomach and nervous system of persons habitually using it."*

For the benefit of those who may be desirous of following up the subject of the foregoing remarks, I beg to append, in a collected form, a few bibliographical notes:—

Professor Christison of Edinburgh:
1. Treatise on Poisons. 3d edit. 1835. Edinburgh.
2. Dispensatory. 2d edit. Edinburgh, 1848.
3. Paper in Transactions of Royal Society of Edinburgh. Vol. XV., Part II., 1842.

Professor Pareira:
1. A Treatise on Food and Diet. London, 1843.
2. Elements of Materia Medica and Therapeutics. 3d edit. London, 1849.

Professor Taylor:
1. On Poisons. London, 1848.
2. Guy's Hospital Reports. No. VI., O.S.

Dr Wall of Worcester: Transactions of London College of Physicians. II. 400.

Dr Scudamore: Analysis of the Water of Tunbridge Wells. 1816.

* A Few Additional Suggestions with a view to the Improvement of Hospitals. By Mr John Brotherton. [Reprinted from the Transactions of the Manchester Statistical Society. Read March 31, 1858.]

Dr Thomson of Glasgow in Appendix to above. Also in Edinburgh Medical and Surgical Journal, Vol. XII.

Dr Lambe of Warwick: Researches into the Properties of Spring Waters. 1803.

Dr Yeats: Hints on a Mode of Procuring Soft Water at Tunbridge. Journal of Science, XIV. 352.

Vitruvius: De Architectura. L. VIII., c. 7. Quot modis ducantur aquæ. Ed. Dan. Barbari, 1567, pp. 262–5.

Galen: De Medic. secundum locos. LVII.

Wood and Bache: The Dispensatory of the United States of America. 3d edit. Philadelphia, 1836.

Professor Graham: Elements of Chemistry. 1842: and Vol. II. London, 1858.

R. Phillips, in Report from the Select Committee of the House of Lords appointed to inquire into the Supply of Water to the Metropolis. 1840.

Dr Bostock, in Report of the Commissioners appointed to inquire into the state of the Supply of Water in the Metropolis. 1828.

Brande's Dictionary of Materia Medica and Practical Pharmacy. London, 1839.

Morson in Pharmaceutical Journal. November 1, 1842.

Mèrat: De la Colique Métallique.

Rosier: Observations sur la Physique. XIII. 145.

Beck: Elements of Medical Jurisprudence. London, 1836.

Turner's Elements of Chemistry. By Liebig and Gregory. London, 1847.

Dr Medlock: "On the Action of certain Waters upon Lead:" Record of Pharmacy and Therapeutics of General Apothecaries' Company, London. Part 2. 1857.

PAMPHLETS

19



19