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Tourelle — Blutungen aus der Nabels
Hanley — Diabete Sucre
Hinkelbein — Uebergang des Chlor-N
in den Harn.

SHIP FEVER,

SO CALLED; ITS

HISTORY, NATURE, AND BEST TREATMENT.

THE

FISKE FUND PRIZE DISSERTATION FOR 1849.

BY

HENRY GRAFTON CLARK, M. D.

MEMBER OF THE BOSTON SOCIETY FOR MEDICAL IMPROVEMENT.

"PER ARDUA."

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TO

THE JOINT SPECIAL COMMITTEE

Of the City Council of Boston,

ON THE DEER ISLAND HOSPITAL,

For 1847:

THIS DISSERTATION,

(THE RESULT OF OBSERVATIONS MADE DURING A BRIEF TERM OF SERVICE AS THE VISITING PHYSICIAN OF THAT ESTABLISHMENT,)

IS GRATEFULLY INSCRIBED,

BY THEIR OBLIGED FRIEND

HENRY G. CLARK.

A DISSERTATION

ON

SHIP FEVER, OR BRITISH TYPHUS.

The history of Ship Fever, as it has been seen on board emigrant ships, in the quarantine hospitals, and among emigrants newly arrived from Europe, is interesting and peculiar. It is very clearly different from any fever which has hitherto been known in this country. Its diagnostic characteristics, in all well marked cases, are distinct and unmistakeable. It is, in the opinion of the writer, under the various titles of Jail, Hospital or Camp Fever,* Putrid Malignant Fever,† Putrid Continual Fever,‡ Petechial Fever,‖ Febris Cancerum,§ Maculated Typhus, and English or Irish Typhus, the identical disease known recently and popularly, under the name of Ship Fever.

* Howard and Pringle. † Huxham. ‡ Dr. MacBride.
‖ Dr. Darwin. § Drs. Cullen and Vaughn.

Taking it for granted, then, that the disease is thus sufficiently identified, and that it will be recognized if it should be occasionally referred to by its principal synonyms; we shall content ourselves by considering, in as concise a manner as possible, its relations to, and points of correspondence with, the only disease with which it is sometimes confounded, and for which, without due consideration, it might possibly be mistaken. This is, of course, *Typhoid Fever.*

Typhoid Fever has been amply illustrated, and most ably described, by the most distinguished French and American writers; among whom, in France, Andral, Louis and Chomel take the first rank, and in this country, *our* "Louis," Dr. James Jackson, and Dr. Gerhard, of Philadelphia.

This part of the necessary foundation for an opinion on the two diseases, has been laid broadly and well; and nothing that could be said here would add a rush-light to the strong and vivid picture, whose outlines, and whose lights and shadows, have been so faithfully drawn.

We have remarked that the two diseases have been, and are likely to be, confounded with each other. By whom? Certainly not by those whose experience has

shown them *both diseases*, or who have ever interrogated the truthful pages of nature. But some of the most distinguished writers and practitioners of the old world, especially in England and Ireland, have not only seemed to mistake the distinguishing characteristics of the two diseases, but they have stoutly denied them a distinct and independent existence.

This is a most remarkable fact, and would be unaccountable, perhaps, did we not know that the *typhoid* fever of France and the United States very rarely occurs in Great Britain, in comparison with the immense number of cases of true *typhus*, which are constantly seen there. The truth is, that the typhoid fever cases have often been either entirely overlooked, or considered as mere varieties of typhus; and nothing but the adoption of the French method of observation in Great Britain will ever, it is likely, lead to a general admission, in that country, of a doctrine really so susceptible of satisfactory demonstration.

The opinions of some most sensible physicians in this country, also, who do not fully recognize the distinctions between the two diseases, may have been derived from the sometimes prejudiced accounts of them from across the water; for there can be no

doubt that many a hot controversy, which has been waged between the French and English writers on fever in times past, has had its foundation and support, more in *national* prejudices and jealousies, than in any real differences upon which the combatants honestly, and for good reasons, differed.

But are there any real distinctions between typhoid and typhus, which can be defined, recognized, or made the basis of any variety in their treatment?

That they differ widely, and that these differences are as great as between any two of the exanthems, or between any diseases which have a few symptoms in common, can be easily proved. And although the identity of the two diseases is insisted on by some distinguished authorities;* and the distinctions between them are not recognized by so able an author as *Dr. Tweedie*, we think any careful observer of the symptoms, the course of the disease, or the anatomical lesions of either, would be satisfied beyond the possibility of a doubt of the truth of this position.

* M. De Claubry, in an essay which received the prize of the Royal Academy of Medicine, says, "There are no means of distinguishing typhus from typhoid fever, in relation either to the lesions, or the symptoms of the two diseases."

It is not intended to go into any extended examination of this question, but a statement of the chief and leading points in which the typhoid and typhus fevers differ from each other, may not be inappropriate. They differ in their access, their progress, and their termination.

Typhoid fever is slow and insidious in its attack; often of unknown origin; only contagious in a modified degree, and under certain circumstances; not infectious; there is generally diarrhœa, and often hemorrhage from the bowels; epistaxis; its diagnostic *tâche*, consisting of a sparsely scattered pink eruption, is chiefly confined to the abdomen and lower part of the chest, and *disappears* on pressure. The duration of the disease is from two to twelve weeks; the average being about three weeks. An ulceration or inflammation of Peyer's glands is *always found* on post-mortem examination. It attacks subjects in *all conditions* of life, and is *not* prevented by any attention to cleanliness and ventilation.

Typhus fever, on the other hand, is of sudden and violent access, often seizing the patient *instantly* upon his exposure to the exciting cause; it originates from well known causes; is confined to those exposed to

contagion from the sick, or from exposure in a bad atmosphere, and filthy and badly situated tenements, crowded with animal exhalations; — it is infectious; — and in a majority of cases is unaccompanied with diarrhœa.* The eruption is very abundant, often being sprinkled over the trunk, head, and limbs. It is of dirty red color, does not disappear on pressure, and frequently, as the disease progresses, become of a dark brown, which sometimes remains even after death. The eyes are dull and suffused. The duration of the fever is about fourteen days. There is *no* inflammation of Peyer's or Brufner's glands; although, at the same time, the whole neighboring intestine is often seriously affected.

Finally, the secondary affections of the absorbent system, and the peculiar ulceration of the large intestines, (the cause of a most fatal hemorrhagic diarrhœa,) are worthy of especial notice as common sequelæ to Ship Fever. They do not occur in typhoid fever.† In fact, the distinctions must be so obvious that the diagnosis of a well marked case ought never to be doubtful.

* This applies, of course, only to the acute form of the disease.

† Dr. Gerhard's "Researches on Fever" contains an elaborate argument on this subject.

Typhus fever was first known in Europe by some of the names ascribed to it by various of the older physicians; such as "Jail," or "Camp Fever," &c. &c. It has been well described by Pringle, and is spoken of by Howard as occurring frequently in the jails and court-houses, &c. It is peculiarly the disease of *misery*. It follows in the march of armies, and hovers with its ill-boding wings over their encampments. It enters the damp, crowded and dirty dungeon; it is familiar in the hovels and ill-drained and ventilated houses of the poor and wretched. It takes passage with the poor emigrant, who seeks on our hospitable shore an asylum from his woes; it wastes his strength on the long passage, and at last, with its fiend-like gripe, thrusts him down into the deep and sorrowful ocean, or only spares him from this, that he may find but a "hospitable grave" upon the shores of the country of his most ardent hopes and expectations.

The treatises of Drs. Armstrong, and "Smith and Tweedie," are so well known as to make it almost presumption to say, that, possibly from a want of personal familiarity with typhoid fever, it would seem to have been the fact, that some cases of

this disease have thrust themselves *unnoticed* among the great mass of those which they were in the habit of observing more habitually. So that these exceptional and rare cases have undoubtedly led them into the error of recognizing some of the symptoms which belong *solely* to typhoid fever, as but the occasional and accidental attributes of typhus, and as such, influencing, in an unfavorable manner, the accuracy of their plans of treatment. It is therefore to be regretted, that the labors of such distinguished and able men should be deprived of so much of their value to those whose own personal observation has not furnished them the means of correcting, by a frequent reference to nature herself, the false lights which have been thus inadvertently thrown upon the disease.

Although there have been occasional epidemics of typhus in this country, a most excellent history of one of them having been given to the profession by Dr. Gerhard, of Philadelphia, yet the disease is *indigenous to Great Britain.* We certainly, therefore, are fairly entitled to complain somewhat, if they have not furnished us with the most accurate description, and the *best method of treating* an exotic, for which, al-

though we can have had no great desire to see transplanted to this country, we have, nevertheless, *paid for very dearly.*

It is quite curious to observe, with how much warmth and pertinacity the various writers upon the two fevers, undertake to compel the most opposite and conflicting symptoms of each to lie quietly together upon the procrustean bed of their own opinions. Witness the Memoir of Gaultier de Claubry, referred to on another page, in which he undertakes to demonstrate the identity of the typhoid fever of Paris, with the jail, army, and camp fevers of the continent of Europe. On the other hand, Mons. Landouzy, in a most interesting and valuable article upon an epidemic typhus which occurred at Rheims in 1839, describes it as exhibiting all the most remarkable symptoms which De Claubry admits to have been present in the camp fevers which he witnessed. But it would be wrong not to allow M. Landouzy to exhibit himself in the very truly professional and philosophical spirit with which he refers to this point. "If," says he,* "in all future epidemics of the typhus

* Archives Générales de Médécine, Janvier et Feb. 1842.

of camps, of jails, of hospitals, &c. we find, as in that at Rheims, complete absence of disease of the spleen, and great differences between the symptoms and those of typhoid fever, we must confine ourselves to the conclusion that typhus and typhoid fever are analogous, and not identical diseases. If, on the contrary, we find in one epidemic, that diarrhœa is absent, in another, the petechial eruption, in another, the rose spots, and so on, we must conclude that these differences depend only upon variations in the action of the epidemic cause, and that the disease is, in its nature and essence, identical with typhoid fever."

It is to be hoped that the spirit of philosophy and truth which animated M. Landouzy, may be infused more fully into the writers and observers of those diseases. This is especially to be desired, because there is every reason to fear, that, with the present rush of emigrants to this country, from England and Ireland, the disease which has until recently been an exotic, will soon become domesticated among us, and ever loiter round, or fix itself permanently in the purlieus of all our great maritime cities, if it do not follow the great avenues of travel into the interior, especially the manufacturing towns. So that the smallest con-

tributions to the literature, or any practical suggestions upon the treatment of ship fever, may have a value, and be worthy of a consideration to which they would not otherwise be entitled.

It seems hardly necessary to follow the histories of the various epidemics of typhus which have desolated, and almost depopulated, whole districts in the old countries; or to go over afresh the horrible relations of the fatal plagues which have more than decimated the inmates of European dungeons, extending oftentimes, with fearful retribution, even to the officers, judges, and jurors of the courts.

Let us examine it more especially as it presents itself to us in the form of *Ship Fever*. Many of the physicians of this part of the country well recollect the feeling of indignation which pervaded the community when the first vessels arrived in our ports, filled with the sick and dying emigrants, who were, before the real cause of the mortality was understood, supposed to have sunk from the want of proper food on the voyage. Leaving their native country in good health, no other cause than starvation was believed to be adequate to produce a mortality, which, in some cases, amounted to a rate equal to that on board

a slave ship. Sporadic, or more properly single and separate cases of ship typhus had been seen in New England, at distant intervals, for some ten or twelve years past; but they were mostly of a mild grade, and generally terminated in recovery; at least such had been the experience of the writer. But the great numbers of vessels arriving here, loaded with emigrants in all conditions of disease and suffering, soon crowded our almshouses, and houses of industry, and our charity hospitals, to suffocation. Nor did the matter stop here; for every receptacle for these cases became a centre of disease, and the actively contagious nature of ship fever became manifest. It was very soon apparent also that the bad atmosphere, resulting from the crowding of so many human beings on board these ships, and their incurably filthy habits on the voyage, had much more to do with the production of the disease, than any accidental deficiency in the quality or quantity of the food, and that it was an evil which was likely to continue at least for some time to come. It was then that independent hospital accommodations were first provided, for the purpose of limiting the disease, if possible, to the emigrants themselves, and in the next place to diminish, by the

increased facilities which these institutions might afford, its very great mortality. It is in these hospitals that the greater part of the experience of the American physicians has been acquired.

We will venture, therefore, as the result of our own observations in a hospital of this kind, to make a few suggestions to those who may hereafter be called upon to treat this disease, particularly in an establishment where considerable numbers may be congregated; and if this paper should be, in this respect, of service to any physician, or be the means of ameliorating the condition of any of the unfortunate subjects of ship fever, we shall be amply repaid, whatever its merit may be considered to be in comparison with the others which will undoubtedly be presented to the intelligent judgment of the "Trustees of the Fiske Fund." The books and treatises now extant, are somewhat deficient in affording information in regard to many little practical details, a deficiency which, it is to be hoped, the proposition of the question at the head of this essay will be the efficient means of remedying in a satisfactory manner. The question has been proposed at a seasonable time, and the information from various sources

which it will elicit, will no doubt be as gratefully received, as it would have been a year or two since by the writer of this article, and as it would be *now*, in the many points in which he feels himself deficient.

In order to consider more fully the nature and treatment of ship fever, let us first define what is meant by the term itself. What group of symptoms collectively, are then necessary to constitute the disease, which would be at once recognized by this name? We will here take the liberty to condense the most admirable definition of it which we find in the excellent work of Dr. Bartlett, on "Fevers."

Typhus fever is an acute affection, occurring at all ages of life; often transmitted directly from one individual to another; frequently sudden in its attacks; attended at its commencement with chills, usually slight, and in many instances, repeated; then, with morbid heat of the skin, in many cases, very intense and pungent; with increased quickness and feebleness of the pulse; with pain in the head, back and limbs; dullness of the mind; drowsiness or stupor; dizziness, deafness or ringing in the ears; morbid sensibility of the skin and muscles on pressure; extreme prostration of muscular strength; dull and stupid expression of

countenance; fuliginous flush of the face; suffusion of the eyes; with loss of appetite and thirst; in grave cases, with a dry, red brown or black and fissured state of this organ; sordes upon the teeth and gums; occasional vomiting, with a constipated or sluggish state of the bowels; the skin of the body and extremities being the seat of an abundant eruption, coming out, in most cases, between the fourth and seventh day of the disease, and declining, at uncertain periods during the second or third week, consisting of small spots, sometimes scattered, and sometimes grouped and confluent, of a dusky, dingy, red color, not elevated above the surrounding surface, and disappearing imperfectly, or not at all, on pressure; the body of the patient giving out a pungent, offensive, and ammoniacal odor; which symptoms differ, very widely, in their duration, in their march, in their severity, and in their combinations, in different cases; several of them being frequently wanting; but enough of them being generally present to characterize the disease; the most constant of which are the loss of strength, the stupor, the suffusion of the eyes, the fuliginous skin, and the dusky, cutaneous eruption; which symptoms may either gradually diminish in severity,

and finally disappear between the seventh and thirtieth day of the disease; or may increase in severity, and terminate in death, between the third and twentieth day from their access, the bodies of patients exhibiting after death no constant pathological changes of any of the organs, but in a considerable number, engorgements of various organs, such as we see in scarlatina, small pox, &c., especially of the brain and its membranes, the blood being generally of a dark color, often fluid, or grumous, the coagula, when found, being soft, and non-fibrinous, and the body running rapidly into decomposition; which disease, thus defined, constitutes a peculiar, individual affection, differing essentially from all others.*

This fever is in its nature an essentially adynamic disease; it is, like most other severe epidemics, a disease of debility, a fact all important to be borne in mind when we come to consider its treatment. The *subjects* of ship fever, too, in the vast majority of cases, are debilitated and broken down, if not before commencing the voyage, at any rate by the confinement in the foul and pestilential atmosphere of an

* Pages 291 and 292.

unventilated steerage, crammed to suffocation with human beings, the habits of many of whom are of the filthiest description. After a visit on board one of these emigrant ships, and a careful examination of her condition, no one would ever after be surprised at the amount of disease generated during a voyage of forty or fifty days. We have seen in the between-decks of vessels of this class, scenes such as we have never witnessed elsewhere. On board one now recollected, and containing between three and four hundred passengers, there were fifty or sixty sick persons in various stages of fever, some with the dysentery, which sometimes follows as a secondary attack, lying in their berths indiscriminately together, adults and children, without the least care or attention to cleanliness, and as we were assured they had done during the continuance of the whole voyage! In another berth were the bodies of two children, who had died within an hour or two of our arrival; and close by them, and almost in contact, the feeble mother, at whose dry and withered breast dragged a miserable and squalid infant. Those who were able, were staggering about with haggard countenances, whose staring eyes and hollow cheeks showed how narrow an escape they had themselves had from

the lonely grave in mid-ocean, which their associates and their relations had long since found. Altogether, with its accumulations of filth, of distress and wretchedness, it was a spectacle, the like of which we would not look upon again.

It is not surprising, then, that from such foci of contagion, the disease should be carried with the emigrants in all directions, as we have seen. Taking its first footing in all the great Atlantic ports, from Canada to New Orleans, it has spread into all the interior towns where there are large numbers of foreigners congregated.

Its extension is limited somewhat by the detention at the quarantine grounds of those who happen to be sick at the moment of the ship's arrival, but it still does infinite mischief by establishing so many new points of departure, and affecting very many of the old residents, especially in the houses of the poor, which are over-full and ill-ventilated. But the poor have not alone suffered; the keepers of the almshouses, which were the first receptacles of the sick, the nurses and the physicians of the hospitals, the clergy and the sisters of charity, have indiscriminately fallen victims to this virulent disease; proving, beyond a doubt, its *contagious nature.*

In the hospital* under our charge, every one of the nurses, who were usually themselves emigrants, were successively attacked, and several of them died. The physician had died, and the only remaining assistant physician and apothecary was sick of the fever, when we first undertook the superintendence of the establishment. Two other gentlemen, one acting as an apothecary, and the other as resident physician, rendered valuable and important services as our associates in its care. The former had, subsequently, a mild attack of fever; but, with this exception, no other person employed on the premises, ~~however,~~ *afterwards took the disease by contagion.* This fact will be worth remembering, when we come to speak of the management of this disease, as showing how far the contagious properties of ship fever can be controlled or diminished.

It is also *infectious.* We have known a considerable number of instances, in which the disease has been propagated, without any question, by chests of clothing, &c., without any personal contact with the sick. This is a point which should not be overlooked. No clothing should be permitted to be removed from the

* At Deer Island, Boston harbor.

region where it has been exposed to the atmosphere of fever, until it has been thoroughly cleansed; and this of course should be done, if possible, by those who have once had the disease. This leads us to remark, that, in all probability, one attack of ship fever, as in the case of the other exanthems, generally precludes another; and although there are well known instances, in which the disease has occurred a second or even a third time, yet as a general rule the protection is complete, and if the secondary cases do sometimes occur, they are in a milder form, and much less to be dreaded. Indeed from a personal familiarity with nearly two thousand cases, we are sure that this is the *rule*, and that second cases are the exceptions to it; for we are quite certain, that we have known more instances of varioloid after small-pox or second attacks of measles and scarlatina, in a smaller number of cases, than of ship fever. It must be understood in this place, that the secondary diseases which occur after fever, and are only appendages to it, are *not* referred to; for these are frequent, and often fatal. The point is, that *second attacks* of the primary fever, after an entire recovery, are exceedingly rare, even with the greatest exposure. It will be seen, that this is of great practical importance in selecting *nurses*.

Again, the disease is very seldom propagated from a single patient in a well ventilated house; so that when patients are in good circumstances, and can be properly nursed, it will not generally be necessary to remove them for the protection of others. In this particular it differs essentially from typhoid fever, which, although its contagious properties may be considered to be much below those of typhus, yet it will often be found, that typhoid fever will persist in spite of the most energetic measures of cleanliness and ventilation. The poison of typhoid fever seems to be of a more subtle character, and decidedly less amenable to any hygienic rules of this sort.

In a large hospital, also, if the *patients*, their clothing and bedding, are kept thoroughly and *constantly clean*, and if the *apartments* are *properly ventilated*, *the disease will not spread*, and the exposure of the attendants is merely nominal. In the hospital* to which allusion has already been made, for the first two months after its opening, all the attendants, without an exception, took the disease; while, after efficient ventilation and cleansing, not a person, with

* Containing about three hundred beds.

the single exception of the assistant physician before mentioned, was affected by it; although, to be sure, many of those who were employed were protected by a *previous attack* of fever. The whole air of the hospital was changed every half hour; the attendants were required to wash the entire body of every patient laboring under the acute stage of fever every morning. In cases in which there was any offensive odor, a little nitrate of lead, (Ledoyen's disinfecting fluid,) or the chloride of soda, was added. All unnecessary articles of clothing were removed from the apartments which were occupied by the sick. Every patient upon entrance was required to be washed, and his or her clothing entirely changed before they were sent to bed in the wards; and it was surprising to see the beneficial changes often immediately produced by this operation, even on the most unpromising cases. The general utility of these measures was shown also by the entire immunity from contagion enjoyed by the various committees and other visitors to the hospitals; none of them ever having been known to contract the disease during the continuance of the epidemic, a period of some six or eight months.

Ship fever, like the other exanthems, is ordinarily

a self-limited disease, its most usual duration being fourteen or fifteen days, the great majority of cases of the primary fever terminating within that period. Nevertheless, so far as our experience goes, at least two thirds of all the deaths occur at a much later period. They are caused by the secondary diseases, which follow after a certain period of partial recovery, so that they may be considered as relapses.

These secondary affections have not received the attention which their importance (with reference to the mortality of the disease) would seem to merit. They may be classed under two principal forms, which, although they sometimes run into each other, are generally quite distinct:

1. General dropsy, which is often accompanied by swelling and sloughing, or suppuration of the parotid and other glands, and occasionally by suffocative œdema of the glottis.
2. A diarrhœa or dysentery, which is usually dependent upon inflammation and ulceration of the ileum and cœcum, and is frequently fatal.

1. The *dropsical affection*, which we have seen in several cases after severe attacks of fever, is scarcely

referred to by authors who have written on fever. It seems to bear the same relation to this fever, that it does to scarlatina, and is accompanied with suppression of urine. We have known it to be fatal in four or five instances, out of about three times that number of cases. In two of them death was caused by the extension of the disease to the glottis, the general state of the patients otherwise precluding any chance of benefit from tracheotomy. In one, death followed extensive sloughing of the parotids; in the others, the patients were apparently exhausted by an obstinate diarrhœa, with which the dropsy was complicated.

Dr. Bartlett speaks of an inflammation of the parotids, but does not make any reference to general dropsy, or to the sloughing of these glands.

2. *Chronic diarrhœa and dysentery.*—This most fatal sequel to ship fever comes on, in most instances, apparently from some error in diet, in the form of a slight diarrhœa, which makes its appearance usually towards the close of convalescence, and after the appetite is pretty fully established. If not arrested in this stage, it commonly goes on to a fatal issue, although we have seen a few recoveries after a very long continuance of the disease in an aggravated

form. The patient has at length frequent discharges of thin yellowish-white, frothy liquid, of very fetid odor, which, as the disease advances, become sanious or purulent; the tongue is flabby and red; the pulse feeble and frequent; and there is great prostration. The pain is not always severe at first, although there may be considerable tenderness of the abdomen. In the latter stages it often becomes intolerable.

We here insert several cases which exhibit the ordinary characteristics of this form of the disease. They will illustrate its pathology more fully than can be done in any other way.*

CASE I.

Catherine Cochlin, single woman, age 24 years, admitted to the hospital Nov. 22, 1847. She was of medium size and stature, of not very robust constitution. When first admitted, she was found to be laboring under a severe attack of maculated typhus, which had then reached the sixth or seventh day. From this attack she recovered, without the occur-

* For the notes of these cases, and for the use (by the engraver) of the original daguerreotypes of the specimens, I am indebted to my accomplished friend Dr. J. B. Upham, of Boston, at that time one of the Resident Physicians at the Island.

rence of any marked phenomena. Towards the close of convalescence, giving way to the cravings of an inordinate appetite, she partook freely of forbidden articles of diet.

As a consequence of this imprudence, diarrhœa supervened Dec. 23d. She sank under it Dec. 30th, on the thirty-eighth day from the original attack.

Autopsy, 12 hours after death. — Externally, emaciation moderate.

Some tympanitis of abdomen. The interior of head was not examined, the autopsy being necessarily made in the evening, after an unusually laborious and depressing attendance in the fever wards of the hospital. Upon removal of integuments, contents of abdomen presented nothing unusual externally. Liver, perfectly normal. Gall-bladder, natural in appearance, and filled with a dark, tenacious, tarry fluid. Stomach, normal externally; contains half a pint of thin, green fluid. Internal lining membrane normal; natural secretion abundant.

Spleen, of natural size and consistence.

Intestines, externally, showed some appearance of discoloration towards lower portion of ileum. Old adhesions of ascending colon to parietes of abdomen.

Mesenteric glands, showed no marks of recent disease, except some slight enlargement in various parts.

Small intestine, pretty generally injected throughout the greater portion of its track. The solitary glands presented no alteration. Duodenum natural. Jejunum, slightly injected, more at its lower part.

Ileum, presented its mucous membranes congested and thickened; for three feet from ileo-cœcal valve this thickening amounted to real hypertrophy, which appeared in the form of transverse ridges, thickly crowded, resembling hypertrophied valvulæ conniventes, from a line to a line and a half or two lines in depth. This hypertrophy was abruptly arrested in a well defined border around Peyer's patches, giving to them a depressed appearance, which at first led us to suppose them ulcerated, or otherwise altered from their natural condition. But on a more careful examination they were found unaffected, and their mucous membrane entire and unaltered.*

Large intestines. About the region of the cœcum, along ascending colon, and first half of transverse colon, the mucous membrane was slightly thickened with occasional patches of ulceration, not extensive or

* See Plate. Figs. 1, 2, and 3.

deep. From about the middle of transverse section, this thickening was more apparent, and the ulcerations more numerous, and deeper. Near the angle of the transverse and descending portions, the thickening was again marked, with hypertrophy also of the muscular coat. Above this point the ulcerations were unusually deep and extensive. Some ulcerations were observed for three or four inches down the rectum.

Along the whole course of the diseased portions, the ulcerations seemed to be confined to the mucous coat. They were irregular in form, and in size varied from a pin's head to that of a split pea, and a few twice that size. They were covered with a foul, sanious exudation, which could easily be removed by the scalpel. There was no appearance of pus in any part.

The remaining contents of the abdomen were natural.

CASE II.

James Warnock, aged 37, in previous good health, was admitted to the hospital Dec. 6, with fever. He had been several days ill.

It was a marked case of maculated typhus, of medium severity. There was no diarrhœa during the

acute stage. The disease was of the average duration.

The patient recovered from the fever with no untoward symptom. He had been transferred to the convalescent ward, and for two or three days had been able to leave his bed and walk about the room. He had a strong appetite, and, contrary to injunctions, partook prematurely, though in moderate quantity, of solid diet. Immediately after (the 2d of January,) he was seized with diarrhœa. He had at first six or eight discharges in the twenty-four hours, — liquid, yellowish, and slimy. After two or three days the stools increased in frequency, deepened in color, were mixed with depraved secretions, became reddish, and finally almost black, and very offensive.

The patient complained but little of pain in the abdomen: there was no tympanitis, and slight tenderness on pressure. His strength failed rapidly, and he died January 11th, at 8, A. M.

Autopsy, 48 hours after death. — Subject well developed, large, muscular. Emaciation not marked. Chest large and full. Abdomen sunken, but not discolored.

Intestines. — Externally there appears some dis-

coloration of duodenum, and upper part of jejunum, more at lower half of ileum, descending colon and rectum. Both small and large intestines contain in every part a grayish, pultaceous fluid, in considerable quantity. A few small collections of fæcal matter, in color and consistence like chocolate, found in lower portion of ileum. Veins of submucous cellular tissue a little engorged in duodenum, and upper part of jejunum; mucous membrane in these portions natural, as also throughout remainder of jejunum and upper half of ileum, from which point injection begins to be marked, accompanied by thickening. This condition of the membrane increases till within about two and a half feet from ileo-cœcal valve, when both the congestion and thickening become strongly marked, appearing here and there, for the space of a few inches in extent, in the form of transverse lines raised from the general surface.* Near the ileo-cœcal valve, to the extent of four inches, this ridged appearance is remarkable — the elevation being from a line to a line and a half in height, and one or two lines in breadth. Within a couple of inches of the cæcum are observed a few points of ulceration, of the size of

* See Plate. Fig. 2.

a pin's head, extending partly through the mucous coat. In but one instance are Peyer's patches visible, it here presenting in a slight degree the shaven-beard appearance, the thickening elsewhere noticed ceasing at the border of the patch, giving it a depressed appearance. The cœcal extremity of colon shows considerable congestion and thickening, with commencing points of ulceration; these appearances increase along the ascending and transverse portions, and are still more marked in descending portion and upper part of rectum. None of the ulcerations in this case exceed in size a split pea; they are confined to the mucous coat. There is no alteration of mesenteric glands. Remaining contents of abdomen normal.

CASE III.

Patrick Lochlin, 25 years of age, was admitted to hospital Oct. 20, being then in the initiatory stage of the fever. His previous health had been good.

The fever was well marked, severe, and protracted. Convalescence was proportionally slow, but complete. The patient had left the wards, and had been engaged in out-door employments for two or three days, when diarrhœa set in. This was on the 12th of

December. The discharges at first amounted to not more than four or five in the twenty-four hours. They were liquid, yellowish, mingled with mucus, not very offensive. Once or twice they were checked by the powders of ipecac. and opium, assisted by the acetate of lead. The abdomen was sunken, somewhat tender on pressure. The symptoms gradually assumed a graver character; prostration became marked; the stools were more frequent, darker in hue, became mingled with depraved mucus and blood, and were very fœtid. For the last four or five days they were of a slaty color, passed almost constantly and involuntarily.

Death occurred on the 13th January. The patient had throughout no tympanitis, and the soreness and tenderness on pressure were not very marked. He made no complaint, but bore upon his face a peculiar expression of anxiety and suffering. His intellect was unaffected.

Autopsy, 4 hours after death. — Subject of medium size, well developed. Much emaciation; chest full, resonant; abdomen sunken, but a thin tissue of fat beneath the skin over chest and abdomen.

Intestines externally appear a little discolored in

duodenum and first part of jejunum; this dark color (opacity) becomes evident again and more marked at beginning of ileum, and increases as we descend the tube; in large intestines it is most apparent in descending colon and upper half of rectum. Duodenum and upper portions of jejunum contain a greenish fluid; ileum and colon moderately filled with a grayish semi-fluid matter, adherent to their walls. Mucous membrane of duodenum is considerably congested; that of upper portion of jejunum is also darker than usual, but presents no marked injection or thickening; texture normal. Valvulæ conniventes in their natural condition. In the upper portion of ileum the dark color and thickening of mucous membrane becomes manifest; these conditions increase as we descend the canal, and are most marked about four feet above ileo-cœcal valve. The thickening along lower two-thirds of ileum assumes the form of rough, prominent ridges, blackened on their summits. These prominences are at first inconsiderable, and separated by an interval of three to six lines; farther on they are increased in number, as also in breadth and height; four feet above ileo-cœcal valve they resemble hypertrophied valvulæ conniventes, being a line apart,

and elevated from one to two lines, hard, some of them serrated on their summits; they continue thus till within two or three inches from the cœcum. The ridges extend completely around the intestine unless intercepted by the patches of Peyer, in which case the elevation terminates abruptly at the border of the patch, and recommences on the opposite side.* The patches are therefore apparently depressed, and are covered with a dark coating of depraved secretion, which is readily scraped off by the scalpel, revealing the membrane beneath entire. At the junction with the cœcum, for the space of three or four inches the mucous surface appears roughened, granulated, with here and there a few thin flakes of a lymph-like substance. Some few of the solitary glands are here visible, raised and somewhat reddened, and interspersed are a few points of ulceration. The cœcum exhibits the solitary glands enlarged, inflamed, surrounded each with a distinct red border, the central point being in some instances ulcerated.† There is also thickening and injection of the mucous lining, with accompanying hypertrophy of the muscular coat.

* See Plate. Figures 1, and 3. Fig. 4.

These conditions observed along the ascending colon, less in its transverse portion, again very marked in descending portion. The interior of large intestine throughout is much thickened and roughened; in the descending colon and upper part of the rectum the mucous coat is extensively ulcerated. No alteration of mesenteric glands. Remaining contents of abdomen normal.*

The morbid appearances noted in the above cases were observed in a greater or less degree in the bodies of all patients dying beyond the sixteenth day from the commencement of the attack, increasing regularly *after* this period, and disappearing entirely in those which were anterior to it. The hypertrophy, however, was not of so marked a character in all.

We should remark here, that after the first autopsy was made, and the particular pathological condition ascertained, extraordinary care was taken of patients during the convalescent stage; no indulgences were allowed, and the mildest diet persisted in, until the convalescence seemed to be complete; common gruel

* See Med. and Surg. Jour. for Jan. and Feb. 1848.

was prohibited, and arrow root, rice-water, sago and the mucilages substituted. This course seemed to be the means of saving many, as the fatal cases diminished materially, and relapses were less frequent.

The principal change in the medication consisted in the use of a solution of nitrate of silver as an injection, and the application of counter stimulants to the abdomen. The nitrate was administered with a glass syringe, to which we sometimes attached Mr. O'Beirne's tube, introducing it thus directly into the colon, and throwing it up from that point. This treatment was apparently beneficial in some cases, although no treatment availed in many others; but it seems to promise as much as any other when combined with the usual opiate remedies.

After all, the careful treatment of convalescents will, without any question, do more to diminish mortality in these cases (by preventing their frequent recurrence) than any treatment can do, after the actual onset of dysenteric symptoms.

There is still another point worth considering in this peculiar secondary affection. Is it not probable that the diarrhœa, shown clearly not to be that of typhoid fever, may have been mistaken for it,

and that some of the cases which have been set down as typhoid (merely on account of the presence of this symptom during life, unverified by a post-mortem examination), were really the sequelæ of typhus?

It is somewhat remarkable, too, that although this diarrhœa is the cause of a large part of all the deaths from typhus, it has so generally escaped observation, or, at the best, only served to confuse the most valuable treatises upon this subject. It is a most distressing disease, and is well worthy of further investigation. The average duration of the primary fever is sixteen days; that of the secondary affection thirteen; and the period at which this manifests itself from the first access of fever, is beyond the forty-fifth day. The average mortality, including those landed in a dying condition, is about nine per cent.

Lesions after death during the acute stage. — These consist, as in other fatal exanthems, chiefly of congestions of the various organs, especially of the membranes of the brain, and of the parenchyma of the lungs. The bronchial tubes are often found much obstructed with frothy and tenacious mucus. The blood in the heart and great vessels was noticed

to be peculiarly dark, fluid, and sizy; its clot large, loose, and easily broken. The lining membrane of the stomach is sometimes seen to be reddened, and dotted with clustered points near its cardiac orifice, but this was not constant. In cases where the urine had been retained, the bladder showed marks of irritation. In the small intestine, the internal surface of the ileum sometimes presented traces of inflammation, but the pathological condition was more generally limited to a decided injection of this part for an extent of three feet or so from the ileo-cœcal valve (without disorganization); and no case was examined in which this was not observed to a certain extent, which varied with the duration of the disease. The same may be said of the large intestines also.*

It must then be considered as established, that ulceration of the small intestines does not take place in the acute form of typhus; but that the diarrhœa which happens as a sequel to it, depends upon ulceration, hypertrophy, or inflammation of the ileum, cæcum, or colon. *Peyer's glands are usually unaffected in any form of ship fever.*

One important symptom which occurs in some of

* See Plate. Fig. 5.

the graver cases, we have not seen mentioned in any treatise. It is, a remarkable retention of urine. At the same time, there is no suppression, but rather an increased secretion; the catheter procuring sometimes from three to four pints daily. It is an unfavorable sign, and occurs mostly in those cases where the nervous system is a good deal affected.

Another characteristic of this fever is the great indifference which the patient manifests in regard to the issue of the disease in his own case, and in those about him; an indifference which is entirely distinct from delirium, and not at all dependent upon it.

Tympanitis occurs oftener than is usually supposed; and although the gravamen of the fever is usually upon the brain and the nervous system, yet there are very few cases in which the abdomen is not more or less disturbed.

The other symptoms are well described by various authors, and have been already enumerated in another part of this dissertation, so that it is not necessary to refer to them here in a more formal manner; but they will be considered incidentally during the discussion of the treatment.

TREATMENT.

The general hygienic principles to be adopted in the treatment of ship fever, have been heretofore stated, especially in regard to the circumstances which exist where large numbers of patients are congregated. Cleanliness, and thorough ventilation, are necessary under all conditions, both for the welfare of the patients themselves, and the safety of their attendants.

It has been shown that the contagious character as well as its severity of attack, is very materially modified by attention to these points, and for the most most philosophical reason; that the disease always develops itself under circumstances in which these conditions are entirely neglected, or absent. So that in this fever, as in all other diseases, the cardinal principle is, to remove the patient from the influence of the inducing cause.

The most common course of treatment adopted in hospital and private practice, in cases in which there was nothing extraordinary, was very nearly as follows. After clearing the digestive canal, the camphor mixture and spirits of nitre, or the liquor ammoniæ acetatis, were ordered during the day, and at night

if there was any restlessness, a full dose of Dover's powder. Demulcent drinks, in quantities to satisfy the appetite. A laxative of oil or magnesia was administered once in two or three days, if there was no dejection without. If there was delirium, accompanied by a hard pulse, the fever mixture of Dr. Stokes was substituted for the other. Ice-water was applied to the head, and sinapisms to the nucha, the ankles, or the abdomen. If the pulse was depressed, brandy or wine were administered in free doses until the delirium subsided. A blister to the back of the neck, proved almost a specific in many cases of this sort, and was often resorted to as an efficient remedy. Delirium, sometimes of a very violent character,* was present in many cases which ultimately recovered, although its absence was of course considered a very favorable circumstance.

There was nothing from which the patients derived more comfort, than from the frequent sponging of the trunk and limbs with water, which was required to

* In one case, the patient leaped through the shut window, in spite of the nurses, carrying the sash away at the same time into the yard below.

be repeated several times a day during the height of the fever.

Local congestions about the chest frequently occurred from the exposure of patients on their way to the hospitals, or during the disembarkation at an inclement season. They were treated mostly by external applications, such as epispastics, blisters, dry cupping, &c. In some cases, the antimonial treatment, combined with local bleeding, was found to be advantageous. But even in those cases which were complicated with severe pneumonia, or bronchitis, it was often necessary to continue the stimulants at the same time.

Bleeding, either by venesection or by leeches, was seldom thought advisable in uncomplicated cases, and in the few instances in which it was resorted to, leeches or cups were always preferred. We think it will very rarely be found useful, if it is not actually injurious; and the testimony of all experienced writers, we believe, is conclusive to the same effect. There are no doubt *epidemics of typhus* in which this rule should not be so rigidly applied; but in ship fever, and the kind of subjects usually affected by it, adherence to it, we are satisfied, is the best

course. Dry cupping often seemed to be of the greatest service, in instances where the loss of even the smallest quantity of blood was contra-indicated.

Stimulants are, without any question, the chief reliance of the physician in a very large proportion of all the cases of ship fever. Wine (for this purpose) has received the almost universal sanction of the profession, and in consequence a great deal of it has been used under our observation, in the shape of whey, or diluted with water; but we must confess, that excepting perhaps its agreeable taste, we should prefer to administer brandy. A large experience with that article, shows that it is efficient in smaller doses, and more likely to be retained upon an irritable stomach than wine. It is more easily obtained in good quality, and is therefore to be preferred for a quarantine hospital, where large quantities must be used. In private practice, the wine may have some preference perhaps. But either of them are of great value in this fever, and they must be administered often, in the most liberal quantities, measuring them in fact only by their effects. We scarcely remember a case in which one or the other of these articles was not resorted to, at some period

of its progress. As in erysipelas, the most furious delirium will often be subdued, by free draughts of wine, or brandy and water, in so short a space of time that the effect seems almost magical. The ordinary dose of brandy administered was about ʒss. to ʒi. and repeated usually every three or four hours.

Camphor, is a very useful accompaniment to the treatment, when the nervous system is much disturbed, and may be given with advantage in doses of from five to fifteen grains.

Quinine is most useful after the acmè of the febrile symptoms is passed, and will be found to be beneficial in preventing the dropsical sequence to the fever, and of the greatest utility upon its occurrence. Its power in this respect has often been tested in the anasarca of scarlatina, especially when combined with diuretics.

Purgatives, although not exactly inadmissible, should, in all cases, be used with the utmost caution, on account, first, of the adynamic character of the disease; especially, on account of the irritability of the mucous membranes of the intestines, and the tendency to severe secondary diarrhœas. A moderate purge of calomel during convalescence may sometimes be required; but castor oil is better for common use.

Some subjects were brought into the hospital in an almost moribund condition, and unable to swallow. In these cases stimulating injections were administered (per anum). Dry heat, by means of hot flannels, bricks, &c., seemed to be of great service in aiding in the restoration of the enfeebled circulation; and some patients recovered who really seemed, when they first arrived, to be in a desperate condition.

Anodynes. Opium, in the form of Dover's powder, was almost universally preferred as an anodyne, excepting during the secondary diarrhœa, when it seemed to irritate the bowels. It was given a great deal, and almost invariably with good effect. It is much better borne during the delirium of typhus, than of typhoid fever. Its sedative effects in most cases were quite direct, and very seldom preceded by any period of excitement. Its use is of course improper, where there is any tendency to coma.

Sulphuric ether. This was used by inhalation in a few cases, with very good effect; and now, that it is more known and its properties better understood, it may become, eventually, a most valuable remedy in the delirium and restlessness of fever, when narcotics would be contra-indicated.

Anti-septic enemas, are sometimes useful, especially if there is much puffing up of the abdomen from flatus; and the common brewer's or baker's yeast is a convenient article for this purpose.

Diet during convalescence. This should be regulated with greatest care, as a slight indiscretion is almost sure to cause a relapse of the fever, or to bring on the fatal diarrhœa. At first, light farinaceous food, milk porridge, rice, &c. are the best articles after discontinuing the simple demulcent drinks; but even these must be allowed in small quantities, and increased very gradually as the patient gathers strength. Broths and soups are not well borne for a long time after the commencement of recovery, and their use should be delayed until the patient is able to take a little exercise about the house. The drink should consist of chamomile tea for breakfast and supper, and a little soda water, or brandy and water, for dinner.

If the weather is suitable, the patient should be allowed to exercise in the open air, as soon as he is able to walk; for nothing else in our experience so rapidly restores the exhausted energies of a convalescent from ship fever. We have often seen patients,

who, when first removed (in the arms of the nurses) from the pestilential atmosphere of a crowded steerage, were entirely unable to help themselves, after a bath, and a few hours' exposure on a sunny hill-side, so much revived as to be able to walk into the hospital without aid.

Let these facts be well considered during the whole course of the treatment; and whatever other luxuries it may be thought proper to refuse, the pure elements of air and water should ever be supplied to them with the same liberal profusion with which they have been created for the use and enjoyment of all living organized beings.

To recapitulate; from an examination of the history and pathology of this fever, we arrive at the following conclusions: —

Ship Fever is identical with the true typhus of Great Britain.

It is not identical with the typhoid fevers of France and New England, but an entirely distinct disease.

It is most fatal in its secondary forms.

And lastly, that its contagious properties may be greatly controlled, if not destroyed, by suitable sanitary measures.

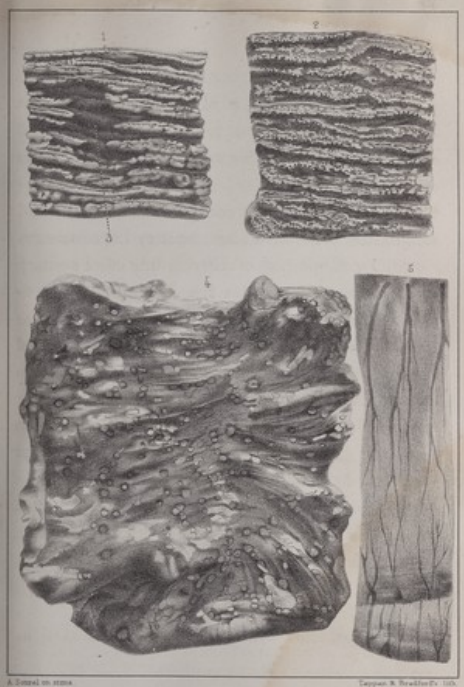
QUESTION OF QUARANTINE.

From the present very advanced state of public opinion, (to produce which, the efforts of medical science have long been directed,) it is evident, that the old systems of quarantine must speedily give way before the progress of the more liberal, humane, and enlightened policy of modern sanitary improvements.

Still, for the purpose of carrying into effect sanitary measures for the dispersion and dilution of the accumulated material of typhus, cholera, and other malignant epidemics; for securing the advantages of temporary medical treatment on shore for those who cannot immediately procure it for themselves; for the prophylactic treatment of those in whom the incubative period of disease, and its consequent power to re-establish itself in new localities, has not yet passed; institutions, similar to this at Deer Island, are admirably adapted, and will be found, doubtless, alike salutary and economical.

THE PLATE.

The lithograph, on the opposite page, shows, with great fidelity, the morbid appearances, which are fully described in the notes of the cases from which the specimens themselves were taken.



1 & 2 Portions of Ileum. — 3. Peyer's patch. — 4. Cœcum, showing the ulcerations of the secondary affection. — 5. Inflamed portion of Colon.

By Bennet Dowler. M.D.

INTRODUCTION.

Among the memorable transactions of humanity, and the great events which have marked its eras and progress, as the catastrophies of the battle field, the downfall of kingdoms, destructive earthquakes, and remarkable appearances in the sidereal heavens, related in ancient history, Epidemics occupy but an obscure place. The slightest notice of yellow fever is no where found among ancient writers, although they have not failed to record, incidentally or directly, the time, place and progress of numerous epidemics, with more or less particularity, so that their characteristics, may, now, after the lapse of so many centuries, be ascertained. It is now nearly three thousand years since the first temple arose in honor of Æsculapius. Four or five centuries later he was worshipped at Rome, where epidemics became both frequent and fatal. Homer opens his great poem by alluding to an epidemic which destroyed dogs, mules and men; another, 430, B. C., most destructive at Athens, was minutely described by Thucydides, himself having suffered from it; an epidemic which fell under the observation of Hippocrates whose treatment of it was reckoned so successful that he was presented with a massive crown of gold, and the highest public honors. Five years later Athens was again visited.

Epidemics B. C.

Rome.

Athens.

Many epidemics prevailed at Rome before our era. In 263 and 212, (at the seige of Syracuse) and in 131, B. C., the Roman and many other nations suffered from pestilential visitations, as mentioned directly or incidentally by ancient authors.

Near the commencement of the Christian era, Celsus—and in the next century Galen—gave the world their learned works on medicine. In the sixth century the plague was general; and in A. D. 565, small-pox was first described in France, as it was in the tenth century by the Arabian physicians, Rhazes and Avicenna. Before the middle of the thirteenth century, medical schools existed at Montpellier and Damascus. The Parisian college of surgery soon followed. Descriptions of scurvy and plica, soon after, were recorded. Books on medicine appeared in greater number, and some new diseases were described in the fourteenth and fifteenth centuries, such as whooping cough, the sweating sickness, and St. Vitus' dance, which later was epidemic upon the Rhine.

Epidemics since the Christian era.

During this long period, so briefly sketched, yellow fever does not appear to have been noticed until the discovery of America by Columbus. Had it prevailed in ancient times, its prominent features, so very remarkable, at least in its advanced stages, would doubtlessly have been recorded.

Omitting on the present occasion its post-Columbian history for about three centuries, in order to reach its advent in New Orleans, in 1796, it is intended not

to give its full history in this city, but to offer the reader a few (out of many) memoranda upon this subject.

Plan of this Essay.

The sketches now submitted to the public are intended to include the obscurest period of the history of yellow fever in New Orleans, from its invasion up to the first third of the present century. Since the spring of 1836, I have been an active participator in, and a vigilant observer and a faithful recorder of, the sanitary history of this city, as I hope more fully to show on some future occasion. Except a view of the current epidemic, (not fairly history, because not yet elapsed, much less analyzed statistically) my voluminous details of all the great epidemics, since 1836, will necessarily be omitted for want of space in this limited memoir, as well as nearly all the data collected for the history of yellow fever for two centuries before its appearance in New Orleans. The waves of oblivion have already rolled over much of the sanitary history of New Orleans from 1796 to 1833. Of the fragments I have picked up, only a few can be given agreeably to my present plan. It is hoped that others, more fortunate, will be able to contribute more in illustration of this obscure though recent portion of sanitary history; otherwise, it will remain a blank forever. The epidemics since that of 1837 inclusive, have received or will receive such historical notice as will save them from oblivion, so that our successors will be able to give them a fair consideration, while posterity may find something to commend as well as blame, in the conduct, skill, and industry of the present Æsculapians.

Early sanitary ers of Louisiana — important facts.

The labor, if not the result of my researches into the early history of yellow fever in New Orleans, has been great. The few and imperfect notices and documents originally published, and now accessible, devoid as they often are of statistical requirements and numerical appreciation, are of small value—and even these are fast drifting to the shoreless gulf of oblivion. The more distinguished events of Louisiana, military and civil, the more dramatic phases of humanity upon the distant shores of the Mississippi—the exteriorations of society upon which history is ever too prone to dwell exclusively, have found several able historians,* while the medical history, sanitary progress, climatic vicissitudes, topographical changes from forests to plantations—from plantations to cities—from swamps and palmetto lands, to sugar and cotton fields, have received little consideration during a most important period, when the Indian, Caucassian and African races first met and mingled in a boundless wilderness, diversified by vast rivers, plains, prairies, lakes, lagoons, swamps, colossal grasses, reeds and forests. The vital progression, health, longevity, as influenced by modes of life, clothing, lodging, crowding, building, exposure, labor, diet, temperance, cleanliness, bathing, domestic habits, social, educational, mental and physiological characteristics—all these must have produced results for good or evil which might guide the physiologist and the sanitarian in his reason-

Elements of Medical history.

*The late Judge Martin, and Mr. Darby, of Washington, have contributed most ably to the history and geography of Louisiana. Mr. French has done and is doing good service by the publication of old memoirs and documents; all of which, are, however, almost wholly sterile in sanitary, climatic and medical details.

Martin. Darby. French.

Mr. Norman's beautifully illustrated volume, "New Orleans and Environs," contains an interesting historical sketch of Louisiana.

Norman.

Among the historians of Louisiana, the Hon. Charles Gayarré, for the extensiveness of his researches and the opulence of his contributions, stands unrivalled.

Gayarre.

ings. The philosophical sanitarian cannot be too cautious in assigning epidemics and deteriorations of public health, to the sidereal influences—to the vengeance of Heaven, or to importations from the most distant nations. Contagionists have attributed our yellow fever (the paternity of which no nation is willing to own) to Siam, where it was never known, and for no better reason than that the country itself is at the uttermost end of the earth, and was less known than any other part except the very poles! It may be well to inquire, whether the elements of nature or the elements of society have altered during the last sixty years? Whether the stars are less simple, pure, chaste, moral, sober and regular now, than formerly! A change there is.

The reader of these desultory sketches, will, it is hoped, bear in mind that they are not intended to be strictly professional nor wholly devoid of that character. Principles and explanations, which, to a professional reader, may appear as the simplest truisms, may not prove such to all. While the writer claims no exemption from biases, he trusts he that is not a blind partizan, warring against contagion and quarantine, in regard to yellow fever. He has at least examined both sides of these questions, now paramount in the public mind, in their tendencies for good or evil to the present and future well-being of New Orleans, both in its internal and external relations.

TABLEAU

OF

YELLOW FEVER OF 1853,

WITH

THE EPIDEMICS OF NEW ORLEANS,

CHAPTER I.

FIRST APPEARANCE OF YELLOW FEVER IN NEW ORLEANS—ITS PRELUDES AND CO-INCIDENTS.

1796. Preliminary Epidemics. Atlantic cities

The ravages of epidemic yellow fever—in central and insular America, at no great distance—for two centuries, must have produced in New Orleans, from the day the city was founded, a well-grounded apprehension of impending danger. In the Northeastern portion of America yellow fever was older than New Orleans. It had prevailed in Boston in 1691, 1693, 1795; in New York in 1702, 1743, 1748, 1762, 1791, 1793, 1795; in Philadelphia in 1699, 1732, 1741, 1742, 1743, 1744, 1747, 1762, 1793, 1794; in Norfolk, Virginia, in 1747, 1795; in New Haven, Connecticut, in 1743, 1794; in Providence, Rhode Island, in 1794; in Baltimore in 1794, 1795, and in many other Northern towns; in Charleston, ten times anterior to its appearance in New Orleans, during a period of ninety-four years.

Gulf of Mexico. Spain.

Yellow fever had approached the site of New Orleans sixteen years before the city was founded, having appeared at Biloxi, in 1702, ninety miles distant, a French military station and settlement, founded by Iberville, in May, 1699; and, also, at Mobile three years later. The great prevalence of yellow fever, not only in the new but the old world, as in Cadiz in 1705, 1731, 1733, 1734, 1744, 1764, must have caused ceaseless apprehension of danger to the city, had the disease been of an importable character, and the more so as the population consisted of various nations and races engaged in commercial pursuits lying within the yellow fever region—a population ceded from King to King, and sold from Republic to Republic, agreeably to the caprice or cupidity of its masters

1796.

The year 1796, signalized by the irruption of yellow fever in New Orleans, presented the most incontestable facts deserving of the consideration of contagionists. New Orleans from its foundation (it may be repeated) had been closely connected by geographical position, commercial intercouse, languages, and governments, with both insular and continental America, where yellow fever had prevailed for centuries under Spanish, French and English rule, yet always exempt up to this period.

Let us suggest this postulate: suppose that simultaneously with the first invasion of yellow fever, near the mouth of the Mississippi, a handful of immigrants, who had five years previously arrived in the midst of a vast wilderness, fifteen hundred miles distant, in latitude thirty-nine degrees North, nearly ten degrees North of New Orleans, at a time when there were no steamboats, when it required several months to reach the former by the river—I say, suppose that yellow fever should break out at both of these remote centres simultaneously, would it be fair to conclude that personal contagion could, under such conditions, be communicated? Such events did occur. The town of Gallipolis, on the Ohio river, thirty-nine degrees North latitude, settled in 1791 by immigrants from Paris, on an elevated diluvial ormation then in the midst of a vast wilderness just beginning to be settled, was, n 1796, severely visited with yellow fever, attended with black vomit. The late Professor Potter, of the University of Maryland, struck with this remarkable isolated epidemic, (which he used to dilate upon in his lectures) took the necessary steps to investigate it the very next year after its occurrence, when Major Prior of the army an eye-witness, arrived in Baltimore from Gallipolis, and gave the professor a statement in writing, by which it appeared that half of the garrison and many of the French settlers died in ten days from this malady. This strange event in the desert excited great surprise at the time. The army report, by the surgeon-general, (p. 9) "refers to the journal of a voyage down the Ohio, in 1796, by Mr. A. Ellicott. This judicious observer was a witness at Gallipolis to the disease which raged violently, the fatal cases being generally attended with black vomit. 'The fever could not not,' he says, 'have been taken there from the Atlantic States, as my boat was the first that descended the river in the spring. Neither could it have been taken from New Orleans, as there is no communcition up the river at that season of the year.'"

Is Gallipolis

1796.

First epidemic in New Orleans.

Although an epidemic prevailed in New Orleans in 1769, the character of which has not been determined, yet it is highly probable that the disease was not yellow fever. Before a generation had passed away the yellow fever having, without doubt, appeared in 1796 as an epidemic, the numerous writers who asserted that the latter year ushered in this disease for the first time, in the city, could have informed themselves by consulting living witnesses as to the material facts of the case, and would have been contradicted, had they made erroneous statements as to the period of its invasion. A single authority, that of M. Frasans, has been quoted by several authors to show that it appeared first in New Orleans in 1795—among these is M. Moreau de Jonnès, though from his quotation from the former, 1796 will apply as well as 1795, while cotemporaneous writers agree as to the former. Although M. Frasans seems to represent the contagion-party of New Orleans, long since, his statement is important as fixing the date of the irruption of yellow fever in the city. He says (as quoted by Moreau de Jonnès, *Monographie*, 181) that in 1802, the people of New Orleans, among whom yellow fever had appeared six or seven years before, considered it as of foreign origin, that is, it had been imported from the United States. (Frasans. *Vue de la Colonie, &c.*, 86, 91, Paris, 1803.) It does not seem that he was positive whether the first epidemic was in 1795 or 1796.

Frasans.

It may be proper to mention that the late Dr. Drake, states on the authority of Richard Relf, Esq, of New Orleans, one of the oldest and most respectable Anglo-American citizens, "that yellow fever occurred, he himself being a patient, in 1791.

Relf.

(Dis. Valley Miss. 100.) It does not appear from this statement whether the disease was sporadic or epidemic; probably it was the former.

That the health of New Orleans anterior to the appearance of yellow fever was unsurpassed by any city in America, cannot be a matter of doubt, as I have shown in various publications, a summary of which may be seen in the New Orleans Directory for 1852, wherein numerous authorities, official and private, chiefly in the French language, are given, as Charlevoix, DuPratz, LaHarpe, Lozières, Duvallon, Robin and others.

1796.

Health of New Orleans in early times.

Dr. Lind, though at the expense of his theory of marsh-poison, allows "that the inhabitants of New Orleans suffer no inconvenience from their situation in marshes even during the rainy season;" (Hot Climates, 35;) a remark quoted, not to show swamps are good and convenient, but to show that in his day the city was salubrious; he carefully noticed the prevalence of yellow fever in Pensacola and other Southern towns during the last century.

Lind.

The population of the city, by the census of 1785 was 4,980—by that of 1788 it was 5,338—by that of 1803 it had increased to 8,056. It is probable that it did not exceed 6,000 at the time yellow fever appeared in 1796. Of the mortality of that year no record has been seen by the writer. From that period to 1853 it is almost certain that several cases have occurred every year in the city, often only four or five.*

Population.

In the year 1796, yellow fever appeared in Newburyport, Boston, New York, Charleston, Wilmington, West Indies, New Granada.

Co-incident epidemics.

Although it is not intended upon this occasion to give even a sketch of the methods of treating yellow fever, it may be proper to notice the method of cure proposed in New Orleans during the first epidemic, by a Spanish gentleman, Dr. Masdevall, physician to Charles IV, published in this city, in a work dedicated to the Governor, the Baron de Carondelet, in 1796: The doctor's remedy consisted of an antimonial mixture, *in viper-water;* 5 ounces of emetic wine; 1 ounce of cream of tartar; a tea spoon full for a dose. After the fifth day give an electuary of salt of wormwood, tartar emetic and Peruvian bark, in divided doses, the third and last remedy, (*lavement*) called the *blessed laxative*, was composed of antimonial wine, water, honey and oil. He rejected cordials, blisters and blood letting. He considered life as residing in the blood, as declared by Moses (Leviticus xvii, 14) and denounces venesection as dangerous for that reason, as life and health depend upon it. He maintains that his method is a true specific against all the fevers of Spain and America, as he knew from an experience of twenty years. His most Catholic Majesty commanded the Spanish physicians to follow his prescription and to prescribe nothing else. He blamed the physicians of Havana for not having adopted this "blessed" method of treatment. (Med. Rep. vi.)

*During 1796 the basin of canal Carondelet was excavated. The main body of the canal had been finished previously.

Canal Carondelet.

CHAPTER II.

HISTORICAL NOTES OF YELLOW FEVER TO THE CLOSE OF THE EIGHTEENTH AND THE FIRST QUARTER OF THE NINETEENTH CENTURY.

1797. Philadelphia. Providence. New Design.

I have found no satisfactory record of an epidemic in New Orleans in 1797.* In Philadelphia 1000, and in Providence 45 died of yellow fever. In the remote West, "at New Design, fifteen miles from the Mississippi river, and twenty from St. Louis, it carried off more than one-fourth of the inhabitants, although no person during the preceding twelve months had come to this village from any place at which the malady prevailed. As these facts are attested by Dr. Watkins, who had seen the disease in Philadelphia, and as identity of disease supposes an identity of cause, it is shown indisputably that fevers with the pathognomonic features of typhus ictenodes [yellow fever] do occur in positions which forbid the assumption of importation." (Stat. Report, U. S. Army, 9.)

1798. Dr. Brown.

The yellow fever which appeared in Boston, was, according to Dr. Samuel Brown, preluded by and accompanied with the following gloomy appearances of nature, or rather brown studies of the author, who [gravely sets forth that "The common atmosphere, for the most part, was opaque and smoky, as if the earth's surface were undergoing a slow combustion. It seemed a heterogeneous mixture of particles in a state of opposition and propulsion; respiration frequent and unrefreshing. The sun, in mid-day height, appeared as a volume of blood, dark and angry. As it declined to the western horizon, its diameter widened greatly; and at an hour's height, or more, was almost invisible, or shrouded as with sack cloth. These appearances, however, were not constant."

In New York the epidemic raged with extraordinary mortality. It has been estimated that more than one in thirty of the entire population died in a few weeks, mostly in August.

Epidemics in Philadelphia, Wilmington, New London, Chester, Huntington, Petersburg, Alexandria, (Va.) West Indies.

1799. New Orleans.

The prevalence of yellow fever in New Orleans, in 1799, is referred to in the city journals twenty years subsequently.

"Dr. Dow informed me," says Dr. Rush, "in his visit to Philadelphia, in the year 1800, that the natives and old citizens of New Orleans, who retired into the country during the prevalence of yellow fever in that city, the year before, [1799] were often effected by it, while all such persons as did not change their residence escaped it." (Rush, Inq., iv, 126.)

1800.

Charleston, Philadelphia: "In no year since the prevalence of the fever was the desertion so general." (Rush, iv.) Boston, New York, Vera Cruz, West Indies

Cadiz: Persons long resident in the city, as well as West Indians, were remarkably exempt. General alarm in Europe, particularly in Spain, France, and

*M. Victor Debouchel, in his history of Louisiana—1841—says that yellow fever desolated New Orleans in the following years, namely: 1767, 1797, 1802, 1806, 1810, 1814, 1818, 1822, 1824, 1827, 1831, 1835, 1837—an enumeration which does not, in a number of instances, coincide with the most reliable authorities.

the Germanic States. Quarantine rigid against yellow fever generally called the Pestilence, American Plague, &c., &c. Rush's works translated into German.

1800. Charleston.

Charleston: 134 died, including two children born in the city; the residue strangers exclusively. "Some instances have been observed of our youth, who have returned from a more northern latitude, after an absence of three, four and five years, and in one instance of twelve years, without contracting this inhospitable disease. The only exception to this remark are two; the one after an absence of five and the other of nine years." (Dr. Ramsay.) [Probably the two Creole children were born of unacclimated parents.]

Havana. Valiente.

In Havana, 9,977 perished from yellow fever.

The Intendant of Cuba, El Sr. Don Pablo Valiente chartered the Dolphin to take himself, family and suite to Spain, touched at Charleston; and, having anchored in the bay of Cadiz, he went ashore with his party two days after, on the 8th of July. A month later the yellow fever appeared in Cadiz, whereupon Valiente was arrested upon a criminal charge for having imported yellow fever contagion from Havana and Charleston; the former he left in May; the latter he touched at June 2d, and left eight days after. At neither place was there any yellow fever. No yellow fever appeared on board of the Dolphin during the voyage, though three sailors had died. The Intendant, after eleven months' imprisonment, was acquitted at Seville, and was afterwards promoted by the Government, probably as a compensation for, his wrongs

Seville: out of 80,000 souls, 76,000 took the disease.

Cadiz: 14,000 fled, 57,499 remained, 48,500 sickened, 7,387 died.

1800.

Dr. Hosack, and many others, having adopted Dr. Mitchell's theory of septic acid as being the cause, and alkalies as the preventive of yellow fever. Lime water and the like were reckoned to be vastly important in neutralizing the septic acid, which was considered very corrosive, particularly after black vomit appeared. Dr. Cathrall, of Philadelphia, read a paper before the American Philosophical Society, on the analysis of black vomit, asserted that there was an acid in this liquid which is inert to the taste and smell, and harmless when swallowed. Black vomit having been "kept corked in a phial eight or ten days, it assumed an agreeable saccharine odor; kept two years in a state of rest, the flakey particles became perfectly separated—on agitation, it became immediately incorporated, and after remaining six months, showed scarce any disposition to separate. It is, as he says, neither blood nor bile, nor a mixture of the two." (Med. Rep. iv.) The writer has several specimens of black vomit, ten to fifteen years old, which does not appear to change from age.

1801. New Orleans. Carondelet.

In March, 1801, Baron de Carondelet, in an official document, setforth the importance of improving the topography of the city, so as to drain off into Canal Carondelet the stagnant waters which then abounded "near the city," and which he regarded as "the cause of much mortality;" a measure which he says "would put an end to putrid fevers," (Martin ii, 176-7) in which category he, doubtless, included yellow fever, which a few months later occurred as an epidemic.

1801.

Yellow fever in New Orleans: Dr. Snead, of this city, detailed the mode of treatment and criticised it as inefficient. (Med. Rep. ix.)

Yellow fever in Savannah, Norfolk, Norwich, Ct., New Bedford, Mass., in New

York where 140 died in October. In Charleston the celebrated Michaux suffered an attack; one-eighth of the unacclimated perished.

In Philadelphia, sporadic. (Rush, iv.)

In Havana: Mr. Morton, American Consul in that city, estimates the population within and without the walls at 95,000; mortality 2,366.

Dr. Oyarvida, of Havana, published a work in the Spanish language, to show that yellow fever is contagious, asserting its importation from the United States, avowing that when a person is exposed to it and escapes the disease, this escape is owing to the goodness of God.

In Vera Cruz, Jamaica, St. Domingo, Seville, Medina, Sydonia, (Spain) "on a high hill," Leghorn, Italy, 150 died daily.

1802. The government of the United States applied to the Spanish government for permission to establish a Marine Hospital at New Orleans, for American seamen, many having died there in a destitute condition.

1802. Boston—sixty deaths from yellow fever; at Portsmouth, New Hampshire, 10; Wilmington, Delaware, 86; Philadelphia, 250; Baltimore, unknown.

1802. Yellow fever at Charleston—96 deaths; more than half of the attacked recovered; none died under twelve years old; three died after a residence of eighteen months; a few strangers escaped without an attack; not a native white, black or mullato was attacked. (Dr. Ramsay.)

Vera Cruz—428 cases admitted into the Hospital of St. Sabastian—60 died; in the city 1,500 of yellow fever.

The reign of Sepion. Dr. Mitchell, of New York, (born in 1763—died in 1831) learned alike in physic, physics and politics, influential at home and abroad, exercised at the beginning of the present century an influence over the public mind, rivaling that of Dr. Rush. This great New York professor and member of Congress, having discovered the demon of all epidemics, particularly that of yellow fever, called by him SEPTON, that reigned by virtue of the principle of Acidity in the earth, air and water, causing corruption everywhere; whereupon he inaugurated Alcalinity into power with a scrub broom in one hand and a bucket of lime water or soap suds in the other, by which only "Grim Septon" could be conquered.

Alkalies as preventives.

1802. Dr. Mitchell moved in Congress the appointment of a committee with the view of reporting on the purification of ships by alkalies, in order to destroy this pestilential septon. The Secretary of the Navy adopted the theory, or at least the practice, which latter he ordered to be carried into effect. Books, pamphlets and letters soon appeared against Septon, and for Alcalies. The next year an article appeared in the Medical Repository, having the title following: "Dr. Chalmers on the Acidity of the Atmosphere of South Carolina"! The fading of goods, the rusting of metals, and other effects of atmospheric acidity were gravely announced as indubitable proofs of this theory.

Air acid.

The yellow fever prevailed in several cities of the United States; 600 to 700 died out of 1,600 or 1,700 cases in New York, in August and September; 196 in Philadelphia; 200 in Alexandria, Virginia; prevailed at Cattskill, among the hills of New York.

At Malaga, in Spain, 12,000 to 13,000 died; some accounts represent the number as low as 6,884.

Dr. Rush represents this as a year exempt from epidemic yellow fever in the United States, Charleston excepted. It prevailed, however, in New Orleans

In Charleston, 200 to 300 deaths. Prevailed in Winchester, Virginia.

Vera Cruz: total mortality 1,310. Yellow fever prevailed here, also in the West Indies; in Havana 4,766 died.

Leghorn: 6,000 left for Pisa, as did the French army, taking 180 yellow fever cases with them; no propagation of the diseases at Pisa. (Quar. Rep.) 1804.

Malaga, in Spain, out of 110,000 only 7,000 escaped attacks, and 26,000 died of this disease in four weeks. A more reliable statement (that of Arejula, cited Rep. Quar.) gives the population, in July, exclusive of the troops and prisoners, at 36,008; of these 4,548 fled, 18,787 sickened, and 11,486 died.

Carthagena: out of 32,000 there died 14,940.

At Gibraltar, in a few weeks, beginning with August, out of a population of 15,000, no less than 5,733 fell victims, including civilians 4,864, military 869, that is, nearly two out of five. [Subsequent epidemics prevailed on this rock, as in 1813–14, 1828. During the latter year, from August to the 14th of January, 1,677 expired on this rock from yellow fever.]

Many cities of Spain, including Cadiz, were desolated. The population of Spain diminished one million! The official report of deaths from yellow fever amounted to 124,200 for the year.

It is remarkable that during the rigid quarantine which prevailed about the beginning of the present century in Spain, yellow fever raged more, and caused a mortality incomparably greater than was ever known before or since, in so much that various authorities might be produced in which the diminution of population in that kingdom for a single year has been estimated, as above, at one million, owing chiefly to the ravages of this disease. 1804.

As illustrative of the supremacy of the doctrine of contagion, it may be proper to mention that Carlos, King of Spain, by proclamation, conferred on Don Cabanellas and his two children, an annuity of twelve hundred dollars, making Don Cabanellas physician to the royal household, bestowing other privileges on him for having slept one night with his children in the bed whereon yellow fever victims had died in the Lazaretto. A number of galley convicts, in chains, who voluntarily accompanied the Don, for the night, had one year's punishment remitted from their penalties. The party consisted of fifty persons, who suffered no harm. (Med. Rep. x.) Great was the astonishment of His Most Catholic Majesty and his doctors. 1805.

New York: 302 deaths; 116 having been natives. (Med. Rep. x.) 1805.

Providence, R. I.: 30 cases; 10 deaths. (Ib.)

Philadelphia: 300 to 400 deaths. (Rush.)

New Haven, Ct., Gloucester county, N. J., Chester county, Pa, (Ib.)

Havana: H. Hill, American Consul at Havana, reported to the United States Department of State the names of 86 out of 100 American seamen dying in that city, all but one, of yellow fever. (Med. Rep. x.)

St. Anne's Barrack's, Barbadoes: of 278 men recently arrived from England, 70 died in twenty-three days, ending August 20th. (Bancroft.)

Quebec. 1805. Quebec, near the 47th parallel of North latitude, founded (more than a century before New Orleans) upon Silurian rocks, which rise abruptly more than three hundred feet above the tide which washes its base, about 400 miles from sea, was for the first and last time invaded by the yellow fever, in the middle of August; but, September setting in very cold, the disease was not of long duration, though it was nearly as severe as that of the West Indies in malignity, especially among

the troops, a company of 55 belonging to an English regiment perished except six. (Walsh, cited by Moreau de Jonnès.) This epidemic has been described by the usual criteria of yellow fever—haemorrhages, vomitings, yellowness, &c.

1806. No epidemic in the U. S.: In the penitentiary at Richmond, Va., yellow fever occurred. The Board of Health of New York recommended that certain parts of the city which had been much infested with yellow fever, should be reserved for warehouses only, not leased to families.

1807. Charleston: 162 deaths; Philadelphia 3; Savannah; New York 20 cases. (Med. Rep.)

1808. No epidemic except in the town of St. Mary's, in Georgia; half that remained in town died, including Drs. Hitchcock, Turner and Stowell. (Ib.)

Savannah. (Townsend.)

1809. New Orleans: Mr. Gallatin, Secretary of the Treasury, reported the expense at the Charity Hospital for the treatment of sick seamen, at 75 cents per day, each, amounting to $3,542 31. The number of deaths from yellow fever unknown; sporadic cases in Charleston, Philadelphia and Brooklyn.

1810. In Havana, yellow fever destroyed 4,305; Philadelphia 3 deaths; sporadic at Gibraltar; severe at Cadiz and Carthagena.

Population of New Orleans according to U. S. census: city and suburbs 17,242; precincts 7,310 total 24,552.*

1811. New Orleans (Darby, Stat. La. 267.) Philadelphia 5 deaths. Amboy, N. J.

1812. Philadelphia 3 deaths; Cadiz, epidemic

Earthquake in the valley of the Mississippi; disastrous at New Madrid.

1813. Spain; Philadelphia 6 deaths.

1814. Philadelphia 7 deaths; epidemic at Cadiz.

1815. West Indies; Philadelphia, two deaths.

1816. Philadelphia, two deaths.

New Orleans. The city and state authorities, fully alive to the sanitary interest of New Orleans, enacted laws with this purpose, of the most stringent character. Those Sanitary laws of March 6th, 1816, and of March 18th, 1817, for extensive views, special enumerations, and exact descriptions of whatsoever, were then, or have been since regarded as causes of disease, may be equaled but not surpassed by the present generation. One of these acts, covering fourteen pages, having twenty-four sections, exhausts the subject of Hygienic legislation—on paper, at least—and, with 1817. no apparent effect, as to the march of yellow fever. Mr. Darby says at this period "the streets are not yet paved."

Quarantine laws enacted in the winter or before the hot season. (The date not recollected.)

1817. Aug. 10 Dec. 30th. Epidemic in New Orleans: Mortality of white male adults, 760; of white female adults, 63; a ratio of more than twelve times less than the former. Total mortality for five months 1142.

The Physico-Medical Society report the deaths, in August, 304; in September, 178; in October, 91; in November 91; in December, 74. But from these and various other data, I estimate the deaths from yellow fever this year at 800.

1833 Natchez, 33° 31′ N.: In a small population one hundred and thirty-four died of yellow fever.—Dr. Perlee. Dr. Cartwright refers the epidemic to local causes, as filth, a candle factory, offal, &c. (Med. Rec. ix.)

In Charleston, 270; total mortality 1249. (Mills' Statist.)

*Mr. Darby says that "New Orleans and suburbs contained, by the census, 17,242."

CHAPTER III.

QUARANTINE ERA.

Governor J. Villeré, January 6th, 1818, in his annual message, says: "That during the course of the last summer the yellow fever had extended its ravages over the city, chiefly falling on new comers, but many of our citizens were its victims." He thinks that the disease was imported, and regards quarantine laws favorably. 1817. Gov. Villere.

The yellow fever reappeared in New Orleans; mortuary tableau; deaths of white male adults 324; of female adults 81; white children 87; black male adults 219; black female adults 162; black children 277. The mortality augmented in each month until September, in which 165 died. Total whites 392; total blacks 658; grand total 1151.* 1818. Mortality.

An act, approved March 6th, repeals the act establishing a Board of Health in New Orleans, the health officer and all laws for the prevention of the introduction of pestilential, malignant and infectious diseases; directs the sale of the Lazaretto and all its property; investing the Governor with authority to establish quarantine by proclamation at his sole discretion. 1819.

Yellow fever quarantine is founded on some known law of nature, on some ascertained uniform antecedent or leading fact or it is not. That yellow fever is produced by a cause or antecedent as invariable as the rising and setting of the sun is not the less certain, because it is wholly unknown, cannot be doubted. Even the games of chance, so called, happen in strict conformity to a changeless law as much as the winds, waves and eclipses. If importation be the antecedent of yellow fever in New Orleans, let quarantine against it be not only strict, but eternal If the act of the Legislature of Louisiana in the winter of 1817, establishing a code of quarantine laws was wise, the repeal of those laws in 1818 was foolish. But it may be said that these laws had failed to prevent an epidemic during the summer of 1817. True. But why has the same course been pursued since, and why pursue it again as is intended now? The experiment has been often repeated in various countries and with like results—results mischievous, demoralizing, repulsive to humanity, and tending to increase the mortality of yellow fever during an epidemic. If the people of New Orleans could be brought to believe in the contagiousness of this disease, benevolent as they are known to be, the sick would be secluded, intercourse would be so restricted that many would perish from neglect. Of this more hereafter.

The Mayor and Council of New Orleans provide medical attendance, medicines, bread, meat, wine and the like for the sick poor. The preamble to the ordinance says: "Whereas, the disease actually reigning in the city of New Orleans is prim- 1819. Aug. 9th—charity.

In a letter from the late eminent Judge F. X. Martin, the historian, published in the journals of 1815, he gives a flattering account of the city as follows: Professional men make fortunes; land ten miles above and below the city worth from $2000 to $4000 per acre; a single hand for a year $750 to $1000; "if born in the country or seasoned thereto, a slave is worth from $1500 to $2000 in ready money; a genteel house servant $3000." Prosperity of the city. Martin.

*This is supposed to refer to one acre fronting on the river and running back 40 arpents or acres.

cipally among strangers lately arrived in this city, and not yet insured to the climate and that many of them are laboring under pecuniary distress, Resolved," &c.

Mortality. The city proper: had whites 13,604; blacks 13,592. The city and suburbs contained 45,968 souls. Mortality by months, beginning with January: 70—102—97

1819. —78—120—130—130—313—594—313—134—109. Deaths of negroes are distributed almost equally among the months of the year. By this tableau it appears that

Races. six white men died for one white woman. Total mortality 2,190. These figures,

Nuttal. taken from the report of the Medical Society, apply probably only to the city proper. Mr. Nuttal, the naturalist, in his travels, estimates the victims of yellow fever for this year in the city at from five to six thousand, an aggregate greatly exceeding probability.

Army Report. In the official report of the Surgeon General U. S., it is said: "At New Orleans it was estimated that upwards of 3,000 died of yellow fever; and it was not until after the first of December that it was deemed prudent to return either to this city or Natchez. The interior of the country, in the Southern States, seemed to suffer in a corresponding ratio. In the West Indies the fever exhibited perhaps a still greater mortality." (10.)

Medical Society. The grand total mortality, according to the report of the Medical Society (supposed to include only the incorporated limits) is but 1,337; the males being 1,142; females, 195; blacks, male adults, 182; female, 168. Deaths of blacks little, if any, increased during the three epidemic months, while the deaths of white adults increased from 64 in July, to 485 in September.

The first two cases of yellow fever occurred May 7th and 12th; the last death December 9th.

1819. Dr. Dupuy De Chamberry, of New Orleans, in his historical sketch of yellow fever, as it appeared in this city in 1819, says:

"I formerly believed the yellow fever to be contagious, but since I have been in the midst of it, my numerous practical observations have never been able to furnish me with one proof of this much dreaded attribute. Indeed the result has been quite the reverse; and, I am now convinced that the disease is permanently fixed to the spot, and within the limits of the place which has created it. Not one case occurred beyond the limits of the city, during its prevalence in the years 1817 and 1819, that could be traced to any of our innumerable patients, although daily intercourse was kept up with people of the neighboring estates and plantations. A great number of our inhabitants who carried the seeds of the disorder abroad, seeking refuge from the danger at a distance, suffered an attack of the fever and died; but in no instance was it communicated to their friends. *Fifty times* have I had my hands and face besmeared with the putrid blood, black vomit, or fœtid slimy matter of perspiration. Fifty times have I been immersed in the effluvia issuing from a dead or living subject, and never been infected by the disease. From extensive observations, I infer, that the yellow fever of this place is a disease, *sui generis*, the product of local causes, and neither contagious, nor exportable. Flight from the infected spot is the only preservative, and a distance of three miles appears to be quite sufficient to inspire the fullest confidence."

1819. July 4th to Dec. 1st. Yellow fever at Mobile: Five hundred remained in the city, of whom 274 died. At Forts Jackson, St. Stevens, and Claiborne. Prevailed on Tombigbee and Alabama rivers. (Med. Rec. iv, 161; Rep. Committee, Mobile.)

New York, Philadelphia, 13 fatal cases; Baltimore, —; Charleston, 176; total mortality, 1,092. (Mills' Statist.) In Havana, 5,162 victims.

1820. January 5th. Governor Villeré declares "that the scourge of war is preferable to yellow fever;" "that the city had been twice ravaged in three years," [1817 and 1819;] that it is contagious." He urges the Legislature to pass quarantine laws, in which he has the greatest confidense as a preventive.

Governor's message. Governor Villeré, a firm advocate for contagion and quarantine, in his message of November 22d, in relation to the then existing epidemic yellow fever, says:

"All the Medical Faculty appear definitively to have adopted the opinion that the yellow fever, which, during the last year, has plunged us once more into mourning and desolation, is not contagious." But he argues: "During the months of August, September and October, there has been almost constantly in the prison of this distressed city, *a great number of prisoners*, and not a single one among them has been affected with the disorder." "If the yellow fever were natural to our climate, how has it happened that among such a number of persons heaped together in so small a space as the prison of the city, not a single one should have been attacked."* [Dr. Chabert, a physician of New Orleans, opposed the Governor's Dr. Chabert.

*The immunity in the prison will be inquired into in the sequel.

argument as to the prison, and maintained that the Creoles never take the yellow Medical Society. fever, though they do not shut themselves up to avoid it. (Friend of the Laws, Dec. 23d.)]

The Governor reviews the report of the Medical Society for the current year, dissents from its deductions, which he regards as those of all the faculty, and denies what he terms "*the constitutionality of the yellow fever.*"

1820. New Orleans visited by an epidemic: Deaths from yellow fever in the hospital 82. First admission, July 21; the last, December 21.

Opium. Dr. Lambert treated yellow fever with repeated doses of opium and coffee.

Yellow fever prevailed in Middletown, Ct.; in New York; 150 died from August 21 to October 20; (some doctors affirm, others deny that the fever was yellow fever;) prevailed in Savannah.

The yellow fever having prevailed in Philadelphia, in 1820, for the last time for the period of thirty-three years, after a long and mortal cycle of at least twenty epidemics in about one century, it may be proper in this place to give a summary of its recent reappearance in that city, both as it respects its extent and its quarantine import. Whether the recent endemic in that city is to be regarded as the sad souvenir of times long past, or as the precursor of as sad times to come, neither the contagionists nor the anti-contagionists can tell.

1853. Philadelphia. The yellow fever which appeared in Philadelphia in July, 1853, some time after the arrival of the bark Mandarin from Cienfuegos, has been referred to by the citizens of New Orleans as a proof of the beneficial effects of quarantine in arresting the progress of contagion, but without any reason whatever. This matter having been investigated in the College of Physicians by Dr. W. Jewell, the following conclusions were arrived at and published in the Transactions of the College, which are here subjoined, from the Boston Medical Journal of November; (the fourth conclusion being a mere theoretical opinion, is omitted):

"1. That no disease of a malignant type was prevailing in our city previous to the arrival of the Mandarin.

2. That none of the seamen discharged from the Mandarin have sickened.

3. That none of the laborers employed in unloading the Mandarin have taken the disease.

* * * * * * * * *

5. That in no instance can the disease be traced to any individual, except among those who either visited or resided in the immediate vicinity of South and Lombard street wharves.

6. In no case has the disease been communicated to any person visiting, or engaged in attendance upon the sick.

7. Up to this period, not a single instance can be met with, having its origin to the south of where the Mandarin lay last."

* * * * * * * * *

"Dr. T. H. Bache stated that the number of cases of yellow fever admitted into the Pennsylvania Hospital had been *twenty-three*; of these *fourteen* had died, *seven* recovered, and *two* still remain.* These cases were placed in the common wards, without any attempt to separate them from, or prevent intercourse between them and the other patients, but in no instance had the disease been communicated to the latter."

The comments of the learned Dr. Reese* on these events are subjoined in a foot note, from his medical Gazette of December, 1853.

1823. St. Augustine

Dr. Francis, of New York, upon the authority of Judge Andrews, Mr. Delespine and Col. Forbes, says that the yellow fever which devastated St. Augustine, in Florida, chiefly during the month of October, "did not affect a single individual from the West Indies, nor a native of the country,† nor any one who had previously suffered from yellow fever. Forty or fifty deaths occurred among newly arrived immigrants before the alarm became general. Eleven deaths happened in one day. About 200 were exposed to the influence of the disease. Of this aggregate 140 were attacked, of which 132 died, including three blacks. Forty deaths took place in the garrison, in a body of 120 soldiers." (Townsend, 381-2.) The official army report asserts that this epidemic was "entirely confined to strangers, that is, all persons not enured to the atmosphere of the city by nativity, or a residence of a long series of years. Spaniards or natives,‡ resident in the country, who had the temerity to venture into the city during its prevalence, were liable to its attack, though in a milder degree than immigrants." (31.)

Army Reports

Immunity.

Prevailed in Baltimore.

Without anticipating what is to be said about the impending quarantine of New Orleans, it may be remarked that it is hoped the strict rules which were adopted during the era that has just been alluded to will be reënacted, so that if the quarantine doctors should become converted to non-contagion or to contagion, the law will, nevertheless, be enforced with unceasing vigilance and rigidity, because a single exception—the admission of a passenger, bale of goods, or letter,

*October 6th.

†How strange that the antiquated fable of contagion should still haunt the popular creed; and be made the hobby-horse on which as many flippant political doctors ride into places of profit, under that silly relic of barbarism known as the "Quarantine regulations," which are as powerless in keeping out yellow fever from the cities, in which it is generated by local sources of effluvia, as they would be in imposing restrictions against the waves of "old ocean rising in her wrath."

Even in Philadelphia, where a few score of cases have occurred in a district infected by an old and filthy common sewer, we find certain medical savans hunting for its cause in an old ship, guiltless of aught but bilge water, and this, with an obvious source of yellow fever under their noses. When will this ghost of contagion and importation be exorcised? Not while knaves can make money by spirit rapping, which belongs to the same category."

‡The apparent contradiction in these statements grows out of the indiscriminate use of the word country, which in Dr. F.'s account, means a native of the city, not a native of the rural district, as in the latter statement—all of which will be discussed hereafter.

might, by a single spark, ignite the whole magazine of humanity into a fever-explosion. Under former quarantines in this city, the resident physician and the health officer, took the following oath: "That whatever may be his opinion of the origin or infectious nature of the yellow fever, he will be as vigilant in preventing its introduction as if he knew it to be infectious and of foreign origin, and as careful in detecting and removing the causes which are supposed to produce it in this city, as if he believed it might originate here, and that he will well and truly perform the other duties of his office," &c.; under the head of "other duties," a vast many things are included—one of which required the quarantining of all vessels, how healthy so ever they may be, and how healthy so ever may have been the ports whence they sailed, from the 15° S. lat. to 24° N. lat.; a very liberal belt of 39°—covering the West Indies, nearly all of South America, the whole of Central Africa and Southern Asia. On the South side a triangular piece of America, and the Hottentot portion of Africa, and nearly all of New Holland remained free, provided they did not pass through the interdicted regions.

CHAPTER IV.

NEW QUARANTINE EPOCH—ITS EPIDEMICS—ITS RATIOCINATIONS AND INDUCTIONS, LEGAL, MEDICAL AND SOPHISTICAL.

The quarantine laws passed by the Legislature in February, 1821, creating a Board of Health with the most plenary powers, legislative, judicial, executive, pecuniary, and sanitary, modeled after codes the most rigid, and enforced by the heaviest penalties, were carried into effect in March of the same year. The quarantine ground, established at the English Turn, including incidental expenses, cost over twenty-two thousand dollars. The year proved salubrious—a result attributed to the strict quarantine. The Governor, in January, 1822, congratulated the Legislature upon the good fortune of New Orleans as being "*the healthiest city*" *in the Union*. But at the close of August, the yellow fever appeared; it augmented throughout September, but did not reach its culminating point until October, the month of greatest mortality, having amounted to 665, exceeding that of the preceding month by 83. Governor Robertson's next message breathed sorrow and despair. "It is," says he, "an idle waste of time for me to inquire into the causes, origin and nature of this dreadful malady." * * * "The State resorted to quarantine, under the expectation that it would add to the chances of escape from this dreadful visitation. If this hope be fallacious, if no good effect has been produced, if even a procrastination of its appearance has not resulted from the measure, then should it be abandoned, and our commerce relieved from the expense and inconvenience which it occasions."

The course of events since the publication of the New Orleans Directory for 1852, renders it necessary to recapitulate, and fortify with additional proof, the postulates there laid down, as it respects the quarantine of 1821—the alleged importation of yellow fever, and the doctrine of contagion, advocated recently, as being proved by the events of that period.

In 1823, the Committee* of the House of Representatives on quarantine laws

*F. Grima, Esq., was chairman of the committee.

reported that "during the last year. [1822,] notwithstanding the *strictest compliance with those laws*, our expectations were frustrated at the very moment when we thought we could indulge the hope of the most complete success. The season was far advanced, and in the month of September this metropolis enjoyed the most perfect health, when the yellow fever made its appearance."

Observe, that this report was made by a committee altogether in favor of quarantine. They honestly acknowledged its failure, but recommended its continuance in the most rigid form, because it had not been tried sufficiently long, and because other States had similar regulations! The committee avow their belief in the contagious nature of yellow fever, and even adopt the opinion of the Board of Health expressive of its importation from Pensacola, through the Bayou St. John.

The report of the Board of Health to the Legislature, dated January 15th, 1822, brief, dogmatic, and unsatisfactory, holds the following language concerning the epidemic of the preceding year, (1822):

"The researches made by the board at the commencement of the late epidemic, lead them to believe that the yellow fever *was imported* towards the end of August last, by a vessel from Pensacola, arrived at the basin of Canal Carondelet; and attention was first attracted to the disease in a family by the name of Lynch, passengers in said vessel. This family, of which every member but one fell victims to the yellow fever, had removed to Bienville street, when the disease first spread, and from here extended through the city."

"The Board of Health believe it their duty to do away with the impression made by interested persons, to induce a belief in the inutility of the powers which you have so wisely conferred on the board, for the establishment of quarantines, which these persons wish to see destroyed." * * * * *

"This opinion is diametrically opposed to that of the Board of Health, who believe that the yellow fever is contagious, and that the establishment of quarantines is necessary to prevent its introduction." * * * *

"The unacclimated were the sole victims of this scourge."

The Legislature says that the city was perfectly healthy until the month of September—the Board says until the close of August, when the Lynch family having arrived from Pensacola, communicated the disease to the inhabitants of Bienville street, and thence to the whole city.

Truth is one—error legion. This same Board the previous year, in an official manifesto, dated September 4th *, gave a very different account of the origin of this epidemic, charging the disease to the sun, the weather, and fatigue, and never so much as hinting that the poor Lynches had introduced contagion into the city, which latter, saving five yellow fever deaths, "never was more healthy." The Board testifies to the "*strictness of the measures*" (or quarantine then existing) "will check its progress."

This document is a melancholy proof of the inconsistent and contradictory opinions and actions of men unwilling to relinquish power, who resort to the sun, &c., to account for the origin of the fever; then they fly to contagion; now misleading the public by stating that there are but five cases having "the usual symptoms," and then, saying that their "*strict measures will check its progress*,"—thereby jeopardizing the lives of a whole city upon the supposition of the contagiousness of the disease! What can be more criminal in a Board of Health, whether its

* See the sequel.

members believe in the contagious, or local origin of yellow fever, than the suppression of truth, except it be the promulgation of *falsehood*? Seclusion in the one case, if contagion be true, and flight in the other, if the fever be of local origin, might have saved hundreds of lives, if adopted early enough. The late Dr. Townsend, of New York, a consistent contagionist, in a work on yellow fever, published in 1823, avers that facts known in that city "show that the disease actually prevailed in New Orleans *at least a month anterior to this meeting of the Board of Health*." (313.) He says "that from information derived from various sources which may be fully relied on, yellow fever broke out in New Orleans as early as the *beginning or middle of July*."

This document * of the New Orleans Board of Health, subjoined in a foot note, is given *in extenso*, inasmuch as it ignores the Lynch family, and confirms the report in the Legislature as to the *strictness* of quarantine, now doubted after a lapse of thirty years. The exposition of the Board subsequently adopted, asserting the importation of contagion by the Lynches, is virtually contradicted by facts recorded in the Official Report of the Army by the Surgeon General, published in 1840, from reports of Medical officers. Surgeon McMahon, himself a sufferer from yellow fever, at Pensacola, in 1822, says "that on the 7th of August, a young lady, who had recently arrived from New Orleans, died with the black vomit. Her attending physicians, Drs. Elliott and Bronaugh, had no suspicion of the real character of the disease, until this last harbinger of death made its appearance." Five days afterwards two died: "between the 13th and 20th," says the doctor, "upwards of thirty deaths took place. The disease now spread rapidly. Out of a population of 1000 souls, upwards of 200 have already become its victims. On the 26th the troops evacuated the town. Up to this period their health remained unusually good.* * * Among its first victims was Dr. Elliott, an officer whose loss cannot be too much regretted."

Now all the parade of the Lynch family as having been the importers of yellow fever contagion is perfectly futile, as it would be to argue importation by the young lady just from New Orleans; both of these arrivals were coincidences, not causes of the two great epidemics. During 1821, the year before the arrival of the Lynches, seven persons affected with the yellow fever entered the Charity Hospital—and in the two preceding years the number was much greater—for 1820, eighty-two, and for 1819 seventy-one—enough, surely, without the Lynches.

* Board of Health, New Orleans, September 4, 1822.—"At a meeting of the Board on Tuesday, the 3d of Sept., 1822, the following address was adopted and ordered to be published:

"It becomes the duty of the Board of Health to state to the public, that five cases of fever have lately occurred, in which all the symptoms which are usually exhibited in the yellow fever, were observed. It is hoped, from the favorable state of the weather, that the disorder will not spread, as it has not occurred except in persons who had undergone great exposure to fatigue, and had been much exposed to the sun. The precautions taken to render more strict the measures adopted to prevent all communication between this city and the places abroad and in the vicinity where the disease prevails, will check its progress. This hope is more confidently indulged from the circumstances of no new cases having been reported to the Board as having originated within the last two days. With the exceptions noticed above, the city never has been more healthy, and it is believed that the mortality during the last three months has not much exceeded that which took place during the three months preceding them.

"I certify the foregoing to be a true copy from the minutes. H. K. GADON, Secretary."

Although it has been said that no admissions for yellow fever took place in 1821, when quarantine prevailed, yet I have found, as above stated, seven admissions: M. Burns, January 1st, recovered; J. Gildon, January 3d, died 10th; B. Johnson, March 3d, died with black vomit ten hours after; L. Comeline, June 14th, cured; J. Henderson, June 18th, cured; John McCarty, June 20th, cured; J. P. Jacob, August 7th, died next day.

Dr. Davidson, himself a quarantinist, gave the following account of Jacob's case at a meeting of the Board of Health, August 10th, 1821, as reported in "The Friend of the Laws":

"Case of J Jacobs, reported by Dr. Davidson, on the part of the Board: The symptoms and its fatal termination on the fifth day, together with the appearances observed on examination of the body after death, presented strong evidences of its near approach to yellow fever; J. J. was a stranger to the climate, and had been exposed to the heat of a burning sun and hard labor for the last three weeks on a raft of logs on the bank of the river."

The records of the hospital, and these somewhat reluctant admissions of the doctor, are conclusive. Now I could offer a list of yellow fever cases admitted into the Charity Hospital for every year since its records began, taken after a most careful examination of the same, not excepting that most salubrious year 1821, which preceded the advent of the Lynches in New Orleans, in which quarantine was established. As the continuity is unbroken, there is no need in the world that the Lynch family should arrive—others having had the yellow fever sufficiently to furnish contagion enough for the whole city every year. There was no logical connection between the Lynch family and the epidemic of 1822. Had the family never visited the city, all the previous and subsequent analogy goes to show that a number of cases, if not an epidemic, would have occurred just as in other years, and about the same season—just as an epidemic occurred in 1822 at New York, Augustine and many other places in both hemispheres.

This alleged importation, so fruitful a theme with the contagionists of 1822, and of the present period, is completely disproved by Dr. Heustis' work on Epidemic Fevers, published in Cahawba, Alabama, in 1825. At page 421, he says, in his account of the yellow fever at Pensacola, in 1822, before the arrival of the young lady from New Orleans, above mentioned, that "it was pretended by the advocates of imported contagion that the fever was brought in a vessel which arrived from New Orleans about the beginning of August. The captain of this vessel was among the first that sickened and died of the malignant fever; and this after his arrival in Pensacola." Dr. Heustis expressly states that the young lady from New Orleans arrived subsequently to the captain! Dr. Heustis, also, maintains in view of all these events, that it would not be "reasonable for the advocates of quarantine laws to suppose that where those salutary regulations are *so strictly enforced as they are in New Orleans*, that the disease could be imported from that Eden of health. The opinion of one of the most respectable physicians in Pensacola, was, that the disease originated entirely from local causes. Such also was the conviction of the Board of Health."

Although the present investigation is not intended to be history of yellow fever anterior to its invasion of New Orleans, it may not be improper to remark that this disease appeared in Pensacola in 1765, when, and where a British regiment lost

one hundred and twenty men, and eleven out of twelve ladies by yellow fever. (Lind on climates, 119.)

Dr. Townsend of New York, upon the authority of Mr. Barber, of Pensacola and the public journals, states that the population of Pensacola, which was one thousand, was reduced by flight on the breaking out of the epidemic to four hundred, out of which number two hundred and eighty died of the fever. (249–50.)

The logic of 1822, founded on equivocal facts, weak then, still more diluted now by the stream of time for thirty years, can have little potency.

While the facts, arguments and quarantine operations were still fresh, the public felt convinced of the evil of this system of yellow fever prevention, and determined to petition the Legislature to abolish the quarantine laws: accordingly,

On the 23d of January, 1823, a large public meeting took place, in which it was moved and carried, "*that the late epidemic had tested the total inefficiency of the quarantine laws and regulations;* we consider them not only useless, but in the highest degree oppressive and injurious to the commerce of this city; and that application ought to be made to the Legislature for the purpose of having them annulled." A memorial was addressed to the Legislature accordingly for that purpose.

The Legislature, however, took no decisive action upon the matter. The quartine continued in force. The health of the city was good.

Probably in no year since the first irruption of yellow fever in New Orleans were the cases of this disease so few as in 1823, two cases only having been recorded in the books of the Charity Hospital: James Holden, Irishman, admitted September 11th, and died two days after with black vomit; John Hall, aged seventeen, born in Maryland, last from Red River, admitted August 23, concerning whom, I found the following record: "A well-marked case of yellow fever removed to the hospital with black vomit, taken out of a flat boat, laden with hogs, at the mouth of the Red River, by the steamboat Eagle"—recovered.

1822. The yellow fever prevailed in August and September at Port du Passage, "seven leagues east of Bayonne—one of the finest ports in Europe—well fortified by nature and art, and covered towards the land by high mountains and rocks, communicating with the sea by a small passage between two rocks, affording passage but to one vessel"—a locality represented to be unsurpassed for general salubrity. (Dr. Jourdain.)

1823. Natchez, says Dr. Monette, was more severely visited than any other city of its population, 320 having died of yellow fever. Dr. Cartwright,* then of Natchez, now of New Orleans, and Dr., now Professor Merrill, of Memphis Medical College, gave highly interesting histories in the Medical Recorder, and in the Philadelphia Journal of Medical Sciences, concerning a great epidemic at Natchez in 1823, which the latter affirms "took place under circumstances that wholly precluded the possibility of its importation."

"The town of Washington, six miles distant," says Dr. Cartwright, "was crowded with citizens, the sick and the dying who fled to it, yet in no instance did the inhabitants of Washington take the yellow fever unless they had breathed the atmosphere of Natchez. A daily and free intercourse was constantly kept up be-

*Dr. afterward Professor Calhoun, pronounced Dr. Cartwright's paper on this epidemic "the most interesting paper ever submitted to the public." (See Amer. Med. Recordar.)

tween the two places. It was two weeks after the flight of the inhabitants of the city, before Natchez under the hill was attacked."

At Fort Smith, Arkansas, 35° 22′ N. lat., "yellow fever of a high grade prevailed," without a suspicion of exposure to contagion. (Off. Rep. Army U. S.)

New Orleans never had been more healthy for a quarter of a century—a circumstance upon which the Governor congratulated the Legislature in his message of January, 1824, in which he proclaims that New Orleans was free from "*all contagious diseases.*" But January differs from September, as the sequel will show.

In his message, dated Sept. 11th, 1824, Mayor J. Roffignac gives the following exposition of the causes of yellow fever, in which contagion has no place, without alluding to quarantine, which was then in force, and which had afforded no protection; he proceeds to enumerate measures that have at least the advantage of being comprehensible and useful in an economical as well as in a sanitary point of view:

"The opinion of professional men on the primary causes of the insalubrity of New Orleans, tends only to confirm the idea which must occur to the mind of every attentive observer, on looking at the topographical situation of our city, to wit:—that those causes are of two kinds; the one arising within, and the other without the city itself, and that both ought to be counteracted.

"The internal causes are, 1st. The filth daily created in a populous city. 2dly. The low grounds and pools where stagnant water lies, the wooden gutters, constantly wet and fermenting under the rays of a torrid sun. 3dly. The want of privies in most of the populous districts, which renders it necessary to recur to the disgusting and dangerous use of tubs.

"The external causes are, 1st. The marshes lying North and West of the city, uncovered but undrained, and deprived, by the cutting down of trees, of the shelter formerly afforded to them by the shade of a luxuriant vegetation for which the very miasms that now spread death and desolation among us, were a source of life and vigor. 2dly. To the South and East, the Mississippi, which in its periodical retreat, at the hottest season of the year, leaves on its banks a great portion of the filth, which has been thrown into the current, but is brought back by eddies. 3dly. The winds, which at the moment we feel most secure, may, as was the case in 1822, convey to us the deadly effluvia of the dangerous spots which they sweep in their course. Such are, gentlemen, &c."

On the 15th of November, of this same year, the Governor in his message to the Legislature, notwithstanding his exultations at the beginning of the year at the exemption of New Orleans from *contagious diseases*, says, "New Orleans has been again subjected to the dreadful scourge," and suggests the expediency of closing the business season in midsummer, and recommends a general flight to the unacclimated.

The quarantine had been tried for three years, and yet two epidemics had occurred. The contagionists began to waver, and the joint committee of both houses of the Legislature, disagreeing on quarantine, were discharged from the consideration of the same on the last day of November, 1824.

Experience which is ever opposed to false theory convinced the public that quarantine was not only useless but supremely mischeivous in a city so exclusively commercial that a free, untrammeled trade, with freedom of ingress, egress and progress is not simply useful only, but a social necessity, involving the question of subsistence or starvation. Accordingly on the 19th of February, 1825, the Legislature repealed the quarantine laws which it had enacted just four years previously—at the same time the quarantine grounds were directed to be sold. During the eight years that followed, without quarantine, the yellow fever diminished. It never equalled that which took place under the strict quarantine of 1822, when according to some authorities 2,000 * died of that malady, although the records which I have examined, show only 808—a number sufficiently appalling in the comparatively small population then resident in the city, especially during the hot season; the whole reported mortality for the three months ending with October being 1,362. The ratio of mortality in the Charity Hospital was enormous—out of 349 admissions, 239 deaths, and only 98 cures took place. The maximum mortality upon one day rose to 80—of yellow fever to 60.

In New York yellow fever carried off 243, out of 414, the number attacked. 1822. New York.

CHAPTER V.

GEOGRAPHICAL TABLEAU OF YELLOW FEVER IN 1853.

1853. Geological tableau.

The geographical area of yellow fever in 1853, compared with former invasions, was greatly extended, including Florida, Alabama, Louisiana, Mississippi, Arkansas and Texas—six States of the Union of vast territorial expansion, consisting of alluvial, diluvial, and tertiary formations, valleys, dry prairies, elevated plateaux, irregular terraces, low undulating hills and bluffs, and pine woods, interspersed with bayous, lakes, shallow basins, shaking prairies, large bays, dense cypress swamps, cane brakes, colossal grasses, inundated plains—a region undisturbed by volcanic action, where the geological or telluric causes of disease, if such be really regarded as causes, must be nearly uniform. Of these States five are washed by the almost tideless Gulf of Mexico, presenting a vast depressed, marshy, sandy, shelly, rockless littoral, which curves from the Rio del Norte to the peninsula of Florida, deeply indenting the temperate, yet approaching the torrid zone, having low outlying islands in front, and numerous great rivers flowing through the back ground, bringing detrital matter from the high lands and primitive formations of several mountain chains, with tertiary limestones and coral reefs trending along its eastern portion upon the Floridian peninsula.

As immense importance has always been attached to the topography of yellow fever, which has been generally attributed to swamp-exhalation, it will be necessary to take a closer view.

The elevated zone called the bluffs, a broken diluvial plateau, touching the Lakes Pontchartrain and Maurepas on the South, where it is most depressed, running north between the Pearl and Mississippi rivers; the eastern shore of the latter for hundreds of miles, with some interruptions, is overlooked by these impending terraces, which sustain forests of colossal magnolias, pines, oaks, liquidambers, &c.—a platform which sundry learned medical writers have indicated as a secure retreat from yellow fever, although neither the past nor the present justify this theoretical view. The epidemic of 1853 raged fully as much in this region as in the most depressed plains among the vast cypress swamps and salt water marshes of littoral Louisiana.

* The Rev. Timothy Flint who was in New Orleans in 1823, estimates the mortality at 2,000.

The epidemic was most fatal in this region, from its Southern border upon the Northern shore of Lake Pontchartrain at Madisonville, Mandeville, Louisburg and Covington, to the higher lands of Baton Rouge, Clinton, Port Hudson, Jackson, Bayou Sara, St. Francisville, Fort Adams, Natchez, Grand Gulf, Yazoo and Vicksburg, not sparing the little villages of the piny forests.

Topography. Thus the towns of Louisiana, Alabama, and Mississippi States, elevated from 20 to 400 feet, and more, situated on the tertiary formation, often in the pine lands, remote from swamps, being high, dry, and clean, suffered more, in many instances, than New Orleans situated, as it is, upon the recent alluvium or newer Pliocene, touching the river in front and dipping into the stagnant swamps of the cypress basin in the rear, and intersected everywhere with filthy gutters, sewers, ditches or canals. The elevated zone of pine woods in Northern Louisiana, and elsewhere in the adjoining States, forms a striking contrast to the depressed plains, cypress basins, and marshes of the Southern delta. The epidemic of 1853, like previous ones, goes to prove that marsh-miasma is not the specific cause of yellow fever, as is generally supposed. The very towns which the lamented Drake recently designated, on theoretical grounds, as safe retreats from yellow fever, have suffered most from it.

This topographical sketch of yellow fever will be concluded by a slight outline of a few towns where it appeared in 1853—some in elevated, some in depressed situations, taken almost at random from a multitude, omitting those of Texas altogether.

1853. Pensacola. The epidemic appeared at Pensacola: 30° 29′ N. lat.; elevated 40 to 50 feet—with rising grounds in the rear, the sea before, and dry white sands beneath it, (founded in 1699). This town has for nearly a century been occasionally visited by yellow fever and sometimes nearly depopulated.

Yazoo. The city of Yazoo, "dry, elevated and beautiful," 32° 40′, N. lat., "was shrouded in gloom, sorrow and mourning by this never to be forgotten pestilence." By the first week in October, 150 had died of the disease, which was still progressing—a large mortality for its reduced population.

Baton Rouge. Baton Rouge, 30° 30′, N. lat., 135 miles above New Orleans, on high ground, with a population of 2000 greatly reduced by flight, was, early in November, reported officially to have lost 202 by the epidemic.

Shreveport. In Shreveport, 600 or 700 miles from New Orleans, on the Red River, the epidemic beginning in September, declined early in November, but reappeared towards the end of that month, and still continuing to December, having destroyed about one-fourth of the population, judging from the newspapers and from the verbal statements of visitors from that town. Up to the 2d of September, 165 fatal cases had been reported.

Thibedeaux. In Thibodaux in about one month ending on the 24th of September, the deaths from yellow fever amounted to 147, or 15 per cent. of the resident population, as reported in the papers; but Dr. McKinley, a practitioner in the town, informed me that not more than 500 persons remained during the epidemic, and of these 160 died—more than one-third.

Fort Adams. About two hundred miles above New Orleans, upon the steep declivities of the hills which border the eastern bank of the Mississippi river, stands the town of Fort Adams, which in former years, as well as in 1853, was visited with yellow fever, as were the neighboring plantations upon the hills.

Natchitoches. Natchitoches, 31° 46′ N. lat., one of the oldest towns of Louisiana, more than four hundred miles from New Orleans, on Red River, "beautifully situated on a well developed river bank, extending back to a pine bluff, with fine scenery around it," suffered and still suffers severely, at the latest dates.

Providence. The village of Lake Providence, in a population of two hundred, is reported to have lost one hundred and twenty from yellow fever.

Alexandria. At Alexandria, the disease became epidemic about the middle of September. It declined towards the end of November, but was not wholly extinguished at the latest dates. The disease is reported to have carred off from one-fifth to one-sixth of the population. The heavy frosts at the close of October and beginning of November, did not appear to have had any marked influence upon the epidemic.

From this imperfect geographical enumeration, it is evident that *altitude* did not modify the epidemic of 1853. The general opinion that yellow fever appears only in depressed places, or marshy plains, is contradicted by innumerable facts in America and in Europe.

The report on quarantine and yellow fever by the British Government for 1852, enumerates ninety-six cities, towns and villages in Spain, wherein yellow fever has prevailed in this century. Many of these places are far inland, high, dry, rocky and hilly, and among mountains. Ximena on a hill; Chipiona on a rock; Medina Sidonia on a high hill; Los Barrios in the mountains; Xerez on a hill; Arcos de la Frontera on a very high rock; Utrera, between two hills; Carmana on a hill; Moron at the foot of a chain of mountains; Granada 927 feet high, near the Sierra Nevada mountains, thirty-one miles from the sea; Ronda in the midst of a range of mountains at a very great elevation.

Canal Carondelet. 1796. 1853. The medical geography and yellow fever mortality of Gibraltar, as forming the strongest contrast to New Orleans, and as contradicting the marsh-theory of yellow fever, deserve a slight notice: Gibraltar, a compact, gray, marble promontory, three miles long, seven in circumference, an area of 400 acres, covered in few places with earth, rising 1,500 feet above the sea which washes its almost inaccessible walls, having had a population of 15,000 in 1804, lost out of this number in a few weeks 5,733 souls from yellow fever, or nearly two in five.

Without the remotest wish to add another to the many futile expositions of the specific cause of yellow fever, I may be allowed to refer to two coincidents which attended the first and last epidemic irruptions of this disease in New Orleans. The original Basin of Canal Carondelet was excavated in 1796; the capacious Basin now being excavated for the same Canal, about a mile from the city and from the former, was to a great extent dug out just before the epidemic. Frequent visits to this spot with the view to its geological character, gave me opportunities of noticing whatsoever transpired in that district in the spring, before the epidemic appeared. The laborers, nearly all Irish, enjoyed very good health, although the emanations from the Bayou, where the scene of labor lay below the terminus of the old Canal, were most offensive. The water was so impure that many of the fish were killed, adding to the offensive effluvia. This, however, was attributed, not so much to the filth from the streets, as to the deleterious refuse matters from the gas-works of the city.

Jail. In the New Orleans Directory for 1852, I gave a summary of my researches, based on documentary, traditional and living testimony, showing that in both the

old calaboose and new prison, yellow fever had never prevailed, even during epidemics, although no means had been adopted, as quarantine, fumigations and seclusions, to prevent the introduction of the supposed contagion of yellow fever. The conclusion drawn upon that occasion is thus cautiously expressed: "There is, if we may reason from what is known, but one certain method of escaping yellow fever in New Orleans—*incarceration!* That may always fail hereafter, but so far it has not." Failed it has, during the far-reaching epidemic of 1853, but the failure has been so limited that the general rule is not yet invalidated. Through the politeness of my friend, Dr. Cartwright, I have received a document copied from the jail record, from which it appears that the average number of prisoners in the Parish Prison from June 2d to October 4th was 170, a large portion unacclimated; among whom twenty-two were attacked with yellow fever and six died, two having had black vomit. Twelve of these prisoners were admitted after the 11th of May, anterior to which several cases of yellow fever had appeared in the city, as it has been asserted. Four of these prisoners were admitted in May, one in June, three in July, two in August, two in September, and all but three had been admitted during the year 1853. Now if twenty be deducted for the acclimated, the residue 150, if they had been at large, exposed to the sun, dissipation, &c., would probably have lost fifty of their number, belonging, as they did, to the reckless class.

1852-3. From the same gentleman I learn that the records of the jail show there was but one death more in 1853 in the jail than in 1852, among the same average number of prisoners, and for the corresponding period of the year.

Frost. About the 25th of October a white frost appeared, for a few nights, at many of the interior towns of Louisiana, which was received as the harbinger of returning health, but which did not, in a marked degree, arrest the march of the epidemic. Warm weather, however, soon returned, and has continued to the present (the third week of December;) but this did not revive the epidemic in places where it had declined, as in New Orleans and many other places.

Clinton. In the town of Clinton, in the parish of West Feliciana, lying between the Mississippi and Pearl rivers, 100 miles Northwest from New Orleans, the epidemic began about one month before this frost, but at the latest dates (December 10th) it had not yet disappeared—75 having died out of 350 or 400 who did not fly from the town as did about 1,000 persons. Several blacks died.

In places where the epidemic had steadily and greatly declined, the return of absentees, and the influx of strangers did not reproduce the epidemic, as was generally expected. The arrival of absentees, mariners, steamboatmen and immigrants, amounting to about 50,000 in New Orleans, did not, in any appreciable degree, affect the ratio of declination. The mortality, from yellow fever, officially announced for the week ending December 18th, 1853, being three, discloses a fact of supreme significance against the contagiousness of this disease, inasmuch as the city is, if any city can be, reeking with contagion.

CHAPTER VI.

MORTUARY TABLEAU OF NEW ORLEANS AND MOBILE DURING THE EPIDEMIC OF 1853.

1853. Population. The population of New Orleans when the epidemic broke out is estimated at 150,000, or 4,541 more than that of the city census of the year 1852, which gave a total of 145,483. It is probable that 30,000 left the city, whereby the population remaining was reduced to 120,000, including 30,000 or 40,000 that had never passed through an epidemic, and perhaps many thousands that had passed untouched through the epidemic of 1847—some of whom suffered in 1853—an enumeration, however, merely proximate, without any certain basis.

Numerical analysis. As the epidemic increased the mortality from other causes was little affected, particularly after the month of July. For five months ending June 25th, the average weekly mortality from yellow fever was about 4—from other diseases 116; the next five weeks gave an average for yellow fever of 280 per week—other diseases 109. The next four weeks ending August 28th, gave a yellow fever average mortality of 1,211 per week, or by weeks respectively, 967, 1,288, 1,346, 1,243—aggregate 4,844; while for the same time the average from other causes was 157—not including an average weekly mortality of 71, reported as deaths from causes unknown, and generallly supposed to have been deaths from yellow fever. During the next four weeks ending September 28th, the average weekly deaths from yellow fever was nearly 200, while for the same time the average from other known causes gave 65, and deaths from unknown causes a fraction over 16.

From the last week in May, when the weekly mortality was for yellow fever 1, and for all other diseases 139, the non-yellow fever mortality vibrated but little until after the week ending the 23d of July, having ranged from its maximum 158, for the week ending 25th of July to its minimum 129, for the week ending July 9th, but for the week ending July 23d it rose to 188; the next week it reached its maximum for the season, that is 192, at which time the epidemic was rapidly increasing, 1,499 having died, 1,121 in the two preceding, and 1,325 in the three preceding weeks. About the 9th of July the epidemic, as such, began, so that the ratio of mortality from causes other than yellow fever, was not much disturbed during the greatest fury of the epidemic until the week ending September 4th, after which it declined to 162; but for the next week it rose to 120, nine less than on the 9th of July, when the epidemic appeared, and 19 less than the week ending May 28th, when the first death from yellow fever was reported. In the week ending the 21st of August, which gave the maximum of deaths from yellow fever, that is 1,346, the mortality from other diseases was but 152, exactly the same as for the week ending July 2d, when, as yet, all the deaths from yellow fever beginning with May, were but 47. So that the ratio of mortality, independent of the yellow fever element during its unparalleled prolongation, was but little changed except during the last two precursory weeks of July. Thus the epidemic caused but a slight oscillation among other maladies, while its pestilential waves rolled over the devoted city.

If the epidemic be considered in a larger view, by months, it will be seen that its collateral influence is scarcely manifest. Mortality from causes other than yellow fever, for June, 581; for July, 559; from yellow fever for June, 40; for July, 1,406, not including 45 for the former month, and 112 for the latter, under the head of unknown or "not stated."

In August, the two days in which the mortality from the epidemic was least, were the 1st, 106, and the 31st, 95; the decline in September was gradual from the 2d, 102, to the 30th, 9. The progressive decline, with some fluctuations, continued throughout October and November.

The greatest number of deaths in any one day, 283, of which 239 were from yellow fever, occurred on the 22d of August. The greatest number of deaths from the fever, in any one month, was in August, amounting to 5,189, or by adding the unknown, 5,242; by adding all the deaths 6,235; an average exceeding 201 per day; about 9 every hour; one every six or seven minutes! for a whole month!!

1853. Mobile. Mobile: Here the epidemic began early in August, reached its maximum mortality in the first week or ten days of September; the deaths from yellow fever for the week ending the 9th day of that month reached 194, which, added to the mortality from other causes gave 241, a fraction less than 35, as the average of each day; during the second week the mortality began to decline slightly; in the last week the deaths from yellow fever had decreased more than two-thirds; the decline was progressive throughout October; on the 26th of this month the aggregate mortality from the first of August amounted to 1,256, of which 889 were from yellow fever according to the official report; but of the number reported as dying from causes unknown, it was supposed that half were from yellow fever, which will give a yellow fever aggregate of 1,072 without counting sporadic fatal cases which occurred subsequently. The average number of deaths for each of these eighty-seven epidemic days, is a fraction less than 15. With the increase of mortality from yellow fever, an increase of mortality from other causes took place also during this period.

The epidemic in Mobile beginning more than a month later than in New Orleans, reached its culminating point three weeks later. Of shorter duration in Mobile, its proportional mortality was larger than in New Orleans, a fact that applies to nearly all the towns which the epidemic visited.

ARITHMETICAL TABLEAU OF THE MORTALITY IN NEW ORLEANS, IN 1853, FROM MAY 26th TO OCTOBER 22d.

Period 1—May 26–31—Sporadic.

Total	110
Yellow Fever	3
Other Diseases	97
Diseases not stated	10

Period 2—June—Epidemic Inception.

Total	666
Yellow Fever	40
Other Diseases	581
Not stated	45

Period 3—July—Epidemic Increment.

Total	2,077
Yellow Fever	1,406
Other Diseases	559
Not stated	112

Period 4—Aug.—Epidemic Culmination.

Total	6,235
Yellow Fever	5,189
Other Diseases	689
Not stated	357
Discrepancy	29

Period 5—September—Epidemic Decrement.

Total	1,565
Yellow Fever	485
Other Diseases	961
Not stated	88

October 22.

Total	563
Yellow Fever	139
Other Diseases	310
Not stated	57
Discrepancy	3

RESULT.

Total Mortality for 149 days*	11,156
Total Miscellaneous Diseases	2,697
Total Yellow Fever	7,782
Total unnamed (mostly yellow fever)	669

*Mr. Maginnis' "List of Interments" begins twenty-six days earlier, and ends eight days later than the enumerations which I have compiled. His aggregate exceeds the above by nearly one

By adding the two latter, the aggregate mortality from yellow fever will be 8,451, or by deducting one-third from the unnamed diseases, 8,228, without enumerating deaths from yellow fever, from October 22d to December 22d.

In these totals the discrepancies are reduced from 32 to 8. The whole mortality from yellow fever is estimated, in round numbers, at 8,400 for the year 1853.

The maximum mortality of the yellow fever of 1853 arrived sooner in the season than usual, and is more truely represented by that of the plague in London, in 1665; namely—June 590 deaths, July 4,129, August 20,046, September 26,230, October 14,373, November 3,449; total 68,817.

Howard Association. According to the report of the Howard Association, published late in December, the society had under its care during the epidemic of 1853, no less than 11,088 yellow fever patients—5,203 males, 5,885 females—of whom 2,942 died, and 8,146 were cured. Expenditure, $159,190 32. Average for each patient about fourteen and a third dollars. Of this number (5,845) much more than half were Irish; German, (2,890) nearly a quarter; French, 436; United States (716) less than one in sixteen of the whole. Hence, it appears that Ireland and Germany give 8,735; other countries 2,353.

The Association, during the epidemic, received from all parts of the Republic the sum of $228,927 46; more, indeed, than they had need of, leaving a large surplus to be put out at interest for this charity.

Omitting Spain and the United States, the yellow fever zone contributed but nineteen; the plague zone of the east, as Palestine, and Greece, but seven to this formidable aggregate of 11,088.

Mortality of the sexes. The predominance of female patients in the above enumeration is remarkable, in as much as that sex is the least susceptible to the yellow fever, and contribute to the mortality from this disease in a ratio greatly inferior to males. The most probable explanation is this—females preferred the Howard hospitals to the Charity hospital and the city hospitals, established by the Board of Health.

In order to ascertain approximatively the proportion between the mortality of the sexes, I selected the first day of August, counting all the interments from fever, as distributed among the letters of the alphabet, and among the following cemeteries, in Mr. Maginnis' "List of Interments:"

	Males.	*Females*		*Males.*	*Females.*
Cypress Grove No 2	8	2	Lafayette	24	11
Protestant	1	1	St. Patrick's	15	19
Charity Hospital	21	9	St. Vincent	13	8
Total				82	41

Hence, it appears that the mortality of females, is, for 1853, exactly half as great as that of males. This high ratio of female mortality is, however, one of the most extraordinary features of the late epidemic. Of 1,450, who died of yellow fever in August, September and October, 1841, but 220 were females, or nearly one in seven. The ratio of mortality among children will probably be found enormously high from fever in 1853, compared with preceding years. This will appear obvious by Mr. Maginnis' list, compared with the following extensive analysis of the epidemic of 1841; thus—I made thirty-three series, each consisting of thirty persons; I then took the youngest one in each series, (among these 990 dead) which

Children of 1853–1841.

thousand, (995) and is doubtlessly as accurate as the crude materials would allow. This document will afford grave-yard arithmeticians facts worthy of extended analysis as to ages, sexes, and so forth.

gave these ages: 15—17—17—2—5—20—19—16—20—17—15—17—18—19—8—2—7—18—18—19—8—6—8—2—15—3—18—14—2—18—3—5—19. Scarcely an infant in the whole series!

1853. In order to test, approximatively, the ratio of infantile deaths from fever, I counted the ages of all fever victims who were interred in the following cemeteries, on the 10th of August, namely, Cypress Grove, No. 1 and No. 2, and St. Patrick's, amounting to eighty-nine known ages, and two called "infants," (say ninety-one) among which were two aged 2; one aged 3; one 4; which with the two infants, make six out of ninety one—a result which could not have been anticipated from the history of anterior epidemics, as the very young and very old, as well as women and negroes, had always suffered less than other classes.

CHAPTER VII.

NUMERICAL TABLEAU OF CREOLE MORTALITY IN 1853 FROM YELLOW FEVER.

Mr. Maginnis on Interments in N. Orleans. The proprietor of the True Delta, Mr. Maginnis, having made the necessary arrangements early in the epidemic of 1853, to obtain a correct list of all the deaths (12,151) in New Orleans for the six months ending with the first of November, and having published the same early in December, showing the name, age, place of nativity, disease and date of interment, compiled at length from the physicians' certificates and other documents, I beg leave to offer some numerical reasonings based on this valuable document, particularly with the view of illustrating the disputed question, whether Creoles enjoy immunity from yellow fever or not? Whether Creoles die, and in what proportion? The method I have adopted is as fair as can be conceived, and will give in this particular, a compendious view. I selected the two first weeks of August, the middle of the epidemic, as distributed among the twelve cemeteries, and among the different letters of the alphabet, counting all the persons who died of fevers of what kind soever, as the list does not distinguish the fevers—the one from the other—an arrangement the most unfortunate for the merits of the Creole question, because, in any case it will be admitted that Creoles would be more likely to die of any other fever than yellow fever. It will be provisionally assumed that all the deaths marked "*fever*" were from yellow fever alone. The whole number of deaths during this period among natives of New Orleans is but 21—of this number one was aged five, and two aged seven days; two aged two; one aged three, and one ten months; one aged one—three aged two—one aged four—one aged five, and two aged nine years. In one case the word "child," in another "infant," are used to denote the age, and in two cases the ages are omitted. Omitting the two infants and the two of unknown ages, probably infants, the average of the remaining seventeen is twenty-six months, or by omitting the two highest ages, the average age will be less than fifteen months. The list here referred to shows that these twenty-one children are all that died of fevers of every kind among all classes of population during fourteen days in the midst of the epidemic. For the sake of illustrating the argument, the least favorable to creolization, as before intimated, let it be assumed that these all died of yellow fever, though there can be little doubt that the whole number were born of unacclimated parents—parents who had not been creolized either by birth or long continuous residence in the city. Even the two aged nine years each, probably were born of

Immunity.

Method.

Numerical analysis.

parents only temporarily resident in the city during the cooler season of the year, whose summer residences are in the country; or they may have been born of parents who lived here nine years ago, as immigrants or visitors, and who had returned to New Orleans. But let it be admitted that both of these children were born of parents constantly resident for ten years—admit that these children had not been sent from the city to the country, or to the North among relatives, to school, &c.; then let these two Creoles be compared with the whole number of deaths from yellow fever, and what will be the result, supposing that they did die of yellow fever and not from some other fever? The whole number of deaths during these two weeks, as taken from the official daily report of the Board of Health, is 2,762—from yellow fever alone 2,252—from diseases not named, supposed to be yellow fever 114; total deaths from yellow fever 2,369. According to this supposition waiving, for the present, the consideration of former epidemics, one Creole in 1,184 died of yellow fever out of a creolized city population four or five times greater than the non-creolized or strangers.

In order to vary the numerical consideration, I selected the first week in September, counting the number of deaths among persons who had been born in New Orleans. This was the more desirable, because at that advanced stage of the epidemic it might be supposed that it would reach persons long resident to a greater extent than in its inception, and, such indeed was the general opinion. Upon examining the alphabetical list as distributed among the twelve cemeteries, nine individuals proved to have been born in New Orleans: one aged two, one three, one seven, one eight years, two aged six months, one eight months, one eighteen months, and one mentioned as an "infant," giving an average age of thirty-four and a half months. This whole number is subject to all the contingencies in the first series; during these seven days the total mortality was 741, of which 560 were from yellow fever, and 33 under the head of "unknown," making 593, leaving 148 for all other diseases. This enumeration does not conflict with, but gives validity to, the explanation given of the former series.

St. Louis cemetery No. 1. The St. Louis cemetery No. 1, which represents the wealthy French of the city and of the country, and those immigrating from France, presents the following mortuary tableau for six months, ending with November the first: deaths from all fevers among individuals born in New Orleans, 6; one aged twenty months, four, respectively, 3, 10, 20, and 22 years, and one styled "infant." The explanations previously given apply here. That only six should die of fever in six months, had no epidemic prevailed, is remarkable, in a mortality of two hundred and six. Had all these six deaths been from yellow fever, it does not, in all probability, invalidate creolism in the least. Three were infants, one a girl, and two still young, who probably had not remained continuously in the city, and who, on returning, contracted the disease.

Protestant cemetery. The Protestant Cemetery is the best mortuary representative of the wealthy creolized Americans, many of whom, however, could scarcely expect immunity during an epidemic, inasmuch as they leave the city annually, in the summer, and educate their children in the North. The interments from all fevers among the natives of New Orleans in this cemetery, for six months ending November first amount to eight, in a total mortality of four hundred and thirty; one of these is "a child," one aged twenty-two months, three aged two years, one five, one fourteen, and one eighteen years. The latter two were, probably, as usual, truants, who did not stay at home much, if at all, but, having returned, died. Previous ex-

plantations will apply here; but, if they be rejected, the mortality from all fevers is, for this cemetery, a little over one for each month.

Cypress Grove No. 1.

The Cypress Grove Cemetery No. 1, is assimilated in character to that of the Protestant. In the former, the total number of deaths among natives of New Orleans from all fevers in six months, ending November the first, is four—one called "a child," one aged two, and one seven months, and one three years, out of 300 deaths; that is, one native infant died in every two months upon an average.

The worst view of these facts does not overthrow the doctrine of immunity nor afford much aid to terrorists. The creolized dig but few graves in the swamp cemeteries, and if they were much damaged by yellow fever, these cemeteries would tell it.

Unknown names and nativities.

The records of the cemeteries of 1853 disclose the most astonishing facts, significant at once of the rapid and deadly march of the epidemic and of the enormous recent increment of the uncreolized element of the population. How many nameless dead bodies! The place of nativity in nearly two-sevenths of the dead is not known, according to Mr. Maginnis' list! amounting to 3,232! In 1843, among 692 deaths from yellow fever, the nativity of only 132 was unknown, a little over one in six. In 1847, among 2,660 deaths from yellow fever, the place of nativity was unknown in only 238 instances, or one in eleven nearly. Further illustrations are unnecessary. It is evident that both the Creoles and the creolized do not come within the category, "unknown." The name and the place of nativity among these classes could scarcely have been unknown, had yellow fever been fatal among them in 1853, a fact so extraordinary would have excited not only attention, but consternation

CHAPTER VIII.

CREOLISM, URBAN, RURAL AND ACQUIRED.

Creole.

The word *Creole* in Northern latitudes is often misapprehended, so as to imply more or less of negro blood.

In Spanish America, Criollos or Creoles, were, in the early days of the colonial governments, the native whites of European extraction; neither the native, Indians, nor native negroes, nor mixtures of the races, were so denominated.

Towards the close of the last, and beginning of the present centuries, Drs. Mosely and Williamson and many others, used the word Creole, as applicable, not only to the whites born in the colonies, but to negro natives also.

"CREOLE—A native of Spanish America or of the West Indies, descended from European ancestors." (Webster's Dict.)

"CREOLE—A name given to the descendants of whites born in Mexico, South America and the West Indies; in whom the European blood has been unmixed with that of other races." (Brande's Encyl.)

"CREOLE.—Nom qu'on donne à un Européen d'origine qui est né dans les colonies." (Dict. L'Acad.)

In Louisiana, every native, be his parentage what it may, is a Creole. They are convertible terms.

Although the word *Creole* in its usual acceptation means a white person, it applies to all races, as Creole negroes; it even applies to the inferior animals, and things; a Creole chicken, egg or cow, is worth nearly twice as much as one from a

distant State; while a creolized horse, after considerable risk, becomes better, being larger than a Creole horse. City creolization, whether native or acquired is, a practical distinction in the business of New Orleans.

Creoles of the city and the country.

The word Creole is generally used in a sense too latitudinarian for precise statistical investigation. It is the resident city Creole, not the country creole—not the Creole who migrates every summer to New York, London, or Paris, that may hope for as good health as is possible to humanity, while two or three hundred others daily fall victims around him—a definition which excludes a great many called Creoles, and one often forgotten, in writing on the subject of yellow fever. Hence arises many apparent contradictions among authors who use the word in different senses.

In former, still more than in recent times, has this fundamental distinction been overlooked. In a great majority of the works on yellow fever in the West Indies, and even in Louisiana, where Creoles are said to suffer from this disease, the true explanation is, that these persons are *Creoles of the country, not of the city:* or at most, they reside in the latter occasionally, chiefly in the winter, and are, therefore, liable to the disease, though they usually have it in a milder form than strangers, and very rarely die.

Dr. Dufour, MS.

In an interesting manuscript on yellow fever by the late Dr. Dufour,* of New Orleans, left in the possession of Mr. Joseph Le Carpentier, copied from the original, in the doctor's hand writing, by Mr. Y. Noel, and kindly presented to me by my friend Mr. Barbot, apothecary, it is asserted by the doctor, that in the epidemic of 1820, as well as in years preceding, many persons, natives of the place, had fallen victims to this malady. He says vaguely enough, "beaucoup de personnes des pays en furent atteintes, particulièrement les jeunes gens:" he treated several of these, two of whom died the same day in the same house, a brother and sister.

A few physicians and others, mostly advocates for the contagiousness of yellow fever, maintain that all the Creoles of New Orleans, not less than strangers, have this disease once during life, for the most part during childhood, and that it proves fatal to many of them. It must be confessed that as yellow fever, with rare exceptions, attacks an individual but once, it approximates in this particular, the law of contagions proper. This sweeping statement, however, is, with few exceptions, erroneous, as may be proved by authentic documents concerning all of the epidemics witnessed by the writer for seventeen years, not excepting the extraordinary one of 1853 itself.

Many escape yellow fever.

It will have been remarked by careful observers that many families have been settled in New Orleans for half a life-time without ever having had yellow fever. Indeed, it has been thought by many physicians previous to 1853, that at least one-third of all strangers settling permanently in New Orleans, escaped yellow fever altogether—a ratio, however, which is too high for the year 1853, (it may be safely affirmed) although many strangers, including entire families, escaped the extraordinary epidemic of 1853.

Nativity.

The simple fact of being born in New Orleans is not, in itself, protective Thousands are thus born of uncreolized parents, who pass through the city, as immigrants, or who reside in the city in the winter only. Their return to the city might, in this way, swell the number of the so called Creoles to hundreds every epidemic.

*Traitement de la Fièvre Jaune. Méthode du Docteur Dufour. MS.

If it be conceded that no creolized person of New Orleans ever dies of yellow fever, it will still be difficult to account for the extreme rarity of deaths from yellow fever among individuals who ought to have been born in the city, upon the doctrine of chance or probability. Hence a greater number of victims, among natives, might be anticipated for these reasons.

Immunity.

The exemption of the creolized of the city is a fact which every epidemic has confirmed: for example—take that of 1841, in which 1,800 died; five of whom only were natives of the city; one aged three weeks; three two years, doubtlessly born of non-creolized parents; except one, a doubtful case, in Lafayette.

1841.

1842.

In 1843, among six hundred and ninety-two deaths from yellow fever but two are certified as having been born in New Orleans, and these two were proclaimed, in a public journal, to be two errors, by the compiler of the dead list for that year.

Creolization.

City creolization, native or acquired, has hitherto carried with it proctection against epedemic fevers of almost every kind, as typhus, congestive, or cold plague, bilious remittent, and even intermittent; the latter, however, is more or less prevalent in the rear of the city, where the cypress swamp and the population meet face to face, contending for possession. Be the cause what it may, hitherto almost complete immunity, a few sporadic cases of these fevers excepted, has been common to all not new-comers. As this immunity is uniformly indicated by the earlier writers upon Louisiana, before the invasion of yellow fever, the exclusion of the latter, if a possibility, would not in all probability be replaced by the former; indeed, immigrants before 1796, were only subject in a few cases to a slight fever, never mortal, as I have more than once proved by French writers of undoubted credibility. These authorities have not failed to mention infantile lock-jaw, and a few other diseases of the city and country, which, as they affirm, formed the only exceptions to the extraordinary salubrity of the climate in former times.

Collateral protection.

Ante-epidemic era.

Creolism.

City creolism is here used as a more precise and restricted term than acclimation, and denotes that immunity from yellow fever, whether transmitted from parents born and resident in the city, or that immunity acquired by long residence, with or without having suffered an attack of the disease; in any case it is for most part hereditary—the exception consisting of a susceptibility to a slight fever as proved in 1853.

City creolization is not peculiar to New Orleans, Mobile, Charleston, Havana, or Vera Cruz; but there are many new Southern towns, or rather new aggregations of new-comers, where its influence is less obvious, certain, and uniform, or places where it may fail altogether.

Exemption.

Congenital city creolism, that is, the constitutional modification incidental to the being born of Creole or thoroughly creolized parents, with continuity of city residence, exempts the individual from yellow fever with nearly the same uniformity that vaccination prevents the small pox or the varioloid. The varioloid is, as all know, modified small pox, happening to one who has undergone vaccination, or the small pox previously, the frequency of which is probably as great as the frequency of yellow fever among city Creoles who have never absented themselves for one or more winters in Northern climates.

Rural nativity

Country Creoles: All born beyond the limits of the city are susceptible to yellow fever on coming into the city or into a village when yellow fever prevails. In 1853, yellow fever has, for the first time, perhaps, prevailed to some extent in the rural districts, remote from towns, among isolated persons who had not visited

them. But in almost all of these instances the disease prevailed in aggregations of people which are virtually towns, as the plantations where the population is concentrated at one centre, often forming a village of from 100 to 500 or more persons. But in the present state of our knowledge of the prevalence of yellow fever in the rural districs in isolated families, scarcely anything can be pronounced positively as to the extent or frequency of attacks among such as had no connection with towns as visitors. Whether, on the other hand, city Creoles who have removed to the country, who have never resided one or more winters in Northern latitudes, have in any instance suffered an attack in the country, or on returning to New Orleans, is unknown. Second attacks are rare.

Plantation villages.

Rural epidemic.

Creolization in the city, with or without having had yellow fever, is equal as a protection against yellow fever, to congenital or native creolism. This immunity is usually acquired in less than ten years, often in five, but to this rule very many exceptions occurred in the extraordinary or exceptional epidemic of 1853.

City immunity, native or acquired in similar cities, as New Orleans, Charleston, Mobile, Pensacola, Havana, Vera Cruz and other places in the present limited yellow fever zone, is probably identical and mutually protective in all such places, while nativity in cities once in the yellow fever zone in which yellow fever has not been prevalent for many years, as in Baltimore, Philadelphia, New York, Boston, Cadiz, Seville and other places, affords no protection.

Mutual urban immunity.

City creolism both native and acquired is, to a great degree, as before remarked, hereditary or transmissible from parents to children. At least the exceptions to this law are few, and fatal results almost unknown, as may be proved by the bills of mortality, though this is like many other indubitable truths boldly denied, particularly since the decline of the epidemic of 1853—the most mortal, erratic, and extraordinary ever seen in New Orleans. It will have been seen what warrant the terrorists have for denying creolization.

Hereditary immunity.

Setting aside the epidemic (of 1853) and reasoning from what is fully proved by the past—the best expositor of the present—it will be seen what little foundation there is for the utter rejection of creolism and acclimatization, which in former years was rung and is still ringing in the public ear.

That many Creole children had, during the epidemic of 1853, a fever—a slight fever—yellow fever if you please, known as such rather by the co-existence of the epidemic than from any severe symptoms among these children—a slight fever never yet described, having generally of but one paroxysm, lasting from six hours to one, two, or three days, scarcely ever requiring medication. That a few of these cases acquired an alarming violence and even proved fatal, is most true—most deplorable. It will, no doubt, be found upon a full examination of these fatal cases, that nearly all belong to the following classes and conditions: although born in the city, their actual residence has not been continuous, but has vibrated like a pendulum between the country and the town, between Northern schools and cities and New Orleans; or they have been born of unacclimated parents whose continuous residence has been less than ten years, often not that many months; or they have been born of parents one of whom is not acclimated; or, finally, they have been born while the parents resided temporarily in New Orleans, (constituting a large class) and, hence, called Creoles, who, subsequently having come to the city, fell victims, and, hence, appear in the mortuary certificates as natives of the city.

Creole childhood.

Unknown names and nativities.

No one acquainted with New Orleans, as it is, can for a moment believe that the large class of nameless persons included in the mortuary returns, called the unknown, as having died of yellow fever, could have been Creoles or residents for any considerable period. A Creole whose name had never been known would be a phenomenon.

The very gentlemen who contend that the Creoles are no more (one doctor told me that they were less) exempt from yellow fever than strangers, contradict themselves by their own written certificates. Figures contradict them. Every list of deaths, including that of 1853, contradicts them. The demon of contradiction can go no further.

CHAPTER IX.

YELLOW FEVER ETHNOGRAPHY—RACES, AFRICAN AND INDIAN.

Negro race.

The immunity of the African race from death by yellow fever is a problem unsolved, but of the highest import in physiology and ætiology. Whether this immunity be owing to color, or to an unknown, transmissible and indestructible modification of the constitution originally derived from the climate of Africa, or from anatomical conformation or physiological law, peculiar to the race, is not easy to determine. It does not appear that yellow fever prevails under an African sun; although the epidemic of New Orleans in 1853 came well nigh getting the name "African yellow fever," "African plague"—it was for weeks so called!

Immunity.

1841.

Although non-creolized negroes are not exempt from yellow fever, yet they suffer little from it, and very rarely die. On the other hand they are the most liable to suffer from cholera. As an example of the susceptibility of this race, take the year 1841: among 1,800 deaths from yellow fever, there were but three deaths among the blacks, two having been children, or 1 in 600, or 1 in 14,000 of the whole black population.

1838.

The same immunity from death in this disease is enjoyed throughout the yellow fever zone by the black race: for example, in the epidemic in Charleston in 1838, the official report shows that among 538 interments of yellow fever subjects, only seven were blacks, or about 1 in 50, and these were, probably, as usual, not city Creoles. This is, however, an extraordinary mortality compared with the same class in New Orleans.

1853.

Numerical analysis.

In order to ascertain the proportional number of deaths from fevers among blacks in 1853, I adopted the following plan, comprehending the six months ending November 1st: I analyzed more than the one-fifth of the list of deaths by Mr. Maginnis, beginning with the letter A and ending at D, running through all the twelve cemeteries for the six months. The result is fourteen deaths among blacks from fevers of all kinds; two of whom, a child aged eight days, and one aged ten months, were born in New Orleans; of the residue four are mentioned under the term "child," one aged nine months, and one two years; one nine years; the birth place of the residue, one excepted, unknown. The two born in New Orleans that died aged eight days in the one case, and ten months in the other, were doubtlessly not the children of creolized parents, many of whom are brought from more Northern States and kept here for sale, not only to the citizens of the city, but to planters in

the country. Among thousands of these, it would be not surprising that fourteen should die of all kinds of fever in six months, if no epidemic had existed, and if the entire race had been insusceptible to yellow fever. In round numbers the total mortality had been 2,500—that from yellow fever about 2,000—during the period above indicated, in which fourteen blacks, many of whom were infants, died of fevers of all varieties, in a black population of 30,000.* Had these deaths all been from yellow fever, they would not, so far as this worst of epidemics goes, affect the argument that while the black race is susceptible to yellow fever, if born out of the city, death is, from this disease among them, a very rare occurrence; a majority of city practitioners never, perhaps, saw a single fatal case.

Races.

The Necropolis of New Orleans represents all the fundamental types, if not every variety of the human races, Caucasian, American, Asian and African—all of which except the latter—become the ready victims of yellow fever, the creolized excepted. I have seen, in the same day, the copper-colored American from the low lands of the Mississippi and the Scandinavian from the icy mountains of Norway, dying of the epidemic. The Indian race is equally susceptible as the white race to yellow fever, although some writers have denied this.†

Indians.

Choctaw.

Dr. Cartwright, of New Orleans, formerly of Natchez, in his account of yellow fever in the latter city in 1823, published in the Medical Recorder, says "five of the aboriginal inhabitants, belonging to the Choctaw tribe, came into the city during the prevalence of the epidemic, and afterward encamped two miles from the city; four took the disease, three men and one squaw. They were most barbarously burnt, [by themselves as a cure.] The squaw was covered with large ulcers, produced by fire, from the pubis to the chin, and was writhing and groaning by the side of her grave, which the well Indian had dug to put her into, as he had prognosticated her death; but the men bore their pain in sullen silence and with savage fortitude—disdaining to disgrace themselves, as men and warriors, by imitating the groans of the squaw, but applied to their own skins the lighted spunk, nor seemed to feel its corroding fire."

Soon after the discovery of America, indubitable records show that the Indians of St. Domingo and other islands were desolated by the yellow fever.‡ The late Noah Webster has shown that this disease prevailed amon the Indians of New England, in 1618 and in 1746, and at other periods.

CHAPTER X.

METEOROLOGICAL TABLEAU OF THE SUMMER OF 1853.

1853.

It is not intended to give the special meteorology of New Orleans during the year 1853. It is impossible to connect the temperature of any locality with yellow fever, so that the appearance of a known degree of heat or rain, will invariably prelude or cause the appearance of that malady. Although the yellow fever zone

* The total colored population by the city census of 1852 was 29,174.

† T. Y. Simonds, M. D., in the Charleston Medical Journal, (November, 1851,) says "I have never yet heard of an instance of real yellow fever prevailing among the copper-colored race, or American Indians."

‡ Breton, in his dictionary of the Carib language (1655) explains the Indian word for yellow fever, literally coup de barre, one of the names adopted by Du Testre and others, expressive of the muscular pains of yellow fever, as if produced by blows from a stick. [De Jonnès Monog. 41.]

Austro-boreal axis of yellow fever.

whose austro-boreal axis from Rio de Janeiro, in South America, to Rochefort, in France, is nearly seventy degrees, not to mention its still greater extension in 1803 to the Siberian peninsula of Kamtchatka, lat 56° 30′ N., about ten degrees further North, thus covering eighty parallels of latitude, including about three-fourths of all the land, and a still greater proportion of the inhabitants of the globe, still the disease has not appeared within these vast expansions, except in a comparatively few places. Towns the most dissimilar in topography and even in temperature, suffer

Heat.

or escape attacks in an inexplicable manner. But of all appreciable conditions, temperature is generally regarded as the most important; nay, it is regarded by many as the true cause.

The most remarkable feature in the weather of the summer of 1853, is that of the diminished heats in the whole tier of Southern States bordering on the Gulf of Mexico in which yellow fever prevailed, compared with the Western, Middle and Northeastern portions of the Republic. L. Blodget, Esq., in charge of the Meteorological Department of the Smithsonian Institution, in an able paper "on the climatic conditions of the summer of 1853, most directly affecting its sanatary character," an official report for June, July and August, from ninety meteorological stations from Canada to Florida and Texas,* shows that in the fourth week of June, the maximum heat from New York to Savannah gave an average of 95.° Now the corresponding week in New Orleans gave, according to Lillie's tables for the corresponding week, only 92° as the mean maxima, and throughout the whole epidemic the average never for any week equaled that of the above mentioned central zone where no yellow fever appeared. The average maximum temperature of the week ending August 26th, in New Orleans, was 91.° while the mortality was the greatest, amounting to 1,667, or more than 238 as the average per day, the temperature averaging one degree more than that of the week ending September the 15th, during which the mortality was only 411, averaging less than 59 per day. The average maximum temperature of the week ending October 22d was 82,° two degrees more than that of the preceding week, though the number of deaths did not differ more than three for that period.

Blodget.

Mr. Blodget says: "A most extraordinary heat occurred on the 29th and 30th of June, beginning earliest at the West by a day and a half for the distance from St. Louis to Washington. This extreme was central in the latitude of Washington, and was limited at Savannah on the South, and Burlington, Vermont, on the North, attaining 96° to 98° in Tennessee, Kentucky and Southern Ohio, and 99.5° to 102° at Washington and in Eastern Virginia and North Carolina. This is without any known parallel in the records of temperature here, and is several degrees above any recorded temperatures at New Orleans, Mobile, or Savannah. The mean of June was much above the normal one, attaining a maximum of excess in Wisconsin and Illinois of nine degrees. In August, a period of general excessive heat occurred, beginning, as usual, earlier at the West. The maximum in Illinois and the adjacent States was 90° to 94°, from the 8th to the 13th: in Ohio and Kentucky nearly the same; and passing Eastward, the district of greatest excess was central New York. The temperature was below 80° at Cedar Keys, Talahassee, and Pensacola, Floridia, through these days, and at no place South reached 90°. The mortality from the effect of great heat with great saturation was frightful—some term more compre-

*N. Y. Jour. Med. Nov. 1853.

hensive than *sun-stroke* seems required to designate the fatal congestion, or whatever may be the immediate cause of death, in these cases. * * * Mean temperatures and amount of rain are given in a tabular form from over ninety stations. The normal curve [or mean] at New York and Northward is a rise of 4° to 4.5° from the mean for June to that of July, and a fall of 3° to that of August. At Philadelphia and Southward it is somewhat less, decreasing to the Gulf coast, where the curvature disappears."

It was not until June that the yellow fever showed itself even in the sporadic form to any considerable degree—the week ending on the 30th of the month gave as the average of maxima 92° in New Orleans. Now, Mr. Blodget's tables will show that on and about the day aforesaid that the maxima temperatures were as follows at the places indicated where yellow fever did not show itself: Alexandria, Virginia, 95°; Knoxville, Tennessee, 94.4°; Oberlin, Ohio, 95°, 17th, 97°; Baltimore, 92.5°, 22d, 96.2°; Camden, South Carolina, 97.6°; Sparta, Georgia, 97°; Eutaw, Alabama, 101°, (the day before 104°;) Lebanon, Tennessee, 95.90; New Harmony, Indiana, 28th, 97.5°; Bloomfield, New York, 21st, 99.5; Philadelphia, 95°, 20th, 96°; Sparta, Georgia, 97°; Brooklyn, Michigan, 21st, 97°; Poultney, Iowa, 20th, 97°. On the other hand many Southern towns were compartively cool—those which escaped as well as those which suffered from yellow fever. Jacksonville, Florida, on the last day of June, 84°; Pensacola, 85°. Again, compare the 15th of August—Smithsonian Institution, 91°, (the 14th, 94°;) Alexandria, Virginia, 92.3°; Savannah, Georgia, 77°; Jacksonville, Florida, 87°; Culloden, Georgia, 82.40; Austin, Texas, 82°. This period including the preceding two weeks and the week succeeding was the hottest part of the season in New Orleans, the maximum ranging from 93° to 94°, being much greater than that which attended the invasion of the epidemic. The week ending the 28th of July, gave an average of 87°, although the mortality at that time from yellow fever fell but little short of 1,400 during the month.

If we compare the summer heat (June, July and August) of the yellow fever zone with Northern latitudes, where yellow fever did not appear, it will be found that even the mean temperatures of the entire hot season correspond very nearly in many instances: the mean of New Harmony, Indiana, for June, 79 3°, nearly the same as Pensacola, which is 80°; Baltimore 77.7°; Savannah 79°; Lebanon, Ky., 79.5°; Camden, S. C., 79.3°; Danville, Ky., 79.3°; Mount Vernon, Ohio, 78 9°, agreeing within an inconsiderable fraction with Cedar Keys, Talahassee, Pensacola and Jacksonville, (78.9°) in Florida, and Eutaw, Ala., Austin, Texas, and other places.

Rain.

The quantity of rain which fell in New Orleans in July, August and September, amounted to 16.81 inches, nearly two-thirds of which fell in July, which is usually the most rainy month in New Orleans—nearly one-third fell in the next month, leaving but the fraction of an inch for the latter, which, with the month of October, is the driest season of the year.

On comparing July and August, the two great epidemic months in New Orleans in 1853, it will be seen that there was nothing peculiar—nothing that can account for the epidemic in regard to the quantity of rain, which was in some places greater or less than in regions free from the fever, and sometimes similar. In these two months there fell 16.81 inches of rain at New Orleans; at West Point, N. Y., 18.28; at Richmond, Mass., 14.235; at Montreal 10.191; at Philadelphia 9.37; at

Richmond, Va., 8.63; at Anne Harbor, Mich., 5.06; at Bedford, Pa., 3.877; at Savannah, 14.632; at Jacksonville, Fa., 10.1; at Pensacola, 4.078; at Cedar Keys, Fa., 15.1; Eutaw, Ala., 14.739, &c.

It is the more necessary to dwell upon these facts, because writers often most pertinaciously argue that yellow fever is owing to rain, when a rainy season and an epidemic happen together, as in 1839, when, during an epidemic, it rained almost every afternoon for nearly two months. On the other hand, when an unusual drought and a severe epidemic prevailed in 1837, it was argued in like manner, that the absence of rain for a like period (with the exception of a few showers) was the source of the evil.

CHAPTER XI.

SANITARY TABLEAU OF THE BUILDINGS AND TOPOGRAPHICAL IMPROVEMENTS OF NEW ORLEANS.

Buildings.

How the style of building in New Orleans has so long escaped the legislator, the grand jury, the landlord and the sanitarian, is marvellous. About ninety in every hundred houses even in the richer portion of the city, are constructed in a manner that must be condemned in any climate, but in none so much as in this city, depressed as it is below the high water mark of the river almost every where, and in the rear nearly on the sea level. The lower floor, which rots about four times in ten years, in a great majority of the houses, especially the stores, rests on the humid soil, sometimes at a lower level than the streets, no air being admitted underneath!

Soil.

The fresh water, newer pliocene, being largely mixed with decaying animal and vegetable matter, moistened by rains, and infiltrations from the river, gutters and swamps, generates perennial crops of *algæ*, *fungi*, *infusoria*, blight, mildew,

Fungi.

mould, &c., which abound in, under and around the lower story of these unventilated houses, where, indeed, crops of mushrooms would flourish, were they not repressed by the tread of the tenant. Hence goods rust and spot, delicate colors are discharged; health, too, is deteriorated from moist and insalubrious exhalations during the day, and at night, as many persons sleep upon these decaying humid floors.

Physicians in visiting the poor, especially in depressed portions of the city, must have often found the flooring of houses floating, and sometimes, after rains quite covered with a water too filthy and offensive for description—laboratories for generating carbonic and other deadly gases, predisposing to disease, and rendering recovery from any kind of sickness tedious, too often impossible. What drug can supply the place of pure air, pure water, dry sleeping and business rooms?

The lower floors (on which the principal business of the city is done, and on which is stored the most valuable merchandise) resist decay but a few months, whereas the most perishable kinds of wood, and even cotton and linen fabrics, with their original colors, will, if kept dry, last for thousands of years, as witnessed in the tombs of Egypt, where the cerements of the dead are comparatively sound, while their coffins (made of sycamore, a wood that speedily rots, where moisture is present) are as sound as they were thousands of years ago, although they had been placed in excavations, often little elevated above the inundations of the Nile

It would appear from a cursory glance at many new business houses now going up in New Orleans, that instead of having one or two feet of free air circulating under the lower floors, the latter have been sunk to a level, lower, if possible, than usual.

In some cities deep cellars are dry. The depressed, inclined plane on which New Orleans stands, below the high water line—the river before, the swamps behind, subject to sudden inundations from enormous rains, all combine to prove that floors ought not to be placed directly on the mud, though in other cities this mode of building may be less injurious. In New Orleans it ought to be interdicted by law. It is to be regretted that the two conditions that ought to be most desiderated, are the most neglected—the two conditions most necessary to the preservation of health and merchandise, namely: elevation and dryness—drainage and the free circulation of air in and under houses.

Elevation and dryness.

Although the climate of New Orleans and lower Louisiana has been regarded as unusually humid, it might with more justice be viewed as being, for a considerable portion of the year, remarkably dry. Mr. Darby maintains that after the season of inundation, lower Louisiana is, for eight months of the year, drier than any woodland in America.* The desiccating process in New Orleans is naturally rapid, as might be anticipated from its almost constant breezes, elevated temperature, and great number of cloudless days. Were the swamp-zone cleared, ditched and drained, these conditions so favorable to evaporation under a powerful sun, would make the soil as dry as it is rich and productive.

Desiccation

Enough is already known of the science of Hygiene to warrant the conclusion that our crowding filth, a want of ventilation, incomplete drainage and humidity must be injurious to the health and detrimental to the physical comforts of the citizens of New Orleans. Healthy individuals and still more the sick ones need pure air, both when there is and when there is not an epidemic.

Hygiene.

Effectual under-ground drainage is, as it seems to me, scarcely a physical possibility in New Orleans—if possible, the expense could hardly be paid by the treasury of the United States, and if accomplished, it would prove to be an intolerable nuisance. A gentleman recently from Paris, and, perhaps, the ablest quarantinist in New Orleans, informs me that in Paris where underground drainage, with a soil elevation and declivity so vastly superior to New Orleans for this purpose, is mischievous. The Parisians find that the filth of the city accumulates in these subterranean sewers so as to send forth the most offensive and deleterious emanations. Hence, they prefer, after costly experiments, surface drainage, and wash off the filth into the Seine. One heavy rain in New Orleans would change this abstract theory into a concrete mass of filth, which would fill these subterranean canals, which, lying as they must, below the level of the river, and even below the level of the sea and lake, would send forth emanations strong, but not wholesome. These subterraneous passages would afford a good living and secure habitation to the infusoria, algæ, conferæ; vast reservoirs of animal and vegetable organizations, good for microscopical investigation, but not fit to be smelled.

Under ground drainage.

The sewers (*cloacæ*) of ancient Rome passed under the whole city; the *cloaca maxima* of Tarquin, was sixteen feet broad and thirty high, built of hewn stone, three miles long, one mile through a mountain a thousand feet high; the mouth of this sewer is still seen where it empties itself in the Tiber. These sewers, placed under the supervision of the *curatores cloacarum*, had under-ground side walks.

*Stat. La. 99, 100.

In the National Clycopædia, (of London, 1851,) it will be seen that the sewers of this metropolis are four feet three inches high, by two feet three inches wide, to allow a man to pass through them for the purpose of inspecting and cleansing them; others are twelve to fourteen feet high. "The inclination of sewers should always, if possible, be sufficient to enable the water to run freely, and to carry off the solid matter that usually enters with it. In the metropolitan sewers the inclination varies from a quarter of an inch to an inch and a quarter in ten feet. It is some times very difficult to obtain a sufficient inclination to a sewer. The depth of the Watling street sewer is from thirty-three to thirty-five feet. In many cases, however, there is a space of not more than three feet between the surface of the road way and the crown of the arch of the sewer. Wherever it is practicable new sewers are built at a considerable depth from the surface." This would be particularly necessary in New Orleans, where there is much heavy drayage. Now let us see what is the fall from New Levee street, the highest part of the city, to Claiborne street, not to mention streets still more remote, yet on nearly the same level. Fall to Claiborne about nine feet six inches: suppose a sewer four feet six inches high, reaching within eighteen inches of the pavement or surface; the bottom of this sewer will then be only five feet above the level of Claiborne street, reckoning from New Levee, the highest of all streets—allow a fall of only half an inch for every ten feet, much of which is expended near the levee: the distance to Claiborne being 4,338 feet, the fall would be about eighteen feet; or if we adopt the London standard of one inch or one inch and a quarter (to scour off the filth) the fall would be thirty-six to forty-five feet, and if carried through the Metairie ridge, would average for the whole city a fall of about 90 to 145 feet, and to the lake 180 to 290 feet. Observe that the fall from the levee to Claiborne is all expended but about ten inches in the first half of this distance! and that even this terminus, in Claiborne, has been covered two feet deep by the lake during hurricanes, the water having reached within five or six squares of the levee, so that under-ground sewers would have been inoperative except along a narrow belt next the river. If the sewers were placed at the usual depth, the refluent water of the lake would have filled them up completely to their roofs! The bottom of a sewer at the ordinary depth in Claiborne, and in half of the streets of New Orleans, would be below the ordinary sea level! They would be little better than inaccessible reservoirs for collecting the filth and alluvium washed off from the inclined plane on which the city stands Every heavy rain would deposit an immense amount of detritus, which, in the absence of a strong scourring current, would fill all the sewers with solid matter which would emit the most offensive odors.

Surface drainage.

By adopting the experimental instead of the verbal or paper method of drainage, and using a moderate amount of common sense, much digging, and a good deal of capital, just as the Dutch have done in Holland, by which the land has been wrested from the dominion of the sea, New Orleans and its environs as far as Lake Pontchartrain, might be changed from mosquito-lands and putrefying sheets of water, to horticultural, pasturage and meadow lands. The almost uniform levels of this district near the lake is not without some compensating advantages, inasmuch as expensive gradings and deep cuttings for ditches will not be necessary. The elevations and inclinations of the Metairie ridge, may prove advantageous rather than injurious, in several points of view, in giving direction to the waters; while the natural plane on which New Orleans is built, has a sufficient inclination and prolongation towards the lake to give a strong current to the rain water from the levee, as far as the cypress swamps.

With suitable lateral and lake levees or dikes, efficient draining machines, and ditches passing through and connecting the lowest levels, there can be no doubt, it may be repeated, that the swamp district lying between the already leveed river shore and the lake could be completely drained.

St. Petersburg.

There is no part of this district which prevents difficulties, in surface drainage, comparable to those met with in the under-ground drainage of the site of St. Petersburg, a city which was founded in the midst of deep and wide marshy forests, at a level so low that during storms it has often happened since 1715, as it did in 1824, that hundreds of lives were lost by drowning, ships having been stranded in the streets or dashed to pieces against imperial palaces, the inundations having covered the highest grounds in the city.

Amsterdam.

Peter the Great having visited Holland, where he witnessed the system of dikes and drainage in that country, determined to found another Amsterdam, though in a most unfavorable locality. Amsterdam, once the most commercial city of the world, was, as all know, built on piles, on account of the depth of the marsh in which it was founded. The city hall, now the royal palace, constructed in 1648 rests on 13,659 piles, and a church recently built has nearly half of that number.

Peter having rejected piles, adopted a different and almost impracticable mode of draining the site of a city that should transmit his name to the remotest posterity, namely, under ground drainage. Instead of trusting to an abstract ukase, or act entitled an act, &c., he built him a little hut, in 1703, on the river Neva, between Lake Lagdoga and the Gulf of Finland, where, seven years after was built the first brick house, and where he sacrificed one hundred thousand lives, chiefly owing to his method of under-ground operations, the earth having often caved in, burying the workmen. Canal excavation with proper hygienic regulations, fortunately is not necessarily attended with unusual sickness. Before the epidemic broke out in New Orleans, from fifty to seventy-five men were engaged in excavating the great basin, 300 feet wide and from 8 to 12 deep, near the junction of the Bayou St. John and the canal Carondelet, a mile from the city, and a still greater number were similarly engaged at the junction of the bayou and Lake Pontchartrain, among whom no sickness appeared.

Harlem Lake.

In several countries large lakes have been drained. Harlem, a navigable lake, almost as deep as lake Pontchartrain, fourteen miles long and nearly as wide, was but a few months ago disappearing rapidly, and will soon become a dry, fertile basin or plain, though it is still called in the maps of Reece's Encyclopædia "the sea of Haarlam," and all by means of one or perhaps two steam engines which pump out the water.

CHAPTER XII.

CONTAGION—INFECTION—MIGRATION.

Explanations.

There is no probability that any satisfactory conclusion will ever be established as to the contagiousness of yellow fever or the contrary, so long as the words contagion, infection, miasma and the like, are used in a latitudinarian, vague and undefined manner. Contingent contagion, conditional contagion, occult changes, unknown predispositions or ever varying circumstances, as heat, rain, drought, swamps,

vegetable and animal decomposition, assumed emanations from the sick, and the like, are so mixed up as to afford ground for endless controversy. If one explanation or assumed condition shall fail, another is held in reserve to be called in to aid the wily logician in his extremity. The dry season of 1837 and the wet one of 1839 served at the time to explain two epidemics.

A word is, in an investigation of this kind, best explained by a thing, a type or example, as an unerring criterion, and not by another word equally vague and darkly enveloped with unessential, contingent, wholly unknown or ever changing hypothetical conditions. If the supposed contagion of yellow fever be by contact, as the itch, or by a volatile contagion, as in small pox, let it be tried by these typical or fundamental tests, and not by the assumption of one or many other contingent circumstances which may happen as coincidents, not causes, nor even invariable accompaniments.

Contagion. Contagion in its most literal and restricted sense implies the actual contact of a well person with a dead, or sick person, or his apparel, etc, by which a specific posion is transmitted from one to the other, reproducing a similar disease, as in small pox, cow pox, itch, etc. In a more enlarged sense, this term includes invisble emanations from the sick consisting of a specific poison, doubtlessly, dissolved or suspended in the air, and capable of reproducing a similar disease in any indefinite number of persons who come near the patient, of which small pox again affords the most complete typical illustration. Here the fundamental idea of contact is, perhaps, real, though unseen.

Proximity. Another type or criterion of contagion is this—it cannot act except within a very circumscribed space, in any season, latitude, or climate; it may be limited by isolation from, or non-intercouse with the healthy; its extension probably might reach from pole to pole, if all could be brought in proximity with a single sick individual, although the emanations from his body at a few feet from the same, mixing with the atmospheric ocean, become harmless, not epidemic.

Infection. The word *infection* generally used as synonymous with the word *contagion*, has, too often, played a conspicuous, if not a satisfactory *rôle* in the vague and inconclusive disputations of yellow fever quarantinists. If the word infection mean an emanation of a specific ærial poison from the sick, giving rise to a similar malady in the well, it must be precisely the same as contagion; but if it mean an impure air arising from an animal or vegetable source, or from both combined, then it is but another word for miasma, malaria or bad air. The labored attempts to explain this word—the bad faith in which it has been used—at one time for contagion, at another for the bad air of a sick room, a sick city, a vile scent, or paludian exhalation, go to show that it is a most perfidious word, the shibboleth of dialecticians—a word pregnant with mental reservations. It is the limbo of countless pamphlets, books, and laws upon yellow fever quarantine—the lumber of the last and present centuries.

If *infection* be used to denote the contamination of the atmosphere of a room, or of an urban district or focus, with or without offensive scent—an emanation from vegeto-animal decomposition, not an emanation of a specific nature from a sick man, which, in any climate, season, and latitude, produces a similar malady in the well, then the word becomes intelligible. Such contamination, however, does not originate a strictly contagious disease, though it may, and often does, aggravate the latter. Seclusion from sick persons does not ensure exemption, while the individual lives in the infected district. The locality, not the person, is dangerous.

Migration or flight, unless in the beginning of the epidemic, is of questionable

expediency, for many reasons. For although the chance of suffering an attack may be slightly diminished by leaving the city after the epidemic has become general, the chance of a cure in the city is increased. The excitement and fatigues of traveling are likely to develop and aggravate the disease. Migration.

The epidemic of 1853 has clearly proved that when the yellow fever zone is expanding throughout the Southern States, while the epidemic is steadily declining, so as to be nearly extinct, flight to New Orleans is a prudent measure. When the epidemic was marching upon the towns and villages of Louisiana, after its declination in New Orleans, many persons came to the city for protection, and to the discredit of the doctrine of contagion, escaped.

Early migration is an all-important measure. It would be too tedious to give even a summary of the multitudinous and striking examples furnished by the late Dr. Chervin, of the efficacy of migration, both in the peninsula of Europe and elsewhere. Baron Dupuytren, in a report to the Academy of Sciences in Paris in 1825, says: "We regard as incontestible the principle of evacuating immediately the places where yellow fever is declared to be, and every thing for this purpose should be adopted. The utility of such a measure must always justify its vigorous execution." Dr. Thomas, after thirty years' practice in New Orleans, from 1818 to 1848, declares* that he never saw or heard of a well established example of the communication of yellow fever to any person in the country by patients who had contracted the disease in the city during a visit while the epidemic was prevailing. The same exemption proved constant when unacclimated residents of the city who fled, but nevertheless sometimes sickened and died, or recovered in the country, after having lived in the same houses and slept in the same beds with the country people. Chervin. Dupuytren. Thomas.

The completion of the railroads now in progress will enable both the urban and rural population of Louisiana, at a small expense, to dodge yellow fever in almost the most literal sense of that term, by leaving the infected localities. Railroads.

The most salubrious retreats during the summer season for the citizens of New Orleans, might be expected to be found in the pine region bordering upon the lakes and the Gulf of Mexico, notwithstanding the single exception of 1853. The late Dr. Drake gloried in these pine woods, as "the healthy localities"—"dense and lofty forests, presenting to the eye a vast system of intercolumniation, which, seen at night, by the running fire that occasionally consumes their shed cones and long leaves, with the dry grass among which they have fallen, presents a grand and striking spectacle. Such are the celebrated *Pine Woods*,* to the protecting influence of which the people of New Orleans and Mobile commit themselves for safety in yellow fever seasons, expecting to enjoy an equal immunity from intermittents and remittents."

Now, although these piny platforms are not beyond the reach of epidemic yellow fever occasionally, they are far more salubrious abodes in the heats of summer than the far off sultry cities of the north, which are every year overcrowded by southern absentees. The coniferous pines which lie at the doors of New Orleans, and which the good doctor, a lover of Nature, admired, are totally ignored by the lovers of fashionable saloons. What a pity that his account "of the people of New Orleans and Mobile" is not a true one!

* Traité Pratique de la Fièvre Jaune observée à la Nouvelle Orléans, Paris, 1848.

* A Mississippian says that in these Pine Woods, "It is too healthy to support a physician, too honest to need a lawyer, and too free from debt to furnish any salary to the clerk of a circuit court."

CHAPTER XIII.

THE HISTORY AND MYSTERIES OF QUARANTINE—CLASSICAL MODE OF ATTACKING EPIDEMICS.

Quarantine in Europe, established during and after the prevalence of the black plague, was enjoined under the penalty of death and confiscation; but it was found, after long trial, unavailing, as the pestilence returned repeatedly. The law required the patient to be taken out of the cities into the open fields, there to die or recover. No one under pain of death could visit the sick, unless specially appointed for the purpose. An individual coming from an infected district was put to death. Many articles of household furniture were burned, others were exposed to sun and rain for a specified time.

Law.

Forty.

If quarantine be not a fraud on the many, for the benefit of the few, it is at least a superstition devoid of any philosophical evidence in some of its most fundamental details—as for instance, in its mystical adherance to the number FORTY, adopted in the dark ages (1485) by Councils or Boards of Health, being a monstrous compound of medical, legal, and theological fancies, founded on the duration of the forty days flood; Moses' sojourn of forty days on Mount Sinai; Christ's fast of forty days in the wilderness of Judea, and the Lent fast of forty days in the church ceremonial. Hence the Italian *quaranta* from the Latin *quarantina*—forty days more or less—during which time persons, animals, goods, letters, and ships are interdicted,

Detention.

confined, restrained by *cordons sanitaires*, or lazar-housed to the scandal of a fast age, which chides the tardy movements of the locomotive and steamship, and is barely satisfied with the velocity of lightning, which brands with the word *fogy* every thing devoid of rapid progression.

Old Britain is more progressive than Young America in quarantine. The voluminous report against quarantine, especially in reference to yellow fever, by the government committee of Great Britain, submitted in 1852, foreshadows the opinion of the forth-coming report against quarantine even for the plague. That report will show what measures "will supercede the necessity of those greivous interruptions to commerce and international intercommunication which quarantine, so universally imposed on account of plague, has hitherto occasioned."

Since it appears that quarantine is soon to reign nominally, if not practically, in Louisiana, its rules and mysteries deserve to be studied and scrutinized, unless yellow fever can be banished by the mere vote of the majority in the two houses of Legislature. The Mississippi river can not be kept down by the act against inundations, nor raised by an act for the benefit of stranded steamboats. Pass from the abstract to the concrete—from the word to the thing—from theory to practice: What is to be done? Action! Action, only! Disinfect air, earth, skies, ships, goods, and humanity. On with the lustration.

"The most important and valuable method of disinfection is ventilation, and, whatever other may be added to it, this should never be neglected. The reputation of chlorine, acids, lime, charcoal, etc., as disinfectants depends on their property of decomposing the offensive gases which are so often mixed in the atmosphere with the matter of infection, *but it is questionable whether they have any influence on the infectious particles themselves.* However, as the emanations from putrid substances

Methods.

render the body peculiarly liable to the reception of infection, some of these means should be employed where any offensive smell is present. The best of these is chlorine, which may be applied in the form of the chloride of lime, which should be poured over any thing from which odor is emitted; it should be sprinkled about the floor and on the walls; or shallow vessels containing it should be exposed to evaporate in the air; or pure chlorine should be disengaged in the form of gas from the materials from which it is manufactured. Dr. Henry has rendered it probable, by numerous experiments, that the infectious qualities of substances which cannot be conveniently washed, as trunks, packages of valuable merchandize, etc., may be sufficiently destroyed by exposing them to a dry heat of 200° for less than an hour."*

Heat.

As it was during the strict quarantine rule, at the close of the last and in the first decennial period of the present century, that yellow fever attained its cyclic culmination, it may be proper to give a summary of the quarantine laws of that disastrous era; and the more so as the same enactments seem to be desiderated at present. The following extracts from Assalini's celebrated work on plague and yellow fever, translated by Adam Neale, New York, 1806, have been kindly furnished by my friend Dr. Cartwright. "Quarantine Code of Marseilles, Toulon, Venice, Cadiz, &c. There are a lazaretto, lodgings, hospitals, and magazines at the quarantine ground. When a vessel arrives, the captain presents to the Board of Health his certificate where the vessel is from, the number of the crew and passengers, and the kind of cargo. This certificate is taken through a grating by a pair of long pincers, and is not read until it has been thoroughly perfumed and dipped in vinegar. If the certificate gives notice of the plague, the vessel is considered *brute* or foul. The passengers and ship's crew are strictly reviewed at a distance, and placed under quarantine. The merchandize is deposited in the enclosure in the magazine. The magazine at Marseilles, for this purpose, is very beautiful. The passengers are put into the enclosure, with one or more guards of the committee *(comité sanitaire)*, while the ship's crew remain on board. The porters and purifiers of the merchandize open the bales of cotton or wool in the middle, and thrust in their bare arms. They break open the chests and trunks, and expose to the air the bales, &c., &c. After having exposed every thing, night and day, for thirty-nine days *(sérène)*, and after having perfumed the passengers and ship's crew three times they are permitted to enter the harbor. If, during the quarantine, any one falls sick and dies, the quarantine is prolonged or recommenced. If sickness and deaths continue, the laws of health condemn the vessel to the flames. Those who compose the crew, after being stripped of all their clothes, and *having their whole bodies shaved* and washed in sea water, are admitted into the lazaretto. The vessel, with its merchandize, is towed to sea, where it is either sunk or committed to the flames. If any of the porters or purifiers get sick and die, the laws of health pronounce the goods infected, and they are burnt.

Assalini.

Code.

Ceremonial.

"1st, Contaminating; and 2d, non-contaminating cargoes.

"1st. Cotton, wool, silk, furs, &c., contaminating.

"2d. Paper, stuffs and samplers, gold and silver ware, glass, not contaminating. Such things are cleaned by perfuming or putting in water or vinegar.

"Herbs, fruits, and flesh of animals are non-contaminating, and can be purified by putting in water."

* National Cyclopædia, vii. 470—I. Lond. 1851.

"Experience has proved that these seclusions, or shuttings up, have never succeeded in arresting the progress of the plague. This disease always commences by attacking the poor in the most unwholesome quarters of the city; after which the health of the inhabitants in good circumstances become impaired, and at length death levels indiscriminately the poor and the rich. The season changes and all at once the epidemic ceases.

"The Franks, residing in Egypt, to guard against the plague, shut all their doors and block up with care the smallest holes until St. John's day eve. The cats of the family are shut up in cages, like fowls, and if, unfortunately for them, they chance to leave their prisons and make their escape, on their return they are killed without mercy, according to the sanitary laws. Near to the gate of the house are placed three large earthen vases filled with water, a bason with vinegar, a furnace with coal, some odoriferous herbs, antipestilential powders and pastes, iron pincers, a large knife, and other utensils. Each family has a domestic who is not comprised in the shutting up, and who is employed to transact all commissions. He comes every morning with the necessary provisions he has bought at market. The porter, who is the steadiest member of the family, and the most strict observer of the sanitary laws, after being reconnoutered, the domestic descends with the key, opens the door and retires. The domestic enters the court, puts the provisions, such as meat, fruit, fish, herbs, etc., into the vases full of water; the money he puts into the vinegar. Papers, bills of exchange, etc., he puts near the furnace and retires. The porter then shuts the gate. Then having taken in his hand a magic ring, he stirs the meat, fish, herbs, etc., in the water. He then takes the money out of the basin of vinegar, and having lighted the coals, he throws on them some perfumes. He then takes the papers with the pincers and places them over the furnace two hours in the smoke. Sealed letters are purified by piercing them with a stiletto in two or three places and dipping them in the vinegar."

During the black plague in Europe, the merchants carried their money into the churches, depositing it at the foot of the altars, and, returning home, calmly departed this life. The priests dared not touch the golden heaps for fear of *contagion!* (Ozanam. Hist. Méd. iv. 87.) How can a sincere contagionist dare to touch New Orleans bank notes?

The late Cyclopædia of London says that "letters coming from and passing through the plague countries *are opened* and fumigated at the lazaretoes." The affairs of war, love, politics and speculation might be damaged by the *opening of letters*, independently of the detention. In Oriental realms where a belief in fatalism is prevalent, a quarantined man submits to a resistless destiny, in preference to the knout, the bastinado, the bowstring, an impalement or a fusilade.

Boisseau. M. Boisseau, of Paris, in his work on fevers, says that "before and since quarantine was established in France, yellow fever has not appeared, although the communication has been frequent between the ports of Spain and France. This disease fortunately having never reigned epidemically in our country, it cannot be decided whether we have been preserved by quarantine or by circumstances independent of all human intervention."

France. French quarantine, (as good as any,) is, in many of its features, founded on assumption wholly unwarranted by positive knowledge or probable truth. The *patente* or certificate of health given by consuls to vessels leaving port has several distinctions, as the *patente nette* showing that the vessel leaves a country unsuspected

of infection; the *patente brute*, for which quarantine is required on one coast from ten to thirty, on another fifteen to forty days. The *patente suspecte*, from a country not altogether free from suspicion, requiring from five to twenty days quarantine on one coast, and from ten to thirty on an another; the *patente brute* varies from ten to thirty in one place to fifteen or forty days in another, as if there was the smallest proof that these numbers are better than half or ten times the numbers specified.

The theory that something must be done to arrest epidemics has been, is now, and ever will be popular. When, twenty-five centuries since, epidemics began to ravage Rome, the worship of Æsculapius was introduced into that city, with the view of preventing pestilence.

Classical and Howard methods. Brutus, the Roman Consul, more that twenty-three and a half centuries since, sent an ambassador to Delphos, to consult the oracle how the plague, then epidemic at Rome, might be stayed. Had he organized a Howard Association, like that of New Orleans, with a treasury of 1,200,000 *denarii* for distribution for nurses, medicines, food, and medical attendance, himself taking an active part in the work of charity, he might have done the city some service, without calling in the aid of imaginary oracles.

Lectisternium and the Board of Health. Epidemics ravaged Rome in the years 451, 432, and 396, B. C., whereupon pestilence was again attacked vigorously by the ceremonial called *Lectisternium*, or funeral banquet of the gods, instead of adopting the obvious remedies, like those of the New Orleans Board of Health, in 1853, that is the establishment of temporary infirmaries, the appointment of physicians, nurses, and funds for the poor. A terrible pestilence began in Rome, 293 B. C., which, having lasted three years, was attacked by a sort of sanitary commission, consisting of ten ambassadors, who, instead of looking at home for causes or explanations, or sick people in distress, went on a journey to Epidauris, in Greece, the natal city of Æsculapius, near which the god of health had a most magnificent temple.

This sanitary commission having gone up a valley five miles from the city, reached the famous temple always crowded with invalids and priests or doctors, implored the aid of the Epidaurian god. They brought back to Rome the god himself, under the figure of a serpent—a highly valued prize, which, how much so ever it may have delighted the Romans for a time, failed at last, as the plague returned twenty years afterwards with augmented virulence. This sacred snake, placed in a temple built on an island of the Tiber, disappeared among the reeds on the shores of that river.

It is highly probable, however, that the worship of Æsculapius was beneficial in Rome, and would prove so in New Orleans, inasmuch as it requires as preparatory measures, sobriety, fasting, bathing, tranquility, and the like, in order to fit the mind and body for divine intercourse, visions, dreams, and therapeutic revelations. Neither Æsculapius nor his lovely daughter, Hygeia, Goddess of Health, is worshipped in the usual abodes of yellow fever, typhus and cholera, where whiskey, bad air, humidity, crowding, irreguiar hours and the neglect of cleanliness are substituted for the Æsculapian rites, and no act of the Legislature can change this condition of things, how potent so ever may be its language.

CHAPTER XIV.

QUARANTINE AND CONTAGION CONTINUED, WITH ILLUSTRATIONS.

Contagion. At the beginning of the present century and for some years after, the yellow fever element was so mingled with the great concerns of humanity, that it excited the public mind to an unexampled degree; in the cabinet and in the field, in the legislative halls and in medical schools, both at home and abroad, in the colonial governments. It had long been the conqueror of armies and navies, and now threatened to desolate the peninsula of Europe. Its contagiousness was a leading topic on which reports, pamphlets and books went forth, raging like the epidemic itself. Neutrality was scarcely possible in a matter so deeply involving the interests, passions and transactions of humanity. Opinions founded on mere hypothesis concerning the cause of this malady which remains to this day unknown were not for that reason less, but even more positive and dogmatic. Affidavits and affronts, certificates and satires, logic and duels,* personal contagion and personal invective, bad air and worse legislation, divided the professional and non-professional public on this question. The non-contagionists, however, greatly outnumbered their opponents. They, for the most part, controlled the legislation of the States of the Union, by their efforts or their arguments. But no sooner were they off their guard than the contagionists appealed to the fears of the public, and urged the legislature *to do something* for the protection of *the people*, by making laws against the importation of yellow fever, whereupon new laws were often enacted with no effect in this behalf. The anti-contagionists, like Sisyphus, must roll the stone perpetually—then, now, evermore. Now is the favorable moment in New Orleans, just after the

Contingent logic. great epidemic. Something must be done. Formerly, nearly all the worst epidemics in Louisiana, and particularly in the peninsula Europe, took place under the strictest quarantine *régime*. If quarantine goes into effect now, a new era will probably have commenced. For, upon the doctrine of chance or probability, no such severe an epidemic may occur for a generation, and quarantine, if such a thing were really practicable, will not be slow to claim the credit; but if 1854 should be no better than its predecessor, then yellow fever quarantine will be for the hundredth time repealed, and yellow fever will be attacked in a more scientific way—

Oriental logic first by doing no harm, and next by sanitary measures within the narrow range of the human understanding. An eastern monarch taught his subjects that the sun rose only at his command, but he always gave the command at the *proper time*, that is *sun rise*.

Village logic. Contagionists have during this, as well as during all former epidemics, collected facts to prove their theory. A pedlar, from an infected district, arrives in a town—his pack is opened—he, the family, and many of the villagers die of yellow fever. Exactly the same occurrences (which are mere coincidences,) take place a hundred times, where there has been no pedlar—no box of goods opened—no travellers from the infected districts. In one town, a crate of crocks from New Orleans is said to have been the medium of transmitting contagion to the village; but at that very time nearly all the other towns for 500 miles around were falling under the malign

*In some cases both parties, the contagionist and the non-contagionist were killed.

influence of the epidemic. It would be most extraordinary if crates, boxes, passengers and pestilence should never happen to get together—not as causes and Co-incident. effects—but as coincidences, necessary in the ordinary course of business. If the pestilence got into town before the arrival of a bale of goods, the former did not cause the arrival of the latter. If the man who opens the goods dies of black vomit, together with all his family, a hundred other families take the disease without any such apparent exposure, and die in like manner. A planter fences up his grounds, and secludes himself, family, and slaves, and all escape; another does the same thing, and all are attacked.

The great majority of the learned in Europe attributed the black plague to the Astral logic. conjunction of Saturn, Jupiter, and Mars on the 24th of March, 1345; just as many now attribute the late epidemic to events that happen to coincide in time and place.

Those not irrevocably wedded to contagion, might find it useful to study the Effluvia logic. events which have passed before their eyes within the last seven years.

The late Mexican war furnishes the most complete refutation of the contagiousness of yellow fever in the absence of quarantine, so far as negative evidence can go. If the United States Government had tried to devise an experiment, on a vast scale, to ascertain whether yellow fever could be propagated by ships and armies, it could not have achieved its purpose more effectually. In 1846-7-8, this malady existed in Tampico and Vera Cruz, and was very severe in New Orleans in 1847. The troops and the material of the army, leaving New Orleans for Vera Cruz, and Vera Cruz for the interior of Mexico, did not suffer themselves from yellow fever, nor spread contagion through the towns and country. In 1848, thousands of the returning soldiers passed through Vera Cruz in June, where yellow fever existed, and on reaching New Orleans in July and August, a few died out of fifteen thousand who remained in the city and its environs some time, without communicating any disease to the city by means of their goods, army materials, and selves. Thousands thus, without having been quarantined, remained in the city for a time, and quitted it for their homes, in other towns and places, without having communicated the disease to any one. After the reduction of Vera Cruz, yellow fever appeared, and many invalids and sick persons were sent to New Orleans and other places for treatment, in the transports which carried out the troops, yet they did not propagate the disease any where. Thus at least fifty thousand experiments made in Tampico, Vera Cruz, and New Orleans, not to name other places, produced no proofs of personal or other kind of contagion, though in both the first named places yellow fever prevailed moderetely among residents not acclimated.

The Board of Health of New Orleans, in an official announcement, shows, that for the month beginning with the 26th of November, 1853, that 6,767 passengers from foreign parts, chiefly emigrants, had arrived at our wharves in forty-seven seagoing vessels, by the river route. Now, if we add the number which had previously arrived, to the number which has since arrived from sea, the aggregate will scarcely fall below 10,000, while by other routes, chiefly by the river, the emigrants, asbentees and other unacclimated persons, as the steamboat population, coming to the city, in September, October, November and December, forty thousand more may be added, making fifty thousand—fifty thousand living experiments against possible contagion—fifty thousand exposures to all of the possible sources of contagion—the houses, goods, etc., of persons recently dead, including emanations from the sick and dying.

during the decline of the epidemic and during the whole of this period—all proving harmless!

Rio de Janeiro

If the yellow fever be contagious or transportable, why has it not been carried beyond the tropic of Capricorn during centuries of active intercommunication? Why did it appear only *North* of the Equator, with two or three exceptions, always near the line, until 1850, when it traveled for the first time to Rio de Janeiro, which however is within the tropic?

Such vast, yet significant experiments quite overthrow those few cases where the opening of a box or bale of goods is followed by yellow fever—mere co-incidents not causes. There is not the least reason to think that the world, combined for the purpose, could create an epidemic yellow fever, or even a single case in any city street, or house, upon the globe.

The enlightened governments of Europe, whose inter-tropical possessions enable them to judge from large experimental intercouse, have not only gradually lost confidence in quarantine as a preventive of yellow fever, but they oppose it as altogether mischievous—at least such is the case in Great Britain. Quarantine in our own country is nominal, illusory, and never comes up to the theory of real quarantinists. The deception is, therefore, less mischievous than an honest enforcement would be.

The provisional assumptions of contagion, seclusion and quarantine in yellow fever, once altogether proper and wise anterior to experimental tests, are now no longer such. In the hour of despair and ignorance, the theory that the building of a large city in a country where earthquakes and volcanoes prevailed, would prevent them, might be tolerated until after a fair trial. But, if experience prove that earthquakes continue as before, the building of cities for this purpose should not continue.

Ultima ratio regum.

If faith is best proved by works, the contagiousness of yellow fever in New Orleans falls to the ground; because, in practice, it is disregarded both by the acclimated and the unacclimated, inasmuch as doctors, nurses and neighbors visit the sick in the freest and most fearless way, and with equal impunity with those who keep at a distance from the sick. Experience shows, both in hospital and private practice that proximity to the sick does not enhance the danger to one living in "the infected district."

In the rural districts and in the towns where fear was great, and experimental knowledge of the fever little, the people adopted a different line of conduct—the principle of seclusion and non-intercourse. The traveler, denied the hospitalities of the house because he had merely passed through an "infected district" or village, wandered along the road seeking shelter in vain for the night. Towns suffered for want of provisions, because their rural neighbors feared to approach the sick. Sometimes *depôts* were established near these self-beleaguered towns, where the sick or their attendants and families went for supplies and thereby escaped starvation. The artillery placed at the landings and wharves, threatened to send grape and cannister shot into boats and vessels that dared to approach from infected districts. Individuals, as well as towns, carried out the principle of seclusion, and were alike unsuccessful.

Although the quarantine party is to a great extent composed of men of the highest integrity, talent, patriotism and disinterestedness, yet it is feared that some who profess quarantine loudest are, at heart, infidels; if they are sincere they are

not consistent. By what code of morality can they justify themselves in dispensing with quarantine in any case like the following example taken from the Daily Delta, of September 13th, 1853? Capt. Baxter's statement as given by the Editor:

"Captain Baxter left here [New Orleans,] with the Cherokee on the 12th August last, when the epidemic was at its height, with one hundred and sixty-nine passengers, the majority of whom were unacclimated, and liable to the yellow fever. During the voyage, there were ten of the crew down with the fever, and on the arrival of the Cherokee in New York, there being two still sick, they were ordered into the hospital, where one of them died; the other recovered."

Were the crew, and passengers, (without mentioning the ship and cargo) kept 40 days in the Lazaretto, undergoing fumigation? Not at all. Captain Baxter adds:

"They were all permitted to land in New York after eighteen hours, and the sick members of the crew were alone compelled to go into hospital detention."

Such a quarantine is but a kaleidoscopic illusion. If the New York authorities entertained the belief that yellow fever *is contagious*, they would not, in this strongest possible case of importation, have wilfully exposed the lives of half a million of people, unless they are worse than pirates themselves. Their acts more than their words, show that they have no belief in quarantine as a preventive of yellow fever. The same infidelity is obvious in the actions of the few contagionists in New Orleans. They no more avoid yellow fever patients than they do rheumatic patients, or charity. They are better than their doctrine.

As yellow fever appeared in New Orleans at an unusually early period of the season, and long before its invasion of other towns in the Southern slope of the Mississippi valley, the town authorities, in many cases, imposed quarantine laws for their protection, early in August, as Natchez, Baton Rouge, etc. No exemption—great mortality—neglect of the sick—and other evils followed,—some of which grew directly out of quarantine itself, and were by no means creditable to humanity. While experience shows that quarantines do not prevent yellow fever, they do prevent free intercourse with the sick, nursing, attendance, and the physical comforts, by which the disease can alone be combatted with the greatest success. Fortunately, however, humanity is usually stronger than quarantine in practice; non-intercourse, seclusion and abandonment, which quarantine directs, or necessarily implies, are too revolting to the moral sense to be practiced towards friends, neighbors and relatives, and consequently, in yellow fever, these not being carried out in practice, quarantine will always be violated, until morality and charity shall be extinguished.

If quarantinists are sincere they ought not to export any cotton (one of the articles in which contagion is most easily transmitted) because the contagion is in the city every year. A learned physician of New Orleans, Dr. Simonds, has published a table showing the annual per cent. of mortality in the Charity Hospital from yellow fever, in every year for thirty years, ending with 1849—so that the stream of yellow fever, with whatsoever of contagion it may possess, is uninterrupted, no year having been wholly exempt in this institution, not to name the city at large. (Dr. Fenner's Reports, i. 123.)

If New Orleans contagionists succeed in getting the city and State governments to establish the contagiousness of yellow fever by a special act, let the same act forbid the exportation of cotton, even to our enemies, in time of war. In time of peace, it would be still more unjust to send infected cotton to the subjects of her

Britannic Majesty, or to the subjects of the Emperor of the French. It would be still more criminal to export cotton and contagion to Philadelphia, New York, Boston and other cities, as a return for their opulent donations to yellow fever sufferers during the late epidemic.

It may be said that a contagionist, how sincere so ever he may be, is not bound to care for his neighbor's interests and health; but honesty requires him to care for both. It is doubtful whether the English Minister was strictly moral when he declared that he "cared for England and English interests *alone*." The same dubeity hangs over Commodore Bainbridge's toast—"My country if right, but my country right or *wrong*."

If yellow fever be contagious and transportable, quarantine ought to be enforced by grape and canister, gibbets, prisons and fines, though commerce should perish altogether. The late Dr. Townsend, who was a consistent, honest and able quarantinist, says, in his book on the yellow fever of New York, as it appeared in 1822 that all intercourse with the West Indies [and why not with New Orleans?] should be prohibited for five months in every year, beginning with June, in order to prevent the importation of yellow fever. [229.] He says, that "unless an unbroken line of lazarettoes be established along the whole coast, to guard against the pestilence we can never hope to be entirely secure. What will avail the most efficient system of quarantine laws, established here and there in a few cities on the coast, if all the intermediate towns, with which a constant intercourse is going on, freely admit vessels, etc." [231.]

If quarantine is to reign in New Orleans, let it be as rigid as in the Levant. For no Eastern mummery can be more absurd than that practiced at the quarantine stations of the United States at the present time. The strictness of the East has both consistency and reason in its favor, (admitting the doctrine of contagion,) which cannot be urged in favor of the West. A doctor of some Atlantic city of the Union goes on board of a ship from New Orleans—the plague stricken city—he *looks* at the cotton bales, and the passengers, and he straight way ignores his own theory, his oath and the law; for in a few minutes or hours after, the vessel is admitted: no one being able to know how he could possibly have ascertained by a look, whether contagion was or was not in the vessel. If yellow fever quarantine be well founded, such conduct is murder by the thousand.

If the laws of the land and of nature have established the fact of the importability of yellow fever by means of persons and merchandise, and if quarantine be necessary to prevent this importation, then quarantine never can be dispensed with by a look or a whim; that is, the laws of nature cannot be changed in this way.

The future.

While Æsculapians have no special gift of foretelling which will, and which will not be an epidemic year, history furnishes presumptions, analogies, and deductions, more or less favorable to the future in New Orleans, even though the next few years should be as insalubrious as the past. Epidemics have not only a limited period of increment and decrement in any one year, but they usually have more prolonged periods of increment and decrement, through series of years, often constituting what may be called a cycle of variable duration, after which they generally cease. So it was with the plague in Europe; so it was with the yellow fever in the Spanish peninsula; so it was with the cities of the United States in the north, as Boston, New York, Philadelphia, Baltimore, and other places. Its invasion of the Southern

tropic at Rio, so recent and severe, together with its gradual decline in the North temperate zone, may be the precursors of its further Northern declination, and Southern advance, so that both Charleston, Mobile, New Orleans, and other Southern towns and districts have now, at least, the same probabilities in favor of approaching exemption that many other cities further North had, more than half a century ago, before yellow fever appeared on the banks of the Mississippi. New Orleans is now, and has long been, near the Northern border of the yellow fever zone. If yellow fever has, as may be the case, reached its culminating point in this city, its history elsewhere in the temperate zone indicates a progressive decline.

Charleston, desolated at the close of the seventeenth century, was nearly exempt from yellow fever in the first quarter, and in the two last quarters of the eighteenth century. New York was exempt for forty years, ending in the last decennial period of the same century—a period longer than the exemption of which the present forms a part—the prolongation of which may be suddenly arrested for anything that human foresight or science can show to the contrary. The history of the past affords no guarantees that its scenes shall never be repeated. It is as idle to deny as to predict this lamentable contingency. It is consolitary to reflect that the plague as well as yellow fever has almost entirely left Europe, and that the latter disease is scarcely known in the Atlantic States of the republic. No thanks to quarantine! If any visible causes can be assigned for this exemption, the most probable are the extensions of knowledge in hygiene, physiology and physical or sanitary improvements. Thanks to science!

CHAPTER XV.

THE ENS EPIDEMICUM—THE KNOWING THAT ONE DOES NOT KNOW—THE OVERTHROW OF EPIDEMICS.

Felix qui potuit rerum cognoscere causas.

The public desires and receives with alacrity facts and arguments explanatory of the causes of yellow fever, and, hence almost every writer on this malady, whether born to solve this problem or not, thinks it his bounden duty to satisfy the public, and to glorify science, by conceiving clearly and revealing fully what no one thoroughly acquainted with, both the amount of our positive knowledge and deplorable ignorance of these essential antecedents or causes, can pronounce upon with certainty. A humiliating but true confession this is. Heat, rain, moisture, swamps, vegeto-animal decomposition, contagion and numerous other alleged causes are unsatisfactory and inadequate, as might be shown by travelling over hundreds of inconclusive and contradictory volumes, filled with special pleadings, diluted logic, theoretical biases, and irrelevant facts. The mere authority of great names in science satisfies many—names which serve to guide the multitude, as the bell wether guides his willing, faithful sheep, all of which will jump just as high as he jumps even after he has knocked the fence flat on the ground.

It is most certainly the duty of every writer on yellow fever to explain the cause of it if he can, but it is equally his duty not to sin against the decalogue of logic, any more than against the decalogue of Moses. Fortunately the *conditions*, if not the *causes* of yellow fever are to a considerable extent known: for example,

it is known to be connected, no matter how, with the warm season of the year, with unacclimated constitutions, with aggregations of people in towns and villages, etc. It rarely attacks rural populations unless they crowd together so as to become virtually towns.

A correct appreciation of these conditions is next in importance to the discovery of the cause of yellow fever—probably the former may prove after all to be more important; for the discovery of the cause, by no means warrants the conclusion that it is necessarily a removeable or a remediable one. The seeds of plants taken from Egyptian mummies contain the vital principle after the lapse of thousands of years and will grow when the proper conditions shall be present, as heat, moisture and earth, while the vital cause is in the plant. It is therefore a fundamental error to require a writer to explain the *ens epidemicum*, or to receive the alleged doctrine of contagion as the only alternative, when he cannot show what the cause *is*. Do not, say the sciolists, overthrow, unless you can build up.

It is better to acknowledge ignorance than to advocate an error. It is better to keep a question of this sort open than dogmatically to close it against investigation. In the former case the truth may be discovered; in the latter, never. To know ignorance is preferable to ignorance of ignorance. To know, that as yet, we do not know is the first step to be taken. Despair is not philosophical. The possible, who can limit it? If the cause of yellow fever has not been discovered, it may yet be. If discovered, whether within or without the body, it may, or it may not be controllable. If it should never be discovered any more than the cause that produces on the same soil a poisonous and nutritive plant, it is probable, that at least its essential conditions and laws may be ascertained so as to afford advantages and protection equal to those derivable from the discovery of its true cause or combined causes. All the lessons of philosophy teach that yellow fever *has* a cause or combination of causes, without which it cannot appear—with which, it cannot fail to appear, being not the less certain, because unknown in the present state of science. Its antecedents and sequences must prove, when known, as invariably connected and simple as any part of physics.

Ens epidemicum, smoke and cannonading Fires.

The practical method of attacking the *ens epidemicum* or epidemic entity is no more satisfactory than the theoretical,—as the enfilading the streets with artillery, the combustion of tar, or the smoking of the enemy out of a place. Dr. Rush and others enumerate such examples. The plague left London, they affirm, as soon as coal was introduced into the city as fuel. Now the part of New Orleans most affected by yellow in 1853, was the very part most afflicted by coal smoke, namely, near the St. Mary's Market, where the foundries of the city are concentrated, as Leed's, McFee's, McCan's, Armstrong's, etc. The burning of gunpowder, and artillery firing in the streets and public squares have sometimes been followed by the retreat of the *ens epidemicum*—just as the eating of a salt herring was followed by the recovery of a Frenchman and the death of an Englishman, from fever. The patriotic Board of Health, of which the philanthropic Mayor was chairman, had done all that was possible to stay the march and to mitigate the evils of the epidemic—scraped the streets—lustrated the gutters—provided for the sick—buried the dead, and wisely quarantined the influx of the *uninfected* immigrants,—they had done more;—for they gave their personal attendance to the sick and dying; in the midst of the crisis, the public mind swaying to and fro like the storm-stricken forest, they yielded to the prevailing opinion. At sun-set the epidemic was regularly, for a time, attacked

with great guns. But gunpowder failed. It did worse. Sleep to the sick is the turning point for life—the first glimmer along the dark horizon of the yet dubious morning—sleep was broken—the intellect vibrating between reason and delirium, shattered by the clangor of arms and fever, raved with redoubled violence, and was sometimes quenched at once by a horrid convulsion amid the roar of cannon.

Among the numerous plans advocated by many, was that of tar-burning. This, too, was tried. But the epidemic raged the more. Terror was supreme.

Large fires were kindled in London to destroy the Plague in 1665: "Dr. Hodges says. Heaven wept for the mistake of kindling them and mercifully put them out with showers of rain." In the town of Wheeling, enormous coal fires were kindled in the cholera of 1833. They had no other effect than that of causing one to think (during the nightly round of visits to the sick) how uncertain human life is. Their unsteady flickering lights are significant of the deadly march of cholera. Fires.

"The air extricated from fermenting wines, during a plentiful vintage, Van Swieten tells us, once checked the ravages of the plague in Germany." Carbonic acid

"Ambrose Parey says the plague was checked in a city in Italy, by killing all the cats and dogs in the place and leaving them to putrefy in the streets." Putrefaction.

A fourth part of the population of Europe was falling before the Black Plague. The Jews had been charged with causing the epidemic with a view of destroying all Christians from the face of the earth. They were massacred without mercy, though sometimes tried by commissioners: a writer of that time says, "certain commissioners have been appointed to judge the Jews; I believe none will escape." Israelites.

In France the Medical Faculty of Paris assembled in order to find out the causes and devise sanitary measures to arrest the progress of the epidemic. The doctors, after due deliberation, in a most solemn official manifesto, or medical bull, decided in the most positive manner that the epidemic was "owing to the constellations which combatted the rays of the sun, and the warmth of the heavenly fire which struggled violently with the waters of the sea, originating a vapor in the great Eastern sea of India, corrupted with fish, enveloping itself in fog. Should the same thing continue *not a man will* be left alive, except the grace of Christ preserves him. We are of opinion that the constellations, with the aid of nature, strive, by virtue of their divine right, to protect and heal the human race, and to this end, in union with the rays of the sun, acting through the power of fire, endeavor to break through the mist." The faculty at the same time predicted in the most oracular manner the future movements of the aforesaid constellations: "Accordingly, within the next *ten* days, until the 17th of the ensuing month of July, this mist will be converted into a stinking deleterious rain, whereby the air will be much purified. Now as soon as this rain announces itself by thunder or hail, every one of you should protect yourself from the air; and as well before as after the rain, *kindle a large fire* of vine wood, green laurel, wormwood, chamomile, etc. Until the earth is again completely dry, and *three* days afterwards, no one ought to go about; only *small* river fish should be used; rain water must be avoided in cooking; bathing is most hurtful, and the least departure from *chastity* fatal." Parisian Faculty.

CHAPTER XVI.

MORALIZATIONS UPON THE EPIDEMIC OF 1853.

It is doubtlessly wisest not to wed a mournful philosophy. The illusions, as the realities of life are mingled with good as well as evil. Horace, La Fontaine, and Byron have spoken despondingly of the mission and the hopes of humanity:

"Vita summa brevis spem nos vetat incohare longam.
Quittez le long espoir et les vastes pensées."
"Know that whatever thou hast been 't were better not to be.
There's not a joy this world can give like that it takes away."

Such inconsolable knowledge not being derived from the dreams of the novelist, nor from the fictitious woes personated by the tragedian, is not only unrelieved, but augmented by imagination, by the anticipations of impending danger during the rapid march of a desolating epidemic, which prostrates hundreds of friends and neighbors in a day, and, like the flash from the tempest-bearing cloud in a starless night, discloses to survivors the perilous rocks upon which the bark of life may be broken in a moment by the fast gathering storm of death.

The bloodiest battle-fields of modern times scarcely can compare with the New Orleans epidemic of 1853, which destroyed five times more than the British Army lost on the field of Waterloo. There were among the people those fluctuations of hope and fear which armies feel amid the shocks of battle, founded on chance and destiny:

"There's a Divinity that shapes our ends,
Rough hew them how we may."

"The ball on which my name is not written, cannot hit me, says the soldier in the field of battle—and how, without such belief, could he maintain such courage and gayity in the most imminent peril."

The moral consequences attributed to epidemics a few centuries ago, are so discreditable to humanity as to appear almost incredible, and certainly do not appear in the present age. The accounts transmitted to us concerning the black plague, which appears to have resembled yellow fever in many respects, show that demoralization raged equally with the epidemic;—all the ties of friendship, of blood, of morality and of religion were dissolved, or merged into brutality, sensuality and licentiousness. [Ozanam. Hist. Méd. iv. 87.] The world must have grown better. Nowhere, least of all in New Orleans, is such a sad picture of humanity seen as having any connection with epidemic visitations;—instead of demoralization, benevolence illustrates the dreadful march of death, and sheds its sunshine upon the closing scenes of life—the supreme hour of dissolution.

The deplorable scenes of demoralization which medical historians have portrayed, as occurring in former times, had their origin, for the most part, in the fear of contagion, which led to the abondonment of the sick, and reckless conduct, under the belief that contagion and death would soon arrive.

The most hideous fictions were propagated in distant cities concerning the conduct of the citizens of New Orleans, during the epidemic which has just completed its orbit. A Journal, published in an Eastern city, where yellow fever once

prevailed, but now happily exempt, holds the following, as well as still more objectionable language, concerning New Orleans, in 1853: "Doomed city of the dismal swamp, abode of death! Immense charnel house. * * * Those who are safe, who have been enveloped in the plague-sheet, and have been set loose armored against a future attack—eat, drink, and are merry, almost persuaded of immunity from all disease, since preserved against that which is the most dreaded of all. * * * * * Cheeks not blanched by the proximity of ! "*la mort inexorable.* * * * Hear other hammers than those battering down coffin-lids. * * * Carriages pass and repass not belonging to the funeral *cortege*. The drivers of hearses are not more lugubrious than draymen and porters. 'I'm safe—I've had it,' etc. The "*safe*" portion of the population, instead of having been indifferent to the sick, or devoting themselves to balls, operas, and theatrical amusements, as represented by those misinformed journalists, devoted themselves to the well-being of the afflicted and poor.

The wind which blows out a small taper, kindles up a large fire among more substantial materials. In New Orleans the fire of charity burned but the more brightly as the storm of pestilence augmented. In these days of mourning, disaster and death, the only pleasure in which the citizens actively engaged, was the melancholy one of attending on the sick, of soothing the dying, and of closing the eyes of the dead. Many, very many availed themselves of the opportunity to perform these offices of charity, quietly, steadily, devotedly!— Charity.

"Like as a star,
That maketh not haste,
That taketh not rest,
Was each one fulfilling
His God-given Hest."—GOETHE.

If New Orleans has not the credit abroad, for having deported herself nobly, and conformably to the highest requirements of sacred humanity, amid the exigencies of a terrible calamity, it is owing to misrepresentation, and not to a lack of merit. She may more easily excuse the undeserved reproaches of distant strangers misled by false rumors, than some ungrateful recipients of her kindnesses at home. But silence is not less meritorious than positive beneficence. Bereavement.

The shades of death have just gathered over at least 8,000 yellow fever victims in New Orleans! *Eight Thousand!* A brief enumeration, yet it contains volumes of wretchedness—long annals of bereavement, of widowhood, of orphanage—full of unutterable griefs, solitude, destitution—sad Souvenirs of the Past! Cheerless preludes to the *sombre* Future.

There is something ineffably melancholy in reviewing the fate of a large class of strangers, whose names and fatherland none knew. The poet has sought by the presence of friends to soothe the dying—

"On some fond breast the parting soul relies,
Some pious drops the closing eye requires."

But many died unwept and unknown. Their coffins piled up two or three tiers deep like wood, were carted to the grave in the cypress plain—

Receive them, unrelenting Grave!—

"Strong are the barriers round thy dark domain—
And fetters sure and fast,
Hold all that enter thy unbreathing reign.
"In thy abysses hide
Beauty and excellence unknown; to thee
Earth's wonders and her pride
Are gathered, as waters to the sea."

Contrasts.

The earth, air, and sky seemed to be in the midst of the pestilence; such as Goethe described, which appear in the strongest contrast, when humanity is desolated:

"Know'st thou the land where the pale citron grows,
And the gold orange through dark foliage glows?
A soft wind flutters from the deep blue sky,
The myrtle blooms, and towers the laurel high.
Know'st thou it well?
"O there with thee!
O that I might, my own beloved one, flee!"

Yet, in the midst such scenes the Angel of Death poured out the phials of his wrath. Coffin rumbled after coffin; the funeral columns defiled almost constantly for months from every street

"To join
The innumerable caravan that moves
To the pale realms of shade."

Sun-set.

As the day declined the funeral march became dense, continuous, and often blended. It was then that nature was serenest, while the sun was sinking into the cypress forests, his slanting rays dying with variegated hues, the trembling waves of the river, recalling to mind the sublime descriptions of Scott and Goethe; the first relating to a tropical sun-set, and the second one in the temperate zone:

"No pale gradations quench his ray,
No twilight dews his wrath allay;
With disk like battle target red,
He rushes to his burning bed,
Dyes the wide wave with bloody light,
Then sinks at once—and all is night."

"See how the green-girt cottages shimmer in the setting sun! He bends and sinks. Yonder he hurries off and quickens other life. Oh! that I have no wing to lift me from the ground, to struggle after, forever after him! I should see, in everlasting evening beams, the stilly world at my feet,—every height on fire,—every vale in repose,—the silver brook flowing into golden streams. * * * I hurry on to drink his everlasting light,—the day before me and the night behind—the heavens above, and under me the waves."

Poetry of the pestilence.

These contrasts between the beauty and repose of nature, and the march of death, gave rise to several poetical contributions, which were cut short in some cases by "the pest-king," whose power they were recording—the muse, trailing her fast-failing wings in the polluting streams of blood and black vomit:

———"All hoping is past!
The black draught of Death is ejected at last!"

So reads one of the unfinished black vomit poems of 200 lines, by a physician who died of the *vomito*, which he sung.

Another says:

"The sun sinks down o'er each death-crowded street,
Whilst dread, delirious screams the hearing greet;
Night settles o'er with awe and fear and gloom.
What means yon glaring blaze, yon cannon's boom?
Ha! victory's tokens for the conqueror Death!
Who slays his thousands by the *fever's* breath!"

Night.

Night was ushered in, for a short period with cannonading, and, for a considerable time with conflagrations from burning tar, the towering flames of which, cast a sickly, flickering light among the streets, upon the river, and into many a delapidated window upon yellow, rigid corpses, awaiting interment on the morrow.

CHAPTER XVII.

TABLEAU ANTE-MORTEM AND POST-MORTEM.

Although I have not witnessed the romantic novel reading, love making, and merry doings of yellow fever patients at the very close of life, as described by several writers in Mobile and New Orleans, yet I have seen many examples in which the more sober pursuits of business, and plans for the future, were discussed by sane persons but a few hours, or even only minutes, before death. Dr. Cartwright, in his account of the epidemic yellow fever at Natchez, in 1823, says that "in the last stage, in which *fever* in its etymological sense of the term disappeared and all severe pain, the patient, before debilitated, often regained his strength so far as to be able to walk about the room. When there was no evident cause for these two symptoms they invariably portended a fatal termination. A shoemaker, the the day before death, got out of bed, went to work, and nearly finished making a shoe." (Med. Rec.) He says that in the hospital four or five patients, in the last stage of the disease, acquired great strength, left their beds, got brooms and the like, and after parading through the rooms, died suddenly.

M. Robin, in his travels in Louisiana, from 1802 to 1806, mentions the case of a physician attacked with yellow fever, who unconscious of any sickness, continued to attend his patients until just before death. When interrogated, he declared he was in good health (*fort bien—fort bien*); while others died in rapture (*dans le transport*).

There is probably no violent, acute disease less painful than yellow fever, although there is none scarcely more repulsive to the beholder, as seen in the black-vomit, in enormous hæmorrhages from the mouth, nose, ears, eyes, and even the toes; the eyes prominent, glistening, injected, yellow, and staring; the face discolored with yellow and dusky red.

Without trenching too much on professional ground, let us approach the yellow fever corpse and throw off the winding sheet so as to expose the face, chest and arms. The poets, true to nature, have often found in the physiogomy or anatomical expression of the dead, much beauty—

"Lips bland and beautiful"—
"Eyes
So fair, so calm, so softly sealed
The first, last look of death revealed."

"It has been observed," says Professor Dickson, "that the countenances of those killed by gunshot wounds are usually placid, while those who perish by the sword, bayonet, pike, or lance, offer visages distorted by pain, or by emotions of anger or impatience."

Eckerman closes his "Conversations with Goethe," the great poet of Germany with the following tableau of his dead body:

"The morning after Goethe's death, a deep longing seized me to look yet once again upon his earthy garment. His faithful servant, Frederic, opened for me the chamber in which he was laid out: Stretched upon his back, he reposed as if in sleep; profound peace and serenity reigned in the features of his noble, dignified

countenance; the mighty brow seemed yet the dwelling-place of thought. I wished for a lock of his hair, but reverence prevented me from cutting it off. The body lay naked, only wrapped in a white sheet; large pieces of ice had been placed around, to keep it fresh as long as possible. Frederic drew aside the sheet, and I was astonished at the divine magnificence of the form. The breast was so powerful, broad, and arched; the limbs full, and softly muscular; the feet elegant, and of the most perfect shape; nowhere on the whole body a trace either of fat or of leanness and decay; a perfect man lay in great beauty before me; and the rapture which the sight caused made me forget for a moment, that the immortal spirit had left such an abode. I laid my hand on his heart—there was a deep silence—and I turned away to give free vent to my tears."

The physiognomy of the yellow fever corpse is usually sad, sullen and perturbed; the countenance dark, mottled, yellow, livid, stained with blood and black vomit, and swollen; the eyes prominent and blood shotten, and yellow. The veins of the face and of the whole body often become distended, and various and very curious phenomena may be discovered upon a closer inspection,—a few of which may be enumerated on this occasion;—among these is the circulation of the blood, which, independent of the heart, seems to be in some few cases, as active as in life. The following example selected from many will illustrate this new fact:

The experiments began twenty-five minutes after death, and continued one hour and thirty minutes, the history of which fills ten pages; from these the following extracts are taken: A thermometer remained in the armpit 55 minutes; the first five minutes gave 105°; five minutes 106½°; five minutes 108°; ten minutes 108°; ten minutes 108°; ten minutes 108°; ten minutes 108½°; pelvic region seven minutes 111°; five minutes 111°; five minutes 110°; stomach 109¾°, chest 107°, etc. Great distension of the veins. A ligature was placed on the arm, a vein was opened, about two ounces of blood jetted out, after which a trickling took place for a considerable time, amounting, by estimation, to twelve ounces. The circulation was found to be very rapid about the head; (the patient had died suddenly with apoplectic symptoms;) the skin of the face and neck was injected, dark, livid and somewhat mottled, there was no cadaveric hyperæmia or injection of the dependent parts; the external jugular veins were distended as if ready to burst. Greater tension I had never witnessed in glottidian œdema, nor in convulsions, nor in the last throes of parturition. The left jugular was opened, as for ordinary bloodletting, *but no bandage or pressure* was used, the head being raised, so that the orifice was nearly on a level with the breast bone. The blood jetted completely, without wetting the skin, forming an arch, the diameter of which, continued to extend for five minutes; at the end of eight minutes the arch had contracted, owing, apparently to small clots on the margins of the orifice, and the skin having once become wet, the blood, without being materially diminished, ran down the neck, jetting occasionally on removing clots from the orifice. For about one hour the flow was copious, but, at the end of that time, was diminishing rapidly. I caught nearly three pounds at first, but as much of it did not jet out, but ran down the neck, I could only estimate the amount (which I did) at five pounds, or eighty ounces from the jugular alone. As the bloodletting progressed, the congestion and discoloration of the skin of the face diminished.

Now it will be seen, that the orifice in the jugular did not discharge the blood as fast as the circulation replaced it—there was a surplus, because, the venous tension or jetting augmented for five minutes, and had not ceased during eight minutes. There was, as already mentioned, no bandage or pressure. It is fair to presume, that it would be quite impossible in this way, to bleed a living man, half as much, as collapse of the vein, clots, fainting, etc., would prevent it. Hence, the circulation in the veins was probably more active and persistent, than in health! Let it be supposed that the upper or *distal* end of the jugular, contained one ounce, when opened—this being discharged, no more could replace it, only by a circulatory force. But here, the tube is filled eighty times in a few minutes.

The heat of the patient in the early stage of yellow fever is usually very great, but it falls off towards the close of the disease, in both the convalescent and dying stages; but, among the dead, in many cases, it rises higher than in life, from a quarter of an hour to six or seven hours after death, rising (sometimes to 113°) and falling in the very same and in different regions both internally and externally. The laws of this heat do not fall under those belonging to physiological, morbid, physical, or chemical caloric, as now recognized in these sciences; they constitute, therefore, a separate branch of thermotics entirely new. This heat, after death, is not peculiar to yellow fever subjects. Nor are the following phenomena: Let an arm of the corpse be straightened out at a right angle to the body and be slapped with the operator's hand or a piece of board, in the proper place, between the shoulder and elbow, whereupon the corpse will continue, in many cases, at intervals for hours, to raise its arm from the floor through the vertical, from which the hand descends to the upper surface of the trunk, generally resting on the breast, or face, being sometimes sudden like a blow or slap. From one to five-pound weights are thus raised, if tied in the palm, and carried to the breast. The palm thus loaded cannot return to its original or extended position without aid; but if replaced, will repeat its motions, as proved by many hundred experiments which would fill more space than this memoir.

The following summary of these facts, so far as published, is so compendious, that I beg leave to give them, from a recent work "on Life, Sleep, Pain and Death," by Professor Dickson, of Charleston, an author who for scholarship, and voluminous concontributions to Medicine, occupies the first rank in our literature:

"The researches of Dr. Bennet Dowler, of New Orleans, have presented us with results profoundly impressive, startling, and instructive. He has, with almost unequaled zeal, availed himself of opportunities of performing autopsy at a period following death of unprecedented promptness, that is, within a few minutes after the last struggle, and employed them with an intelligent curiosity and to admirable purpose.

"I have said that, in physiological death, the natural decay of advancing age, there is a gradual encroachment of death upon life; so here, in premature death from violent diseases, the contrasted analogy is offered of life maintaining his ground far amidst the destructive changes of death. Thus, in cholera asphyxia, the body, for an indefinite period, after all other signs of life have ceased, is agitated by horrid spasms, and violently contorted. We learn from Dr. Dowler, that it is not only in these frightful manifestations, and in the cold stiffness of the familiar *rigor mortis*, that we are to trace this tenacious muscular contraction as the last vital sign, but that in all, or almost all cases, we shall find it lingering, not in the heart, anciently considered in its right ventricle the *ultimum moriens*, nor in any other internal fibres, but in the

muscles of the limbs, the biceps most obstinately. This muscle will contract, even after the arm with the scapula has been torn from the trunk, upon receiving a sharp blow, so as to raise the forearm from the table.

"We also learn from him the curious fact, that the generation of animal heat, which physiologists have chosen to point out as a function most purely vital, does not cease upon the supervention of obvious or apparent death. There is, he tells us, a steady development for some time of what he terms "post-mortem caloricity," by which the heat is carried not only above the natural or normal standard, but to a height rarely equaled in the most sthenic or inflammatory forms of disease. He has seen it reach 113° of Fahr., higher than Hunter ever met with it, in his experiments made for the purpose of exciting it; higher than it has been noted even in scarlatina, 112°, I think, being the ultimate limit observed in that disease of pungent external heat; and far beyond the natural heat of the central parts of the healthy body, which is 97° or 98°. Nor is it near the centre, or at the trunk, that the post-mortem warmth is greatest; but, for some unknown reason, at the inner part of the thigh, about the lower margin of its upper third. I scarcely know any fact in nature more incomprehensible or inexplicable than this."

Independent of experience, the physiologist cognizes no inherent necessity in life itself, nor in any of its organized forms of manifestation, nor in any of its structural adaptations and finalities, for a catastrophe so melancholy—so repugnant to the instincts of humanity, as death. Indeed the analogies of the material universe wherein stability reigns, or varies only in constantly recurring cycles, seems to teach that man, for whom all things appear to exist, is, what his irrepressible instincts claim, immortal—exempt from death! The stars rise undiminished as on the morning of the creation, and "pursue the even tenor of their way" through infinite space. The earth, a little scarred on its face by volcanic irruptions and accidents, is undecayed by age, "spins silently onward with spheres which never sleep;—her unwithered countenance being as bright as at creations' day." Trees live thousands of years, and some fishes for centuries. The inferior animals neither foreknow, nor apprehend impending death at every step in life. This unpleasant secret is made known to man alone. A current, he can no more resist than the unfortunate boatman caught by the descending rapids of Niagara, hurries him over a precipice into a realm as tenebrious, (after all the researches of mere physical science,) as that into which the fabled Styx debouched in the days of antiquity.

Poets and philosophers have sought to bring out in the foreground pictures more cheering, so as to veil the sombre tableau of death in the distance.—Bryant's picture is one of the most pleasing:

> "So live, that, when thy summons comes to join
> The innumerable caravan, that moves
> To the pale realms of shade, where each shall take
> His chamber in the silent halls of death,
> Thou go not like the quarry slave at night
> Scourged to his dungeon; but sustained and soothed
> By an unfaltering trust, approach thy grave
> Like one who wraps the drapery of his couch
> About him, and lies down to pleasant dreams."

With unsurpassed beauty, La Fontaine calls death the evening of a fine day:

La mort est le soir d'un beau jour.

EXPERIMENTS

ON THE

COMMUNICABILITY OF CHOLERA

TO THE

LOWER ANIMALS.

BY

W. LAUDER LINDSAY, M.D.,

LATE RESIDENT PHYSICIAN TO THE SURGEON SQUARE CHOLERA HOSPITAL, EDINBURGH.

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Experiments on the Communicability of Cholera to the Lower Animals. By W. LAUDER LINDSAY, M.D., late Resident Physician to the Surgeon Square Cholera Hospital, Edinburgh.[1]

It has often appeared to me surprising, in studying the literature of cholera, that, although the etiology of this dire disease has received a vast,—I might almost say unprecedented, amount of attention from the profession in every part of the civilized world, during the last twenty years, and all the known machinery of science and art has been put into vigorous operation to ascertain its essence or stem its progress, only with the result hitherto of baffling all human ingenuity; and notwithstanding the thousands of volumes that have been published on its etiology, symptomatology, pathology, and treatment, comparatively few attempts have been made, so far as I am aware, to propagate the disease from the human subject to the lower animals,—in them deliberately and experimentally to study all its varied phases. It has been too much the custom among authors, I fear, in the investigation of a subject of such acknowledged difficulty, to prefer the construction of specious and ingenious theories upon supposed or uncorroborated facts, which, perhaps, have been too hastily examined, to throwing aside prejudice and theory of every kind, as fettersome and dangerous, and patiently, silently, and dispassionately observing the workings of nature. It can, however, only be by the careful accumulation of the unprejudiced, simultaneous, and isolated observations of individuals that safe data can be obtained whereon to found just and sound conclusions,—which may be practically acted on and followed out,—as to the nature of a disease about which so little of a specific nature is confessedly known.

Seeing no reason why the same poison which produces or propagates the disease in man should not equally, under similar circumstances, affect creatures so much akin to him in their structure and habits as the dog and cat, it occurred to me to take advantage of my opportunities as Resident Physician to the Surgeon Square Cholera Hospital, Edinburgh, by

[1] The publication of these experiments in their present shape is due to the kindness of Dr Warburton Begbie, visiting physician of the said hospital, to whom I take this opportunity of acknowledging my deep obligations.

endeavouring to communicate the disease to these animals, through the medium of the different excretions of the patients therein treated. This I proposed to myself to do chiefly in three ways, viz :—

I. By feeding them on the evacuations,—dejections, vomit, and urine,—of patients labouring under various stages of the disease.

II. By feeding them on the blood, drawn during life, or taken from the heart and great vessels after death, of man,—or on various of the solids and fluids, *e.g.*, the blood, muscles, and viscera,—of other animals,—affected with the disease.

III. By causing them to inhale the effluvia from these evacuations and blood, and from the clothes worn by, and saturated with the perspiration and breath of, patients.

By the two former modes of procedure, I was led to expect, from what I have seen of cholera, and from the microscopical and chemical examination of the fluids in question,—and I obtained—negative, *i.e. non-specific* results. By the latter I hoped for, and succeeded in producing, *specific* results, viz., what appeared to me to be cholera, which was simultaneously developed in four animals, and was fatal in two of them.

Every one must remember the great importance that was attached by many authors, during the epidemic of 1848-9, to the presence in the cholera evacuations of certain insignificant vegetable cells, which were originally figured and described by Brittan and Swayne under the name of "annular bodies," "cholera corpuscles," "cells," "fungi," &c., and by others to the much more astounding fact that these discharges were found teeming with millions of microscopic animalcules. On the existence of the former, as the supposed poison agents of the disease, was based the very ingenious fungus theory of cholera ; on the presence of the latter, the no less ingenious but more fragile animalcular theory, each having its hosts of supporters. I have seen all the forms of the so-called "annular bodies" described by Brittan and Swayne, in the evacuations of patients under my own charge, and I have also seen the same fluids abounding in minute vibrios in a high state of activity ; but having observed the former only in small quantity, and being able in many instances to trace them back to their original source, the food ; believing moreover, that the latter exist equally in all decomposing animal fluids, I could not look upon either as having any causal relation to the disease, and therefore did not anticipate any specific effects from the administration, to animals,

of fluids, which I knew by microscopical examination, to contain both in abundance. I found that these evacuations, which were given to the animals in all degrees of freshness and putridity, and in all states of concentration and dilution, produced the simple effects of any other irritant or putrid matter, viz., a non-specific diarrhœa, attended, in the case of the more acrid substances, such as concentrated urine, by the secretion from the intestines of an increased amount of mucus to lubricate and defend the canal. The offending matters were speedily thrown off from the system by the stomach or the intestines. This is what, *à priori*, I was lead to expect ; for, even supposing the presence in the cholera evacuations of a considerable amount of a volatile poison, it is easy to conceive that while this might immediately generate a specific disease, when entering the system by such an extensive surface as the pulmonary and cutaneous systems, it might be comparatively inert when applied to a surface comparatively so limited as the gastro-intestinal mucous membrane.

By other writers, again, the blood is said to be the recipient and medium of retention and reproduction of the morbific matter of cholera. With regard to this point, I can certify that the blood taken from cholera patients after death and given to animals, was apparently attended by more severe diarrhœa and exhaustion than followed the administration of any of the evacuations. I have reason, however, to look on this effect more as coincident and accidental, than as essentially depending, for its causation, on the blood in question.

During my attendance on cholera patients, I had been constantly struck with the peculiar disagreeable odour given off by their bodies, in the copious clammy perspiration, which is such a common feature of the disease,—the exhalations from one patient being frequently sufficient to taint a large ward. Moreover, I have frequently noticed nurses and attendants suddenly seized with overpowering nausea, accompanied or not by diarrhœa and vomiting, *i.e.* the premonitory symptoms of the disease,—after having accidentally inhaled the effluvia contained in the breath or perspiration of patients for a greater or less space of time ; and I have known several fatal cases of cholera occurring in persons who had merely washed the clothes of patients dying of the disease, even at considerable intervals subsequent to their death. One of the washer-women of the Edinburgh Cholera Hospital, who died of cholera on the 11th October last, after three days' illness, attributed her seizure, and probably with justice, to the exhalations from the clothes of a cholera patient, which she had

occasion to wash ;—and one of the nurses in the same hospital, was more than once seized with very suspicious premonitory symptoms on leaving her duties in the wards for a day to wash patients' clothes in the washing-house. Experience has led cholera nurses to believe that by far the most dangerous part of cholera patients is their breath and sweat and their clothes, which are more or less saturated therewith. I have read of numerous instances, where the clothes of cholera patients, having been dried, packed up and sent to different parts of the country, were unpacked after the lapse of various considerable periods, with the result of instantly generating or rather propagating the disease in individuals previously healthy, and in places where no cholera had until then occurred. From the observation of facts such as these, I was led to believe it extremely probable that by the pulmonary and cutaneous systems the cholera poison is received into and subsequently excreted from the human body ; and a natural deduction from this belief was, that by exposing animals to these supposed poisonous exhalations, *i.e.*, by acting on the skin and lungs, and thereby at once attacking the blood,—and at the same time putting them as much as possible in the same circumstances as the human subject previous to a cholera seizure,—combining ochlesis, damp, filth, poor and innutritious food, deficient ventilation, &c., I was in a manner justified in anticipating some amount of success. By copious dilution with pure air, this noxious effluvium, like all other poisons when diluted, may be comparatively harmless, even when inhaled ; but, if concentrated in a small space, and an animal of any kind be for a length of time compelled to inspire it, surely it is reasonable to expect that a new focus of disease will speedily be produced. If fomites, I thought, were capable of inducing or exciting the disease in a *man* previously predisposed, why should I not be able, by imitating the circumstances, which are well known to predispose to cholera in him, by bringing together the localizing causes of the disease, to excite it in the dog ? It is not sufficient to say, that few or no instances exist of the transmission of disease from man to the lower animals, though examples of the propagation of various diseases peculiar to these animals from them to man are far from being uncommon. One great cause of this may be that the subject has never been properly investigated. I cannot help expressing an opinion, that cholera, as an epizootic or as a sporadic disease among certain genera and species of the lower animals—as the result of the same natural influences, whatever these may be, which produce the disease in man,—is more common than is usually held to be the

case ; but in them the disease may run a much more rapid and insidious course, and not being thus detectable by symptoms during life, can only be found out by an examination of the *post-mortem* appearances. Cases of rapid death, with symptoms of intestinal disease, are so common among many of the lower animals, that *post-mortem* examinations are seldom had recourse to,—a circumstance which is much to be regretted. Every one who has seen cholera must know that even in man rapid cases,—fatal without any of the ordinary symptoms of purging, vomiting, cramps, or lividity,—are by no means rare, and that, were it not for the simultaneous occurrence of other cases of the usual type, and during the presence of an epidemic, such instances would most probably be attributed to some other cause than cholera. It will be found that in the case of the animals which I compelled to inhale the effluvia from the clothes of a cholera patient I had combined the following circumstances :—

The previous habitual use of poor, innutritious, and putrid food, in the form of the various cholera evacuations.

Confinement, to a greater or less extent, in a damp apartment, in an atmosphere charged with the non-specific but still noxious effluvia from their own evacuations,—the probably specific exhalations of the cholera discharges,—besides latterly, the specific volatile poison from the clothes of a human cholera patient.

Ochlesis, or overcrowding.

Heat to a certain extent,—gas being burned in the room, in which the animals were confined, during the night, for the purpose of lighting a dark avenue, which led from the street to the hospital entrance-door.

Filth to a slight degree,—the floor of the apartment being sometimes covered for from one to three days with their own excretions.

Deficient ventilation,—and atmospheric pollution from the various causes above mentioned,—but only to a limited extent, the room being periodically cleaned and ventilated.

I selected the dog as the subject of experiment, from its most nearly approaching man in the structure of his digestive apparatus, and in the nature of his habits and food. Many of the lower animals, which might readily be obtained for experimental purposes, are herbivorous, and could not be made voluntarily to swallow blood and putrid animal fluids : many moreover, have a stomach so organized, that one of the pro-

minent features of cholera, viz., vomiting, could not be exhibited by them.

In consequence of my being otherwise occupied, and my general instructions that the clothes of patients admitted into hospital should immediately be fumigated with various disinfectants, steeped in hot water, and laid aside in the open air, previously to being washed, having been rigidly carried into effect by the hospital nurses, I had not an opportunity of testing the effects of fomites until a comparatively late period of my experiments (21st November);—then only in one case, on account of the stoppage of the disease in Edinburgh, and that case an unfavourable one. The result, however, was, that of four animals seized on the same day, two died with symptoms and pathological appearances exactly resembling those I have myself seen in the fatal cases of cholera which have occurred in the Edinburgh Cholera Hospital during the winter. But, in order that I might not deceive myself as to the production of a result which I was very anxious to obtain, I made the *post-mortem* examination of one of the dogs, in presence of Dr Warburton Begbie, the visiting physician of the Cholera Hospital, and the pathological lesions of both animals were shewn to several other gentlemen of great experience in the pathology of cholera, as observed both in Edinburgh and London,—all of whom, I think, agreed with me as to the similarity or identity of the lesions witnessed in these animals and in the human subject. To guard against another source of fallacy, I shewed the same appearances to a gentleman of acknowledged experience in the anatomy and pathology of the dog and other domesticated animals, and asked him whether they resembled the lesions observed in any disease of the dog with which he was familiar; his reply was in the negative, and I have failed in finding on record any disease of the dog or other animals having the same or similar *post-mortem* appearances, unless in a few cases, which have been avowedly published as cholera.

When I began these investigations I was not aware whether any and what experiments on this subject had previously been made in Britain or on the Continent; but I had not proceeded far when my attention was directed to a paper on "The Communicability of Cholera to Animals,"[1] by Mr Marshall of London, who mentions briefly the aggregate of the attempts at the artificial propagation of the disease hitherto made throughout the world. To that paper I cordially acknowledge my obligations for putting me at once in

[1] Brit. and For. Medico-Chirurgical Review, April 1853.

possession of all that has been, up to the present day, accomplished in this branch of experimental etiology. Still the mode and aims of my own attempts were in no way affected by the information therein contained; for I found that, while the author mentioned scarcely any good instances of the natural influence of cholera in animals, he detailed but very few positive and no conclusive results of direct experiment. In a word, all the experiments hitherto made appear to be either negative in their results, objectionable, or inconclusive. The mode of procedure in almost all cases has been injection into the veins, cellular tissue, or stomach; and the animals chiefly employed in many of the experiments to which he alludes were rabbits, guinea-pigs, goats, and fowls,—animals which, from their dissimilarity in structure and habits to man, we should not expect to be equally subject to the same poisonous influences. Injection into the veins or cellular tissue is, I think, a mode of experimenting exceedingly objectionable, on the ground of its danger, and the liability of its results to fallacy,—the very violence used, especially in the case of a timid animal like the rabbit, of itself, might do irreparable mischief. Its effects have uniformly been unfavourable and unsatisfactory. Injection into the stomach, again, is a most unfair and unnatural means of forcing the introduction of food. It appears to me infinitely preferable, in such experiments, to have it swallowed voluntarily. My anticipations of the effects of the cholera poison on the pulmonary and cutaneous systems of animals were thus nowise affected by the negative or unsatisfactory results referred to in Mr Marshall's paper.

In the detail of my experiments I have been minute—perhaps unwarrantably so—in mentioning the collateral circumstances; but this has arisen from a belief that, in the investigation of such a subject, where the results will be viewed in different lights by the supporters of opposed theories, it is important to state candidly *all* the conditions, however insignificant they may appear,—no less than from a wish that, in future experiments of a similar nature, the errors and omissions of my own rough and hasty essays may be rectified and supplied.

The room in which the following experiments were conducted, was an apartment some ten feet high, thirteen and a half feet long, and eight and a half feet broad, having two small windows looking to the north, and containing a gas-meter and other appurtenances. For the first two or three weeks after the hospital was opened (which was in the end of September last), and prior to the commencement of the ex-

periments, the porter slept in this room, but he was obliged to give it up in consequence of its great dampness and coldness, the floor being slightly sunk below the level of the ground, and the pipes of a cistern above having once or twice burst, and the water percolated through the roof; it had always a damp, musty smell. In this apartment several dogs were shut up for various periods, and allowed to feed, when so inclined, on different evacuations of patients. These were occasionally mixed with milk, porridge, beef, &c., to insure their being swallowed. As was naturally to be supposed, most of the animals shewed a decided aversion to their food, unless impelled by hunger, and some of them refused food for days; but I generally found *every* kind of matter ultimately swallowed voluntarily. One dog licked almost anything; the others were more cautious and capricious in their appetites. It must be obvious that, operating as I did, in an apartment of a cholera hospital, I could only, to a comparatively small extent and for a comparatively short period, produce and maintain such a combination of circumstances as is usually found to predispose to cholera in man. I did not feel myself justified in keeping the animals shut up in the same apartment, in an atmosphere greatly polluted by the exhalations from their own bodies and excreta, and from the putrid matters on which they were feeding, for a longer period than two or three days consecutively, on the ground alone of danger to the inmates of the hospital. I was therefore under the necessity of having the animals let loose and the room well cleaned and ventilated at short intervals. It happened accidentally that a barrel of chloride of lime and a carboy of solution of sulphurous acid were left in the room while the experiments were being made, and these, though they had no appreciable odour, must have in some degree acted as disinfectants.

The animals experimented on were:—

Dog No. 1.—A male; mongrel terrier; comparatively young; weighing about 10 lb., and having the following measurements:—

Length of head[1] ..... 5 inches.
... body[2] ....18 ...
Greatest girth of thorax .....15½ ...
... of abdomen .....14½ ...
... of head .....10¾ ...

When obtained (on October 24), was very lean and in poor condition; shy, skulking, timorous; ap-

[1] Measuring from the point of the muzzle to the occiput.
[2] ——— the occiput to the base of the tail.

parently quite healthy; after being a few days in hospital became active, lively, and frolicsome.

Dog No. 2.—A female; a mongrel spaniel of the King Charles type; also young; weighing about 6 lb., and having the following measurements:—

Length of head ..... 5½ inches.
... body ....15 ...
Greatest girth of thorax .....11½ ...
... head ..... 8½ ...

A very restless, timorous little animal; appeared healthy, but was very thin and in poor condition on admission (Nov. 7); never seemed to have become naturalized to the house, always meditating and attempting escape;—refused food for considerable periods.

Dog No. 3.—A female; mongrel bull-terrier; comparatively old; weighing about 17 or 18 lb., and having the following measurements:—

Length of head ..... 6½ inches.
... body .....23 ...
Greatest girth of thorax .....18 ...
... abdomen .....12½ ...

Well covered with flesh, and in good condition; quiet, shy, crouching apart from other dogs; avoided food, shewed no vivacity, and made no great attempts to escape. (Nov. 19.)

Dog No. 4.—A male; mongrel Skye-terrier; middle-aged; weighing about 14 lb., and having the following measurements:—

Length of head ..... 5 inches.
... body .....20 ...
Greatest girth of thorax .....18 ...
... abdomen .....14¾ ...
... head .....12 ...

In good condition; healthy, lively for a few days after admission (Nov. 19th); shy and timorous, taking little food.

No. 5.—A domestic cat of the ordinary kind; female; weighing nearly 4½ lb., and having the following measurements:—

Length of head ..... 4 inches.
... body .....14½ ...
Girth of thorax .....11½ ...
... abdomen ..... 12 ...
... head ..... 8½ ...

Middle-aged; healthy, but in poor condition; lively

and active, constantly attempting escape; immediately after admission (Oct. 24), fighting with dog No. 1. for its share of the viands.

Dogs 1, 2, and 3, and the cat, were stray animals, which frequented the public streets, and probably picked up the scanty means of a miserable existence among the ashes and other refuse of private houses, thrown out for the purpose of removal by the police carts. Dog No. 4 was parted with on account of its master not having the means to feed him; so that all these animals might be considered in a condition exactly analogous to that of the underfed, badly clothed, and worse-housed poor, exposed to similar vicissitudes and subject to similar privations.

Though first in order of time, want of space in the present number of the Journal compels me to postpone the experiments with the evacuations,[1] and merely to detail at present the results of the one with the fomites, *i.e.* the facts of the seizure of the four animals, and the *post-mortem* appearances seen in the two fatal cases formerly alluded to. The whole collateral conditions cannot, of course, be appreciated till the circumstances of the prior experiments have been mentioned, all the animals concerned having been fed on various of the cholera evacuations for greater or less periods. I believe, however, that the nature of their food had no further share in producing the disease in this experiment, than as a powerful predisposing cause; the continued use of fluid food, more or less putrid and sometimes irritant, may have induced a constitutional state highly favourable to the efficient action of the exciting cause, *i.e.* the exhalations from the fomites. This question, however, obviously admits of discussion. Suffice it, meanwhile, to say that, from the 26th October, dog No. 1 and the cat,—from the 7th November, dog No. 2,—and from the 19th November, dog No. 3, were more or less confined in the apartment already referred to, and fed on cholera evacuations,—up to the 21st November, when the experiment with the fomites was begun. As I have before stated, the animals were not constantly shut up; but the room was cleaned at intervals of a few days, and they were let loose to run about the hospital or in some of the empty wards thereof. Dog No. 1 chiefly frequented the kitchen or washing-house; but he was occasionally in the wards, while they contained cholera patients, though never for a longer period than a few

[1] These will appear in the next number of the Journal, and will be accompanied by drawings of the microscopical appearances of the evacuations on which the animals were fed, as well as of the discharges of, and the pathological appearances in, these animals themselves.

minutes consecutively. In them he never had any opportunity of swallowing the evacuations, nor of lying among the clothes of patients; nor did he ever lie or sleep in any part of the wards. The cat, when set at liberty, usually betook herself to the kitchen fireside; but she was also frequently in the wards while tenanted by patients, and often lay in empty beds therein. These wards, which were naturally large and roomy, were well ventilated and fumigated:—they seldom contained at one time more than a single patient, whose clothes were always removed immediately on admission. Dogs 2 and 3 were never in any of the wards while tenanted; they were occasionally, however, allowed to run about in an empty ward.

Between the 24th October and 20th November there were admitted into hospital seven cholera cases, five being in deep collapse, and two passing into the consecutive fever; besides two cases of suspicious premonitory symptoms. Of these, four were fatal—two in collapse, and two in the consecutive fever (or perhaps this should more properly be called the typhoid stage of the cholera fever); the rest made good recoveries. On the blood and evacuations of these cases the animals had been fed from the respective periods of their admission.

On the 21st November, a woman, aged thirty-four, named Macaulay, from Chalmers' Close, High Street, was admitted in a state of collapse, passing into the consecutive fever. She had been seized three days previously in the following way: Having been out washing all day, she returned to her home in the evening much fatigued, swallowed hastily a full meal of potatoes, an article of food she was not much in the habit of using, and was suddenly attacked early the following morning (6 A.M.) with diarrhœa: this was followed by the ordinary symptoms of the disease, viz., severe serous purging, with vomiting and cramps. She had been very intemperate in her habits,—scantily fed and poorly clad,—in consequence of having pawned her clothing, she had been greatly exposed to cold and wet,—appeared to have sustained severe violence, as evidenced by several bruise marks on various parts of her person,—and was four months advanced in utero-gestation. She had had fever seven years previously. On admission (in collapse) she had complete suppression of urine, of two days duration, and was greatly debilitated. The consecutive fever was marked by great injection of the conjunctivæ,—continued, almost complete, suppression of urine,—involuntary evacuations in bed,—a great tendency to coldness of the whole body, and a low typhoid depression, attended by considerable stupor and muttering delirium. She died in the acmé of the fever-delirium on the 24th October. The principal features of the

post-mortem examination were: absence of lividity of any part of the body, which was, however, very pale,—no marked dryness of the muscles nor viscera,—punctuate ecchymosis, to a slight degree, on the posterior surface of the heart,—absence of congestion or ecchymosis of the intestines, which were essentially healthy,—no "psor-enterie," nor undue prominence of Peyer's patches,—liver slightly and kidneys considerably fatty,—lower part of abdomen, up to near the level of the umbilicus, occupied by a gravid uterus. These negative characters of the pathology of cholera I have noticed in all the cases in this hospital which have been fatal during the consecutive fever.

Immediately on admission, her body-clothes, which consisted of a chemise, petticoat, gown, stockings, and neckerchief, were thrown in a heap into a corner of the room occupied by the dogs. They were comparatively new and clean, and had been given to her, I understand, by her sister immediately before she left her home to come to the hospital. The greater part of her own wearing apparel, as well as household furniture, she had pawned to supply herself with whisky. These clothes remained in the room for three days (from the 21st to 24th October), when the patient died; they were then removed and the bedding in which she had just died, soiled by her discharges, was substituted. This was left in the room for a night and then removed.

The effects of feeding the animals on the cholera evacuations up to the 24th was merely the production in most of them of a non-specific diarrhœa; their general health did not appear in any way deteriorated. But though the polluted atmosphere of the apartment, and the nature of their food, did not seem appreciably to affect the health of the dogs, it unfortunately happened that one of the hospital attendants,—a stout, healthy, middle-aged man, was, on several occasions, suddenly seized with nausea on accidentally remaining in the room for a few minutes, while feeding the animals, or otherwise,—nausea, followed by a general feeling of exhaustion sufficiently severe to render him unfit for work for the remainder of the day. On the 23d November, having been two or three times beside the dogs in the course of the morning and forenoon, he was suddenly seized with diarrhœa, while at dinner (1 P.M.), and within a few minutes he had three very fluid stools, which, however, were dark brown, fetid, and feculent. It was followed by considerable languor and nausea, and he felt much exhausted during the evening. This state continued next day; but being sent to bed, and various restoratives had recourse to, he felt comparatively well in the evening. He attributed

his various seizures to the close and noxious atmosphere of the dogs' room; with what justice admits of considerable doubt. Still I consider myself bound to mention the fact, as with some it may have its value. At the same time, I must not omit also to state that, about a week previously, he had been at a Highland ball, after which he probably adjourned to a tavern. The effects of this jubilee undoubtedly had their weight as powerful predisposing causes of his subsequent slight attacks; at all events it was noticed that he had "never been quite well since."

On the 24th, at 6 P.M., one of the nurses of the hospital, who had been in attendance on Macaulay, was seized with sudden diarrhœa, attended with vomiting and nausea. The evacuations were copious, fluid, but dark-coloured, fetid and feculent. There was no prostration, nor any tendency to collapse; on the contrary, the pulse was good and the face flushed. Several days previously she had had an attack of diarrhœa of two days' duration. She attributed her seizure to the breath and exhalations from the body of Macaulay, with whom she had sat up all the preceding night. Such seizures, attributed to similar causes, I have already alluded to as not uncommon among our nurses. She recovered completely in two days.

On the 24th November the floor of the dogs' room was covered by a considerable quantity of urine, and by masses of viscid, feculent matter of a dark brownish-green or tarry colour. There was no discharge of mucus from any of the animals, and none of the fæces were very light in colour, nor fluid in consistence. Of course, the effects of the different matters given as food varied in different animals and at different times. Sometimes the diarrhœa was more severe, the evacuations more fluid, and the animals more or less prostrate and ill; but the average result was the evacuation of urine and fæces having the following characters:—

A specimen of the urine collected from the floor was acid; of a deep amber colour; without sediment; slightly phosphatic; non-albuminous; yielded a claret colour with heat and nitric acid, but no tinge of green or purple; contained abundance of urea, the nitrate crystallizing in beautiful, delicate, colourless plates; became very ammoniacal on standing, and effervesced strongly with nitric acid, after being slightly concentrated.

A portion of the dark viscid fæces, under the microscope, was found to consist of fragments of the epidermis and cellular tissue, and of the spines, hairs, spirals, and isolated cells of various vegetables,—chiefly the elements of broth, &c.,

which had been consumed by patients, and ejected in their vomits or contained in their stools; and having been thus given to the dogs, had passed through their system almost unchanged. I could trace the large ovel cells of the potato,—the smaller cells of the carrot and turnip,—portions of the parenchyma and epidermis of the onion and cabbage,—the epidermis of barley grains,—and starch from bread, barley, &c. All these substances had been copiously supplied a few days before in the vomits of some collapse cases, when the cholera seizure had supervened on a surfeit of potatoes and herring, potatoes and beef, broth, &c. There were also a few entozoon-ova, of a dark brown colour, hairs, "annular bodies," and compound granular cells; the remainder of the field was filled up with molecular debris of a bright greenish-yellow tinge, a few phosphates, and crystals of chloride of sodium. Water agitated with this tarry feculent matter, and filtered, had a rich port-wine colour, was slightly alkaline and albuminous, and had a peculiar mawkish disagreeable smell; the addition of nitric acid slightly decolorized it, but the subsequent application of heat converted the colour into a greenish yellow.

From the 21st to the 26th November the animals were fed chiefly on the reaction and fever stools, and the collapse vomit of Macaulay, as well as on the evacuations of previous patients, without any farther results than have been already described, viz., a continuance of semifluid but dark stools, with a copious discharge of urine. Macaulay's alvine discharges were not consumed for about a week; the vomit was swallowed immediately, chiefly, it appears, by dog No. 1 and the cat.

From the 24th November to the 7th December there were no cholera cases in hospital; nor, so far as I am aware, were there any, during the greater part of that period, in Edinburgh or Leith.

On the morning of the 22d, I found dogs 2 and 3 lying quietly and apparently comfortably among Macaulay's clothes; here they had probably slept all night. In the same position, I frequently saw them subsequently. Dog No. 1 and the cat were lying in different parts of the room, about a yard from the clothes. I never noticed either of them nestling among the clothes as did dogs 2 and 3. Dog 3 seemed very quiet; only rose when called to, and then appeared quite well. Dog 2 rose at once and tried to escape; it was trembling and timorous. Dog 1 and the cat were both quite lively. Little food had been swallowed by any of the animals.

The atmosphere of the apartment was so loaded with

noxious effluvia, on the 25th November, that the animals were turned into a well-aired empty ward for the day, and their room was cleaned and ventilated. No food was meanwhile given, and they were replaced in the same room at night. They all seemed quite as healthy as usual. On the 26th, they were confined all day, and in the afternoon were given about ℥x of dark semiclotted blood taken from the heart and great vessels of Macaulay, at the *post-mortem* examination on the 25th; this was speedily consumed, the greater part of it probably by dog No. 1. They were all healthy and well, so far as I could judge by external appearances, until about 11·30 P.M., when dog No. 2 appears to have been seized with vomiting, purging, and cramps, uttering the most piteous cries and moans, frothing at the mouth, trembling from head to foot, shy, exhausted, and in extreme distress. Her cries continued at intervals for about an hour; but, attributing them to mere restlessness and wish to escape confinement, and occurring, as they did, at so late an hour (midnight), I did not visit the animal, and have consequently to regret exceedingly my not having witnessed the mode of her seizure and death.

Next morning (27th) I found her lying on the top of a barrel, over which her head hung stiffly; her limbs contracted to different degrees, and all quite rigid. On the floor was a small quantity of frothy mucous matter of a pale greenish-yellow tinge, which she had vomited, and a larger quantity of a greenish liquid, having an intermixture of greenish semifluid fæculent matter; its odour was exceedingly disagreeable, though not fœtid. Dog No. 3 was lying apart from the rest, crouched up in a corner of the room, quite apathetic and listless, making no attempt to escape, and scarcely looking up when spoken to; her eyes were heavy and suffused, and her whole appearance denoted great prostration. It was evident that she was dying, but there appeared to be little or no diarrhœal discharge round her; the evacuations were small in quantity, semifluid, fæculent-looking, and green; her tail and hips were not soiled in any way thereby. Dog No. 1 lay trembling in another part of the room, one side of his body, with the tail and hips, being much soiled with his evacuations, which were very fluid, but distinctly green, non-mucous, semifæculent, and mixed with a considerable quantity of urine; his eyes were suffused; he made no attempt to escape, though he rose tremulously when called to: he was evidently greatly debilitated and ill. The cat lay on the top of a cupboard, also quite languid and apathetic, making no attempt to escape; her fur moistened by her dejections, which were like those of the dogs, semifluid,

green, and of a fæculent character. The floor was covered with the vomit, dejections, and urine of the animals; the alvine discharges were all very fluid, but at the same time, distinctly biliary in colour, *i.e.*, of a green tint;—there was a large quantity of urine intermixed. The stench emitted by these and by the bodies of the animals was insupportable; it was not the ordinary odour of any putrid animal matter: I know of no smell with which I could compare it. The animals were therefore again brought out, and the room cleaned and ventilated; it was also fumigated by chlorine evolved from chloride of lime by sulphuric acid. The cat made its way wearily and stealthily to the washing-house; its appearance was quite altered; she had apparently had severe vomiting and purging, attended by great prostration.[1] She lay crouching here the remainder of the day, apathetic and exhausted, but without any vomiting or purging so far as I could see. Dog No. 1 also made its way to the washing-house, to the front of the boiler furnace; there it had severe retching and vomiting, with occasional purging during the afternoon; his mouth was covered with frothy mucus of a light greenish-yellow tint; he seemed much debilitated and quite altered in appearance. With the retching was combined the appearance of an effort on the part of the animal to relieve himself of a feeling of choking, but I did not notice any marked laborious respiration in this or any of the other animals affected. Dog No. 3 retired to a dark coal cellar, where she lay immoveable as if dead, until removed again in the evening to the experiment room. The collapse here appeared extreme; but during the day I observed no vomiting nor purging, and she made no cries nor movements of any kind to lead to the belief that she was cramped. Shortly after being shut up for the night, there was a good deal of whining from dog No. 1, but this probably arose from his being tied up, which in him usually excited demonstrations of this nature.

On the morning of the 28th, dog No. 3 was found lying quite stiff and dead in a corner of the room, in a pool of liquid greenish semi-fæculent matter, which had greatly soiled her right flank, tail, and hips; her limbs were contracted and rigid. Apparently she had partaken of little or none of the food supplied her; there was no appearance of any vomit, nor was the muzzle covered with frothy mucus. Dog No. 1 was again somewhat lively and greatly better; he had eaten

[1] My attention was so engrossed by the dissection of dog No. 2, that I neglected examining the symptoms exhibited by the other animals during life so carefully as I would otherwise have done; on this account I did not note the state of the circulation or heat, &c., in any of them.

a large quantity of the flesh, fat, and blood of dog No. 2, which died on the previous day. The cat was also considerably improved in appearance. There was no diarrhœal discharge from either animal. The cat passed solid dark fæces of tolerably firm consistence, in the course of the afternoon.

Two or three days subsequently, having no further materials for experiment, I let loose the surviving cat and dog, allowing them to run about the hospital and the grounds in front, and giving them abundance of nutritious food in a solid form. Both animals rapidly recovered, and at present (December 10) they appear to enjoy perfect health; the dog, in particular, which has swallowed an immense amount of cholera-blood and evacuations, is very lively and frolicsome—nay, he has even gained flesh, being considerably fatter and heavier than when first admitted.

Dogs Nos. 2 and 3 must have died between midnight and 6 A.M.,—probably about 4 A.M., which appears to be a very critical period in cholera, especially as regards the seizure. The *post-mortem* examination of dog No. 2 was performed at 10 A.M. on the 28th, about six or eight hours after death, and that of dog No. 3 at 1 P.M. on the 29th, about nine or ten hours afterwards; the viscera in the latter case were slightly warm when the body was opened in presence of Dr Begbie.

The following is a note, taken at the time, of the *post-mortem* appearances in dog No. 2.

Body exhales a strong, peculiar, indescribable odour, different, however, from that of the effluvia of the body of the human subject labouring under cholera. Considerable emaciation; limbs appear very fine, their bones and muscles prominent. Legs drawn up towards trunk. *Rigor mortis* well marked. Tail and hair around anus much soiled with the discharges, which, as collected on the floor of the room, are very fluid, of a yellowish-green colour, semi-fæculent, and have the same peculiar odour as the body. A *Tænia [solium?]* was found among the evacuations. On examining these more narrowly, the fluid portion is found to contain minute flocculi of mucus; is slightly albuminous and alkaline; yields a deeper greenish-yellow colour on the application of heat and nitric acid; collected in too small quantity to take the specific gravity; sediment contains a few entozoon-ova of a chocolate-brown colour, a few prismatic phosphates, one or two "annular bodies," and a mass of finely granular debris tinged greenish-yellow. On allowing the dejections to stand for a day, the quantity of phosphates becomes greatly increased. About the muzzle there is a quantity of frothy-mucous matter of a light greenish-yellow tinge, corresponding

in appearance with the vomit collected from the floor. It is acid; gives, with heat and nitric acid, a fugitive pinkish-red colour which gradually becomes greenish-yellow. Sediment, consists almost entirely of pavement epithelium; many of the scales are shrunk and atrophied, dark and granular; in the latter case they have a greenish-yellow tinge; if perfect and transparent they are colourless. Besides these there are a number of granular corpuscles nearly equal in size to and resembling pus; they have a light greenish-yellow tinge, and many of them, when acted on by acetic acid, exhibit round them a delicate cell-wall; they are rendered less granular, but there is no appearance of a distinct nucleus or nucle simple or divided. Mucus scraped from the mouth contains healthy pavement epithelium. Eyelids half closed; corneæ glazed. Subcutaneous cellular tissue dry, crackling; fat, when removed, quickly becomes hard and dry like suet; muscles dry, but of natural colour.

On opening *thorax*, contents dry, collapsed.

Lungs considerably overlap pericardium; slightly emphysematous at their anterior margins; flaccid; of light red colour; crepitant throughout; float in water; somewhat congested posteriorly; on making a section dark blood exudes, along with thickened mucus. *Bronchi* quite healthy; weight of lungs $2\frac{3}{4}$ oz.

Mucus taken from the trachea contains normal cylindrical epithelium.

Juice scraped from the lungs contains a quantity of normal epithelium and fatty granules, and a number of hyaline globules.

Pericardium moist, but contains no appreciable amount of serum; no ecchymosis.

Heart weighs $1\frac{1}{4}$ oz., natural in size; cavities, valves, and orifices healthy; organ contains little blood. Left ventricle firmly contracted; no clots in either side of heart; muscular substance firm, of a dull red colour; normal in structure; no ecchymosis. Aorta healthy; no congestion nor ecchymosis.

Blood from great vessels at base of heart, dark, fluid; scarcely any appearance of coagulation. On allowing it to stand in a urine glass, surface becomes bright arterial red, lower stratum continues dark and grumous; the whole mass coagulates feebly into a tremulous jelly; corpuscles normal.

Pharynx, *œsophagus*, and *trachea* healthy; no increased vascularity nor ecchymosis.

On opening *abdomen* there is a very fœtid smell; viscera dry and greatly collapsed. No marked congestion of the peritoneal coat of the intestines, which feel as if covered with mucilage,

thickened, and as if containing very viscid glutinous matter. The *omentum* is shrunk into a few bands of the most delicate areolar tissue, containing in its meshes very little fat. The *mesentery* is quite dry and transparent, resembling silk or tissue paper; its veins full of dark blood. *Stomach* contracted; feels, like the intestines, as if much thickened, and having very viscid contents. Rugæ very thick and prominent, of a dark reddish-purple; whole mucous membrane much congested, copiously lined, like the œsophagus, with very viscid stringy mucus, which, however, does not appear as distinct flocculi. Congestion greatest towards pylorus.

In the stomach and intestines are found about three or four ounces of a very turbid opaque fluid, of a dirty greenish-yellow colour, having a very disagreeable mawkish odour; sediment, which occupies about two-thirds of its bulk, is finely granular and of same colour as fluid portion; resembles very fine pease-meal and water; when filtered, is alkaline and albuminous; yields a deeper greenish-yellow tint, with heat and nitric acid. In the mucus scraped from the stomach is found a quantity of cylindrical and pavement epithelium very dark and granular.

Œsophagus healthy in appearance; considerable increase of its mucous lining; no congestion nor ecchymosis of its plicæ or their interspaces; no softening of the mucous membrane. On scraping some of the mucus from the surface of the gullet, and submitting it to microscopic examination, the epithelium is granular; contains several large "gonidic bodies."[1]

The *small intestines* are copiously lined with a thick, viscid, flocculent mucus, of a very pale greenish-yellow colour,—when this is washed out by means of water, it has precisely the appearance of the "rice-water" stools of cholera, gradually separating into a supernatant liquid, which is whitish, opaque, turbid, whey-like in colour, but somewhat thicker in consistence; and a sediment wholly composed of whitish, opaque, mucous flocculi. The mucous membrane is much thickened, and when the gut is placed under water, it assumes a beautiful velvety or villose appearance. Here

[1] I call these provisionally "gonidic," from their resemblance to the gonidia of the lichens. As generally seen, they appear quite globular, usually larger than pus corpuscles; have a distinct wall, colourless and transparent; frequently a distinct central nucleus, also colourless, round which are aggregated a number of rounded granules of a bright greenish-yellow or orange colour, resembling the chlorophylle grains in the cells of plants. These bodies have occurred in greater or less abundance in the evacuations of all the cholera patients under my charge; their precise source I have not had time to ascertain. They pass through the digestive apparatus both of man and the dog, apparently without change. Drawings of their microscopic appearances will appear with the continuation of this paper in the following number of the Journal.

and there, where the mucous coating is thinner, punctuate ecchymosis, with general congestion of the intestine to a slight degree, is observed. But if this thick viscid mucus is removed by washing, the ecchymosis is found to be general, the whole length of the intestine being copiously studded with minute dark points. It appears as if almost all the follicles of the various intestinal glands had become the seat of hemorrhagic extravasation. Peyer's patches are not exempt; they are very distinct, though not perhaps unduly prominent; their margins are thickened, but are not marked by any increase of the general congestion or ecchymosis. There is no marked enlargement of the solitary glands at the lower part of the ilium, but the mucous membrane is greatly thickened, rough, and villous, and the openings of the Lieberkuhnian follicles are very distinct. The increased vascularity is greater in some parts of the intestine than others; it is more a blush of a bright arterial hue than arborescent. The intestinal villi are quite nude.

The same flocculent mucus lines the interior of the *large intestines*, which, however, contains in addition a small quantity of a fluid, yellowish-green, slightly fœtid matter, having, to the eye and under the microscope, the usual characters of fæces; it resembles the evacuations collected on the floor of the room in which the animal died. The ecchymosis is as general and has the same appearance as in the small intestines; the increased vascularity is much better marked. Lower part of *colon* and the whole of the *rectum* are very rugose, in this respect resembling the appearance of the stomach; intensely congested, of a dark purple colour, and thickly covered with a bloody, prune-juice-like viscid mucus. The mucous membrane is much softened and very velvety, but it is much less thickened than in the small intestines. A *lumbricus* was found among the fluid washed from the intestines. On macerating the large intestines, so as to get rid of their mucous coating, the solitary glands are found to be much enlarged and very prominent, especially in the rectum; the surface of the mucous membrane appears as if studded over with pearly sago-grains.

The whole intestines, including the œsophagus and stomach, weigh $9\frac{3}{4}$ oz.

The ricy matter washed from the intestines has a peculiar mawkish smell,—similar to that exhaled by the body of the animal; specific gravity is very low, ranging about 1010,—alkaline—highly albuminous,—gives a greenish-yellow colour, with heat and nitric acid. It is more mucous and viscid, and much more albuminous, than the "rice-water" stools in

man generally are. The flocculi consist of an almost transparent, finely striated, colourless basis, covered by innumerable corpuscles, which are granular, and vary in size from about half to equal that of pus corpuscles, the greater number being somewhat smaller than pus. On the addition of acetic acid, or after boiling with that re-agent, the granularity and haziness of some of these bodies seem in no way affected, while surrounding or attached to them, a delicate cell-wall may be observed; in others, the granules become fewer, darker and larger, and no cell-wall can be detected: while in others, again, a distinct nucleus or nuclei—single or divided, may be rendered visible, and the granular matter partially or completely dissolved. I would avoid giving any decided opinion as to the nature of these bodies; the former may be epithelial or other nuclei, the latter, mucus corpuscles: but I confess my inability at once, under any circumstances, and without difficulty, by microscopic examination alone, to distinguish mucus, pus, lymph, and white blood corpuscles and epithelial nuclei from each other. These corpuscles, though similar in appearance to those found in the flocculi of the "rice-water" stools in man, are much larger, more distinct, and more numerous; this is probably owing to the much earlier period, subsequent to death, at which they were examined. The mucous bands and the corpuscles are rendered much more distinct by acetic acid or tincture of iodine.[1] No intermixture of the debris of food can be noticed.

The yellowish-green fluid taken from the large intestines, and filtered, is highly albuminous, alkaline, and gives a faint and dull brownish-red tinge with heat and nitric acid.

The water, in which the intestines were subsequently macerated for two days (28th and 29th November), likewise presented the appearance of ricy stools, but had a bloody tinge—it was alkaline, and gave a faint greenish-yellow colour with heat and nitric acid,—densely albuminous, the whole coagulable matter falling as a sediment, and carrying with it the greenish-yellow colouring matter produced by heat and nitric acid, leaving the supernatant fluid quite colourless.

Liver weighs 4 oz., of a dark purple colour, much congested with dark fluid blood; otherwise healthy.

Gall-bladder moderately distended, with deep yellowish-green bile, which is quite healthy in appearance. When the

[1] In examining the flocculi of the "rice-water" stools or similar substances, which are exceedingly delicate and almost transparent, I have been in the habit of using iodine, which by communicating to mucus, epithelial cells, &c., a deep brownish-yellow colour, renders them much more easily visible. Bile or any similar dark pigment would answer equally well.

bile is diluted considerably, heated, and a few drops of nitric acid added to it, a dense turbidity is immediately produced, along with a deep bluish-green colour, which quickly passes through purple and pink into brownish-yellow. The biliary sediment, which is scarcely appreciable, consists almost entirely of minute dark globules and granules, resembling oily matter. In the mucus scraped from the gall-bladder, there is found a large quantity of the same dark granular matter, and a few "gonidic" bodies.

Kidneys weigh 1 oz.—firm and congested with dark blood; epithelium fatty (normal state?); capsule tears off readily; surface smooth; Malpighian bodies and tubuli healthy.

Spleen weighs ¾ oz.—flaccid; dark-coloured and congested; otherwise healthy.

Pancreas healthy.

Urinary bladder weighs ¼ oz.—moderately distended, containing about ʒii–iii of urine, healthy in appearance. On opening the organ and drawing off its contents, its fundus is found to be much congested, and covered with a bloody mucus; this was not removed even after three days' steeping in water. On immersion in water, the bladder becomes thick, corrugated, and contracted, and its interior rough and rugose. Its epithelium is hazy, but healthy.

Urine contained in bladder, acid; comparatively clear; non-ammoniacal to the smell; non-albuminous; on heating it and adding nitric acid, an intense dirty green colour is produced, which passes slowly through deep purple into brown. On standing a night after this reaction, continued of a rich chocolate-brown colour, having a scanty granular sediment. The same urine, tested two days afterwards, was non-albuminous, and with heat and nitric acid gave a deep grass-green colour, passing through blue and purple into deep blood red; the latter tint was not destroyed on the addition of a large quantity of acid. On boiling down the urine, to test for urea, it rapidly becomes as dark green as the bile, and exhales a very peculiar and offensive odour, resembling that of putrid fish. On adding nitric acid, great effervescence is produced, and the mass becomes of a dark greenish-brown colour; while in thin layers, on the sides of the evaporating basin, it exhibits various shades of purple, pink, and red. It contains abundance of urea, the nitrate of which, under the microscope, occurs in foliaceous masses of a dark greenish-brown colour, which can be partially removed from it by repeated washing; it also contains a quantity of large prismatic phosphates, and, in smaller amount, dark granular urates.

Uterus and appendages, *per incuriam*, not examined.

Large abdominal veins full of dark, fluid blood; little or no blood in aorta.

Brain and its membranes—skull and scalp healthy; no marked congestion nor venous engorgement in any; ventricles moist, but contain no appreciable amount of serum; choroid plexus congested; cerebral substance firm, normal, no increase of its "puncta vasculosa;" the internal ganglia and the cerebellum normal. [Brain examined 60 hours after rest of body.]

The weight of the body after evisceration is 4 lb.

The following were the *post-mortem* appearances noted, on the 28th November, in dog No. 3.

Exhales a most nauseous odour; *rigor mortis* well marked; eye-lids half closed; corneæ hazy; muzzle firmly closed; stout muscular animal; muscles well developed, dull in colour, very dry; temporal muscles, when removed, appear as rigid as dried ham, and can readily be cut into thin slices; subcutaneous cellular tissue, and fat and cellular tissue of thorax and abdomen also very dry.

On opening *thorax*, viscera are dry and collapsed; cartilages of the ribs ossified.

Lungs weigh 6 oz.; slightly overlap pericardium; of a light reddish colour, covered with bluish-black punctuate mottling; crepitant throughout; congested to a slight degree posteriorly; on being cut into dark blood escapes from numerous points; otherwise healthy.

Bronchi normal; no congestion nor increase of mucus.

Trachea pale; quite healthy.

Pericardium merely moistened with serum; no ecchymosis.

Heart weighs 3½ oz.; normal in all its walls, cavities, orifices, and valves. Both ventricles contain dark, semi-clotted, but comparatively fluid blood, which also fills the auricles and great vessels. No coagula of any kind. Left ventricle firmly contracted; right very flaccid; muscular substance firm, dull in tint; heart appears dilated, especially at its base, probably on account of the quantity of blood contained in the ventricles, no ecchymosis posteriorly; muscular substance normal; aorta quite healthy.

Œsophagus healthy; mucous membrane pale; plicæ distinct, covered with very thick viscid but normal mucus.

On opening *abdomen*, viscera very dry and collapsed; *mesentery* resembles a diaphanous membrane like tissue-paper beautifully ornamented by arborescent lacteals and veins, the latter gorged with dark blood; the *omentum* has the appearance of a lax network of the most delicate silky fibres, covering the intestines, and containing in its areolæ

little or no fat. The fat accumulated around the kidneys and other viscera, when removed from the body, becomes whitish and hard like suet. Peritoneal surface of the intestines feels gluey, but there is no distinct exudation of viscid mucus as there often is in man. Intestines feel doughy and thickened, as if containing pultaceous matter; the surface of the small intestines is marked by a light red blush of general increased vascularity; thatof the large has somewhat of a slaty colour.

Stomach distended, much thickened throughout, but especially at pyloric extremity; interiorly, its rugæ are very prominent and thick, whole mucous membrane softened and pulpy; highly congested, having a uniform dark, bloody, purple tinge, which is deepest towards the pylorus; a small portion of the mucous membrane of the *cul de sac* at the cardiac extremity, along the greater curvature, comparatively smooth and thin. In the interspaces of the rugæ there is found a dark brownish-green frothy fluid, intermixed with a quantity of minute curdy flocculi. From the stomach was squeezed, before it was opened, about ℥ij of a greenish chocolate-coloured fluid, having very little mucus intermixed. When filtered it is found to be slightly alkaline; has little or no odour; highly albuminous; yields with heat and nitric acid a brownish-red colour, which gradually becomes greenish-yellow. Mucus scraped from stomach contains a few "gonidic" bodies. The whole mucous membrane is soft and velvety, and copiously covered by very viscid, ropy mucus.

From the *large intestines* there was squeezed about ℥i, and from the small about ℥ii of a thick, viscid, tenacious mucous matter of a deep brownish-green tinge, containing a scanty admixture of ordinary fæculent debris. This matter exactly resembles, in appearance and consistence, soft soap mixed with a little water; it is found adhering firmly, as a thin layer, to the whole extent of the canal, and is with difficulty washed off. It has an exceedingly nauseous, though not a fæculent odour, resembling that of the body of the animal; agitated with water and filtered, it yields a very turbid fluid of a brownish yellowisn-green colour, which is slightly alkaline, highly albuminous, and gives with heat and acid an orange-red or brownish-red colour, which ultimately becomes greenish-yellow. Under the microscope the viscid matter is found to consist essentially of delicate striated mucus, entangling a number of granular corpuscles, similar to those mentioned as occurring in the mucous flocculi found in the intestines of dog No. 2, along with a few fragments of vegetable tissues, "annular" and "gonidic" bodies, phosphates in irregular crystals, and a quantity of molecular debris of a bright greenish-yellow colour.

When this viscid muco-fæculent matter is gently removed, the subjacent mucous membrane is found greatly thickened, softened, and velvety; it is copiously lined with a coating of thick whitish or pale brownish-yellow mucus, which, on being scraped off and mixed with water, forms the same rice-watery matter as in dog No. I. It appears as if the mucous membrane had undergone a gelatinous degeneration, there being no line of demarcation between it and its thick covering of gelatinous matter, the one seeming to pass insensibly into the other. This mucus adheres so tenaciously that it is with difficulty removed, even after a lengthened maceration; it does not extend over a number of Peyer's patches, especially in the upper part of the gut, and these consequently appear as if deeply sunk in the mucous membrane, their margins being very thick, puffy, and much congested. There is marked congestion of a bright red hue throughout the canal, but no ecchymosis; it occurs in patches, and is very irregularly distributed.

The *duodenum*, at its commencement, feels much thickened, this thickening being continuous with that of the pyloric extremity of the stomach; the hypertrophy seems seated chiefly in the mucous membrane; it is also somewhat distended; its interior is marked by several patches of deep congestion. This congestion appears only to a slight degree externally, in the form of slightly increased arborescent vascularity of a vivid red hue.

In the lower part of the *ilium*, and throughout the great intestine, the vascularity is much increased, the mucous membrane and its mucous lining having a uniform bloody tinge; the plicæ of the interior of the large intestine appear as dark bloody ridges. The lower part of the *colon* and the whole of the *rectum*, besides being covered with an abundant bloody mucus, has a slaty colour, and the whole appearances of this portion of the intestine exactly resemble those seen in some cases of dysentery. The glands are quite obscured by the thick mucous coating. On being macerated for a few days, the blood and mucus were both gradually washed out, and the surface of the gut was then found studded over with pearl-like grains of sago, *i.e.*, the enlarged solitary glands, as in dog No. 2.

After maceration, Peyer's glands also were rendered much more distinct; their apparently eroded or excavated base and thick congested margins rendering them very prominent.

The whole intestines, including the stomach and part of the œsophagus, weigh 1 lb. 5½ oz.

The water in which the intestines were macerated exactly resembled "rice-water" stools, except that it had a bloody

tinge; it was very turbid,—alkaline, albuminous,—of specific gravity, about 1005 to 1010, and gave a greenish-yellow colour with nitric acid and heat. The flocculi of the sediment had the same microscopical appearance as those of the "rice-water" stools in man, viz., a delicate striated hyaline mucus, with a greater or less number of granular corpuscles, molecules, and granules, which were colourless, or had a very faint greenish-yellow tint; there were in addition a number of "gonidic" bodies and epithelium scales, and a quantity of dark granular pigment.

The intestinal villi are all nude.

Mucus scraped from any part of the gut is quite pure, unmixed with debris of food or colouring matter of any kind. It appears as a striated hyaline substance, so delicate and transparent that it can scarcely be seen in the field of the microscope. There are few or no granular corpuscles intermixed with it.

Liver weighs 11 oz., large, dark coloured, congested; apparently normal. Hepatic cells fatty to a slight degree. *Gall-bladder* moderately distended with bile of a dark green colour, which is normal also in consistence. When diluted, it gives the usual reaction with heat and nitric acid, the purple, pink, and red tints being very rich and deep. Biliary sediment, which is not appreciable, contains a large quantity of granular matter and oily globules. The mucus scraped from the interior of the gall-bladder contains a quantity of cylindrical epithelium, isolated or in patches, united by a membrane along the apices of the individual cells; there is also some pavement epithelium and free nuclei, all having a bright greenish-yellow tinge.

Kidneys weigh 3¼ oz., are large, firm, darkly congested; apparently healthy; surface quite smooth; capsule strips easily; epithelium very fatty.

Spleen weighs 1¼ oz., flaccid, dark coloured; structure normal.

Urinary bladder greatly thickened and contracted, plicated or rugose, both externally and interiorly. Its exterior is marked laterally by very tortuous veins filled with dark blood. Contains only about half a drachm of very turbid, but non-ammoniacal urine. Interior reticulated, pale; no congestion nor ecchymosis. Interior of urethra much congested, especially at its commencement. Bladder weighs ¾ oz.

Urine is mixed with a granulo-flocculent whitish matter in small quantity; is of a deep amber colour, acid, non-albuminous. With heat and nitric acid it gives the same reaction as the bile, *i.e.*, if one or two drops of acid be added to the heated urine, a zone of a bluish-green colour is produced at the bottom of the fluid; if three or four drops are added, an orange-red zone is developed at the bottom, with blue and green zones immediately superjacent; by agitating, while warm, the whole fluid acquires a deep grass-green colour, which rapidly passes through bluish-green, blue, purple, and orange-red, into a light chocolate tint, which is permanent. It contains a considerable amount of urea. The sediment contains a quantity of minute oil-globules, aggregate or isolated,—exactly similar to those found in the bile.

Uterus is comparatively free from congestion or ecchymosis externally; but vagina is congested internally, and towards its outlet has a somewhat slaty colour. There is also general congestion of the interior of the Fallopian tubes, each of which also contains three patches of punctuate ecchymosis, with surrounding slaty discoloration. Weight of uterus and appendages one ounce. Epithelium from the interior of the uterus, Fallopian tubes, and vagina, healthy.

Brain and its membranes, with the scalp and skull, appear quite healthy. The subcutaneous cellular tissue, and all the muscles of the head, are particularly dry. No marked venous oozing from the scalp nor bone; no marked engorgement of the sinuses of the dura mater, nor of the veins of the cerebral convolutions. On slicing the cerebral matter, the "puncta vasculosa" are normal. Ventricles are moist; small quantity of serum (about ʒss) in the anterior cornu of each; choroid plexus not congested. Internal ganglia and cerebellum healthy. Cerebral substance comparatively firm,—gray matter quite distinct.

Weight of body after evisceration, 14 lb.

The large veins of the abdomen are turgid,—their contents fluid, but not extremely so, and dark.

The *blood* corpuscles are normal. About ʒiii of blood from the heart and great vessels, on standing for a day, exhibited no distinct coagulation, but formed a delicate tremulous jelly; it was of a bright arterial hue on the surface,—dark below.[1]

Crichton Royal Institution,
Dumfries, *March* 1854.

[1] I shall be glad to be favoured by any of the readers of this Journal, or others interested in the subject, with information regarding, or cases illustrative of, the natural influence of cholera on the lower animals, or the results of any experiments, which have been made on the artificial communicability of the disease, during the present or any previous epidemic, in this or other countries.

In addition to the animals already mentioned, the following dogs were employed as the subjects of experiment, viz.:—

Dog No. 5.—A male; black retriever; a large, powerful, healthy, active animal; weighing about 56 lb., and having the following measurements:—

Length of head	9	inches.
... body	34	...
Greatest girth of head	20	...
... thorax	31	...
... abdomen	24	...

Dog No. 6.—A male; mongrel Scotch terrier; 3 months old; lively, healthy, active; weight 8¾ lb.; measurements as follow:—

Length of head	5	inches.
... body	19	...
Greatest girth of head	11	...
... thorax	13	...
... abdomen	11	...

Dog No. 7.—A female; mongrel bull terrier; had been previously subjected to several experiments, with puerperal matter injected into the vagina, without effect; weight 18 lb.; measurements as follow:—

Length of head	about 6½	inches.
... body	24	...
Girth of head	14	...
... thorax	19	...
... abdomen	15	...

Dog No. 8.—A male; young mongrel terrier; timorous, quiet; in very poor condition, (apparently starved); weight 7 lb. 11 oz.; measurements as follow:—

Length of head	6	inches.
... body	18	...
Girth of head	10½	...
... thorax	14	...
... abdomen	10½	...

The experiments were begun on 24th October, and continued, with frequent intermissions, according as patients were admitted and discharged, till 16th December. The substances on which the animals were fed during this period were chiefly the stools, vomit, and urine, passed or ejected in all stages of cholera; they were given in all states of freshness and putridity, concentration and dilution. Occasionally I used the blood and various other fluids and solids of fatal cases—removed at the *post mortem* examinations; these also were exhibited in different conditions of freshness, but generally as soon after death as convenient. When the opportunity occurred, I also fed them on the blood and solids of Dogs 2 and 3, which died of cholera, as has been already mentioned; so that all the media usually supposed to contain or convey the cholera poison were thus employed at different times, and to various extents. On one occasion only, did I inject into the cellular tissue, and then with negative results. In order duly to appreciate the effects of these substances on the canine and feline economy, it is necessary not only to give a brief sketch of their chemical and histological characters, but also to glance at the more prominent features—symptomalogical and pathological—of the cases that were thus indirectly made the media of experiment. Reserving comment, I proceed at once with these details, in a greatly abridged form:—

Case I.—I. M., a woman, aged 55; *fatal*, in the "*consecutive fever*," three days after her seizure. Had a previous attack of cholera, which was nearly fatal, during the epidemic of 1848–9. Collapse and reaction were very mild; the fever very insidious and typhoid. The principal *pathological features* were—ecchymoses on the heart, stomach, intestines, the pelvic and lumbar cellular tissue and ovaries; bronchitis; fatty liver; lesions of the aortic and mitral valves and kidneys; absence of any cyanotic condition of the skin.

Collapse vomit resembled bran and water agitated together; was of a dark-brown colour; acid; slightly albuminous; of sp. gr. 1018; had little or no odour; scum consisted of a frothy, flocculent matter. Under the microscope, the vomit-sediment contained a quantity of mucus in the form of delicately striated hyaline bands; epithelium scales; a number of the so-called "cholera corpuscles," or "annular bodies;"[1] a few phosphates; fragments of muscular fibre, and of vegetable tissues; oil globules, and other food-débris.

[1] For a description of the forms which it has occurred to myself to observe in cholera, *vide* "Clinical notes on Cholera," Association Medical Journal, 14th April 1854.

The matter vomited, while *reaction* was passing into *fever*, consisted almost entirely of mucus, and contained a large quantity of pavement epithelium; a few starch granules; cells from the parenchyma, and fragments of the epidermis of various vegetables, and oil globules. It was acid, slightly albuminous, and gave a biliary reaction.

The *collapse stools* had the same characters as those of the diarrhœa, which in this, as in many other cases, preceded the cholera, viz.:—a pea-soup colour and consistence; a bran-like sediment; sp. gr. 1008; they were alkaline and albuminous, and gave no distinctly *biliary* reaction[1] with nitric acid, though they had a biliary colour. Sediment contained a considerable number of "annular bodies;" a few prismatic phosphates; starch cells; and fragments of muscular fibre, and other forms of food-débris.

The stools subsequently passed (during *reaction* and *fever*) were of a chocolate colour; and contained, under the microscope, blood corpuscles. On standing, an oily scum soon formed, consisting of oil globules, entangling a few phosphates. They very speedily became fetid.

Case II.—M. R., a woman, aged 37; *recovery*. Admitted in deep *collapse;* passed through all the phases of the disease in a marked form, and was discharged well in sixteen days. Prominent features of the collapse were vomiting and cramps; of the "consecutive fever," (or the typhoid stage), delirium and a tendency to coma; there was suppression of urine for three days after admission. During the convalescence, a scarlatinoid eruption appeared on the face, followed by desquamation of the cuticle as after scarlatina.

Collapse vomit whey-like; somewhat resembled the "rice-water" stools; acid; albuminous; had slight odour of whisky; sediment consisted chiefly of oatmeal, and, under the microscope, showed a large quantity of starch, oil globules, mucus, and mucus-corpuscles; muscular fibre; pavement epithelium; "annular bodies;" compound granular bodies, and various forms of vegetable débris.

Collapse stools had the usual characters of the "rice-water" evacuations of cholera; alkaline; albuminous; sp. gr. 1010; devoid of fetor; gave a pink reaction with heat and nitric acid. Flocculent sediment consisted of delicately striated hyaline bands of mucus, covered by mucous and

[1] For my views on the "presence of *bile* in the cholera evacuations," *vide* "Clinical notes on Cholera," Association Medical Journal, March 10, 1854.

other granular corpuscles, which, according as water and reagents were added, resemble pus, lymph, or white blood-corpuscles or free epithelial nuclei.

Case III.—H. H., a woman, æt. 44; *fatal* in *collapse* about eight hours after admission. The most prominent symptoms were cramps, vomiting, and purging; vox cholerica; and complete suppression of urine. Chief *pathological* features were—considerable livor of the body; a sodden condition of the extremities of the fingers; dryness of the muscles and viscera; intestinal and uterine hyperæmia "Psorenterie;" empty, contracted state of bladder; exudation of a glutinous mucus on the peritoneal surface of the intestines; ℥i serum in pericardium; distension of stomach, with thinning, softening, and exuviation of its mucous membrane along the greater curvature; cysts of Fallopian tubes; hemorrhagic extravasation in right ovary; absence of ecchymosis of any of the internal viscera.

Collapse stools had usual characters of "rice-water" evacuations, and contained, in addition, a quantity of pavement epithelium (probably from leucorrhœal matter), a few "annular bodies," and some food-débris. They were of sp. gr. 1008; slightly albuminous; and gave a greenish-yellow reaction with heat and nitric acid.

Collapse vomit a brownish-yellow, frothy fluid; acid; of sp. gr. 1005; sediment resembles bran, and contains phosphates, epithelium, oil globules, "annular bodies," and food-débris.

Case IV.—A. W., a girl, æt. 7½; *recovery*. Admitted in advanced *collapse*—almost *in articulo mortis*—7½ hours after her seizure; passed through all the stages of the disease, and discharged well in twenty-one days. Collapse was very short, but severe; the typhoid stage tedious, but well marked. During convalescence, or towards the end of the typhoid stage, the "cholera-exanthem," in the form of an urticario-rubeoloid eruption, appeared all over the body; co-existent with its duration there was a considerable deposit of uric acid in the urine.

Collapse stool was "rice-watery," alkaline, albuminous, and gave a pink reaction with heat and nitric acid. Quantity ℥ii.

The next stool, passed shortly after, was of a light brownish-yellow colour; slightly fetid; alkaline; had a scanty, branny sediment. The third stool after admission resembled pea-soup in character and consistence; the microscopical differ-

ence consisted in the substitution of food-débris for the delicate colourless mucus of the collapse stools.

Collapse vomit was of a dirty grayish-brown colour; very turbid; acid; slightly albuminous; reaction with heat and nitric acid greenish-yellow. Under the microscope, the sediment contained a large quantity of pavement epithelium, and dark granular matter; no appreciable débris of food.

First urine passed after admission was sp. gr. 1015; acid; albuminous; loaded with urates; reaction with heat and nitric acid, deep orange-red; with nitric acid, cold, a pink. Sediment contained renal casts, transparent, granular, and oily; dumb-bell oxalates; epithelium; "annular bodies," as in the stools, and compound granular bodies; contained very little urea.

Case V.—A. F., a man, æt. 48. *Fatal* in typhoid stage seven days after admission. Collapse and reaction comparatively mild; typhoid stage insidious and asthenic. Prominent symptoms were—vox cholerica, suppression of urine for two days, frequent but mild hiccup, great restlessness, insomnia, semicoma, low-muttering delirium, involuntary evacuations in bed, picking of the bed-clothes, black sordes on teeth, dry brown tongue, and extreme prostration. *Pathological* features were chiefly *negative*. Absence of marked ecchymosis: hyperæmia, or dryness of the viscera; emphysema and collapse of lungs, with broncho-pneumonia, recent and of a typhoid type; fatty kidneys; firm decolorized clots found in both sides of the heart; bladder contained two or three oz. urine, abounding in epithelium and renal casts; stomach and intestines contained a dark, greenish mucous fluid, having in a most intense degree the peculiar odour exhaled by the sweat and skin of cholera patients; its sediment consisted of mucus, softened mucous membrane and its elements (cylindrical epithelium, &c.), and food-débris.

Collapse vomit was of a dark chocolate colour; acid; sp. gr. 1014; had a scanty granular sediment of similar tint, and a frothy scum of a light pinkish tinge; reaction with heat and nitric acid greenish; scum contained a large quantity of *sarcinæ*, besides epithelium, starch, oil globules, and food-débris.

Stools passed during the *typhoid stage* were of a pea-soup character; alkaline, albuminous, fetid; of sp. gr. 1010; on standing, a scum collected on the surface, consisting of oil globules entangling a few phosphates.

Urine passed in typhoid stage was acid; sp. gr. 1012; albuminous; reaction with heat and nitric acid greenish-yellow. Sediment contained renal casts, octahedral oxalates and phosphates; and the evaporated residue consisted chiefly of common salt, in large, irregular cubes, and crystalline masses; plumose and stellate phosphates, globular and amorphous urates, and an oleo-resinous, viscid matter; contained a normal amount of *urea*.

Case VI.—I. S., a girl, æt. 6½; *cholerine; recovery;* dismissed well on third day after admission; collapse mild, reaction complete; no typhoid stage.

Collapse vomit was of a light brownish-yellow colour; acid; slightly albuminous; sediment bran-like; reaction with heat and nitric acid greenish-yellow. Sediment contained a large quantity of echinus-like fatty bodies;[1] starch; epithelium perfect and shrunk; "annular bodies;" fragments of *acari* and vegetable tissues, phosphates, and oil globules.

Case VII.—I. W., a woman, æt 38; *recovery;* admitted thirty-eight hours after seizure in deep *collapse;* passed through all the phases of cholera, and dismissed well on the eighteenth day. Prominent symptoms were—protracted and severe retching and vomiting; tendency to coma; delirium; involuntary evacuations in bed; suppression of urine; appearance of the so-called "cholera exanthem," as in Case IV., during the convalescence; and painful distension of the mammæ. She was nursing when seized, and her uterine functions had for some time been disordered.

Collapse stools possessed usual characters (mentioned in Case I.) of the "rice-water" evacuations, but contained a small proportion of "annular bodies" and food-débris; reaction of some specimens with heat and nitric acid pink, of others greenish-yellow.

Fever stools (*i.e.* passed during typhoid stage) were of a pea-soup character; alkaline; albuminous; sp. gr. 1010–15; with a dark-brown sediment on the surface; an oily scum collected on standing, containing a few pellets of concrete *fat*,[2] having a deep greenish-yellow tinge.

Collapse vomit first ejected was of the colour and consistence of whey; acid; sp. gr. 1008; slightly albuminous; reaction with heat and acid greenish-yellow. Branny sediment, contained pavement epithelium, *sarcina Goodsirii*, starch, "annular bodies," compound granular bodies, muscular fibre,

[1] For drawings of crystalline and other forms of fatty matter found in the vomit of cholera, *vide* paper by the author in "Medical Times, and Gazette," Aug. 5, 1854.

[2] For examples of the occurrence of fat and oil in cholera fæces, *vide* paper on the subject by the author in the Edinburgh Monthly Medical Journal, Aug. 1854.

potato cells, and other food-débris. On standing, an oily scum rapidly collected. Evaporated residue of the filtered fluid contained abundance of phosphates and common salt.

Urine passed early in the *typhoid stage* was acid; albuminous; sp. gr. 1017; had a scanty, white, flocculent sediment, consisting chiefly of pus; but containing also renal casts; epithelium; dumb-bell, and octahedral oxalates; and compound granular bodies (the so-called "exudation corpuscles" of some authors); contained a very small amount of *urea*; reaction with heat and acid deep orange red; with acid alone pink. Evaporated residue contained a considerable quantity of phosphates, urates, and chloride of sodium.

Case VIII.—A. S., a woman, aged 40. *Fatal* in *collapse* 18½ hours after admission. Prominent features were: severe cramps, involving not only the muscles of the feet and legs, but also those of the thigh, abdomen, and thorax; cyanotic condition of body; vox cholerica; and *post mortem* movements, accompanied by a rise in the temperature of the body. (The latter phenomena were comparatively common in the other fatal cases here noted.) Chief *pathological* conditions were: rigor mortis well marked; punctuate ecchymosis of heart and intestines; thickening, softening, and exuviation of the mucous membrane of the large intestines, the separated patches closely resembling in appearance flakes of dysenteric lymph; "Psorenterie" in ilium; muscles, pericardium and viscera dry; latter much collapsed; retrograde tubercle in lungs; recent bronchitis (or, more probably, a hyperæmic condition resembling it in its pathological phenomena); left ventricle of heart firmly contracted, and containing no blood; blood corpuscles normal. Contents of *small intestines*, when filtered, were alkaline, highly albuminous, sp. gr. 1015; reaction with nitric or muriatic acid, in the cold, pink; sediment consisted chiefly of mucus and the disintegrated elements of mucous membrane, but contained also "annular bodies" and blood corpuscles. Contents of *large intestines* were similar in character, but contained a larger amount of blood, and a slight admixture of semifluid fæces, of a dark brownish colour.

Collapse vomit was of a pinkish-red, acid, albuminous; reaction with heat and acid greenish-yellow, with nitric or muriatic acid, cold, pink. Sediment, which occupied three-fourths of the whole bulk of the fluid, consisted of undigested articles of food, chiefly potatoes, beef, and the elements of broth, on which she had dined shortly before admission. The microscope detected, in the finer part of the sediment, the

parenchymatous cells of the potato, carrot, turnip, cabbage, and onion; fragments of the epidermis of these vegetables, and of barley grains; starch from bread, potatoes, &c.; isolated spirals and cells of the same vegetables; epithelium, both pavement and cylindrical; mucus and mucus-corpuscles; *sarcinæ*; fatty and oily matters. Fragments of several *acari*, and several large masses of *concrete fat*, were found in the oily scum. These fatty masses consisted of vesicles, each containing a radiating, crystalline mass of margaric acid.

Collapse stools had the ordinary "rice-water" characters, except that the sediment had a brownish tinge, and contained "annular bodies," starch, muscular fibre, and other food-débris; reaction with heat and acid pink. The second and third stools passed were fetid; resembled very thin gruel; alkaline and albuminous; reaction with heat and acid *nil*.

Case IX.—J. W., a man, aged 34. *Recovery;* admitted in mild *collapse;* passed through all the stages of the disease, and dismissed well on the 18th day. The convalescence was marked by the appearance, chiefly on the face, of the "cholera-exanthem," in the form of a scarlatinoid eruption, followed by slight cuticular desquamation.

Fever urine was acid, non-albuminous; sp. gr. 1010; contained a small amount of urea; reaction with heat and acid pinkish-red; evaporated residue contained phosphates, both prismatic, plumose and stellate, and chloride of sodium.

Case X.—M. F., a woman, aged 40. *Fatal* in consecutive fever on the second day. Admitted in deep *collapse* eleven hours after seizure. Collapse stage was short; reaction appeared sthenic and favourable; fever very typhoid, and marked by suppression of urine, great conjunctival injection, stupor and tendency to coma. Chief *pathological phenomena* were: blood contained in the heart and large vessels dark and grumous, showed no tendency to coagulation; alteration of the blood-corpuscles, the red disks being scarcely recognisable as such, but resembling pus, both before and after the addition of acetic acid;[1] bladder contracted, empty; bile healthy; *valvulæ conniventes* of duodenum and jejunum deeply stained with bile; ecchymosis on pericardium and heart, pleuræ, diaphragm, stomach, intestines, broad ligament of uterus and right ovary. Bronchitis, emphysema, and collapse; incipient Bright's disease of kidneys; liver slightly fatty; rugæ of stomach very prominent, and

[1] *Vide* paper in "Monthly Medical Journal" already referred to.

mucous membrane thickened, softened, and pulpy; exuviation of mucous membrane of large intestines in patches of various sizes, resembling in general appearance dysenteric ulcers.

Contents of *small intestines* had the characters of reaction and fever stools; of pea-soup character; fetid; sp. gr. 1012–15; alkaline, albuminous; reaction with heat and acid orange-red. Sediment contained little cylindrical epithelium, but the intestinal villi were all nude.

Contents of *large intestines* were similar in character, but darker in colour, and of a distinctly bloody tinge.

Collapse stools "rice-watery."

Fever Stools of pea-soup character, having a branny sediment; alkaline, fetid, albuminous; sp. gr. 1012; reaction with heat and acid orange-red; the scum, which speedily collected on the surface, contained several masses of concrete *fat*, as in Case VIII., and a few phosphates.

Collapse vomit "rice-watery," alkaline; slightly albuminous; sp. gr. 1008; strong odour of whisky; reaction with heat and acid greenish-yellow. Microscopical character of flocculent sediment resembled that of the "rice-water" stools, with the addition of pavement epithelium. "annular bodies," and food-débris.

Case XI.—J. M'G., a man, æt. 45; *fatal* in *collapse* eight hours after admission. Cramps of the muscles of the lower extremities and abdomen, and livor of the body, were marked symptoms. *Pathological* phenomena were: absence of ecchymosis and "psorenterie;" dryness of muscles, pericardium and viscera; general hyperæmia of viscera; contracted and empty state of bladder; abundance of apparently healthy bile in gall bladder; thickening and softening of mucous membrane of stomach and intestines; muscular substance of heart flabby, and of a dull brown colour; left ventricle firmly contracted, right ventricle flaccid and full of grumous blood; firmly intertwined among the *Columnæ carneæ* were a few decolorized clots.

Contents of *small* intestines consisted chiefly of mucous flocculi; had a chocolate or *café au lait* colour from bloody admixture; the fluid portion was slightly fetid; alkaline; densely albuminous, and sp. gr. 1010; a phosphatic scum rapidly formed on the surface on standing. The microscopical characters of the flocculi resembled those of the "rice-water" stools, with the addition of cylindrical epithelium, isolated and in groups connected at their apices by a continuous membrane. Reaction with heat and acid greenish-yellow.

Contents of *large* intestines are similar in character, but darker in colour, from containing a larger amount of blood; there were comparatively few cylindrical epithelium cells, but on the other hand, a considerable quantity of food-débris, annular and compound granular bodies; less albuminous; reaction with heat and acid brownish-red.

Collapse stools "rice watery;" sediment contained a few annular and compound granular bodies.

Collapse vomit of a pinkish colour, acid, non-albuminous; sp. gr. 1005-10; reaction with heat and acid, pink, which by prolongation of the heat was converted into greenish-yellow; frothy mucous scum contained a large quantity of *sarcinæ* and epithelium; sediment consists chiefly of undigested masses of potatoes and beef, and their disintegrated elements.

Case XII.—Caroline Macaulay, a woman, æt. 34; *fatal* in the acme of the "*consecutive fever*," in three days. Admitted in collapse, three days after first seizure; was four months advanced in utero-gestation. Prominent symptoms were: contracted pupils, great conjunctival injection, restless insomnia, tendency to coma, delirium, involuntary evacuations in bed, suppression of urine, laborious respiration, and threatened abortion. Chief *pathological* conditions were: punctuate ecchymosis of heart; absence of marked hyperæmia; stomach and intestines apparently normal, liver slightly, and kidneys very fatty; blood normal.

Contents of intestines resembled the stools passed during life, but were darker in colour, having a bloody tinge.

Reaction and *fever stools* of pea-soup colour and consistence, alkaline, fetid, slightly albuminous; sp. gr. 1015; sediment presents the ordinary microscopic characters of the "rice-water" flocculi, with the addition of a quantity of food-débris of a bright greenish-yellow (biliary) tinge. The matters evacuated as the fatal termination approached were darker in colour, of more uniform consistence (no separation of a sediment), more fetid, and contained "annular bodies" and phosphates, in addition to a larger amount of food-débris.

Collapse vomits. First was of a grass-green colour, acid, non-albuminous; sp. gr. 1005; clear; sediment, which was scanty and flocculent, contained epithelium, starch, and vegetable débris, mucus, and oil globules. Decolorized by nitric acid.

Second and third were of an ale colour, neutral, devoid of odour; sp. gr. 1010; non-albuminous; turbid; reaction with heat and acid brownish-red.

Fourth was of a dark steel-gray colour, slightly acid, devoid

of odour; sediment contained annular bodies, mucus-corpuscles, epithelium, and vegetable débris.

Case XIII.—W. L., a man, æt. 42; *fatal* in *consecutive fever* in six days after admission in collapse. Reaction apparently sthenic and favourable; fever insidious and typhoid. Chief *pathological* conditions were: dryness of the muscles and viscera; ecchymosis of the pleura, heart, and intestines; general hyperæmia of the viscera; softening and thickening of the intestinal mucous membrane; red blood corpuscles normal, but there was an increase in the proportion of white corpuscles.

Collapse stools "rice-watery;" reaction with heat and acid pink, passing into greenish-yellow on prolongation of the heat.

Reaction and fever stools chocolate-coloured, alkaline, fetid, albuminous; reaction with heat and acid orange-red.

Collapse vomits acid, of very low density, non-albuminous, of various shades of whey-colour, greenish-yellow, brownish, reddish, and gray; reaction with heat and acid greenish-yellow; addition of acid caused considerable effervescence. Sediment contained mucus and mucus-corpuscles, epithelium, annular bodies; a few phosphates and crystals of chloride of sodium; fatty and oily matters to various extents. In some specimens there was a number of granular corpuscles having single or double nuclei, and otherwise resembling pus.

Case XIV.—J. I., a boy, æt. 2. *Recovery* in ten days after admission. Attack followed protracted convalescence from rubeola. Collapse short; subsequent symptoms more rubeoloid than choleraic in character.

Case XV.—A. B., a man, æt. 49. *Fatal* in four days in the consecutive *fever*, which was very insidious and typhoid. Chief *pathological* features were: softening and exuviation of the mucous membrane, and general arborescent hyperæmia of the intestines; partial ecchymosis of the intestines; first stage of typhoid pneumonia; dilatation and hypertrophy of the heart; lesion of the aortic valves; renal and hepatic epithelium slightly fatty.

Case XVI.—J. B., a woman, æt. 53. *Fatal* in advanced *collapse* three hours and a half after admission. Chief *pathological* conditions were: absence of livor of the surface; marked dryness of the viscera, or ecchymosis in any part of the body; rosy hyperæmia of intestines; thickening and sodden condition of their mucous membrane; their contents "ricy" mucus, slightly tinged with blood; retroverted uterus; mucous polypi of *cervix uteri*; fatty liver.

Contents of *stomach* about ʒiii, of a dirty brownish-yellow colour, turbid, acid, albuminous; reaction (of filtered fluid) with heat and acid light greenish-yellow.

Contents of *large* intestines cream-coloured, having here and there a bloody tinge; alkaline, fetid, densely albuminous; reaction of filtered fluid portion with heat and acid orange-red. Sediment contained cylindrical epithelium, mucus, and mucous and other granular corpuscles.

Contents of *small* intestines similar in character; contained quantities of small shreds of softened mucous membrane.

I now proceed to the detail of the experiments with the solids and fluids of the patients just above referred to; beginning as I have already stated, on Oct. 24th.

Oct. 24.—Dog No. 1 and the cat were introduced into the experiment room. Both were at first very shy; and, until impelled by hunger, they refused food; they had been starved on the previous day in order to insure some degree of appetite.

No patients in hospital.[1] Case III. had been *fatal* in *collapse* on the 9th; and Case I. in "*consecutive fever*," on the 11th curt. Case II. was dismissed recovered on the 19th curt.

In the morning gave the animals the collapse and reaction vomit, and in the afternoon the collapse and reaction stools of Case I.; the former were chiefly consumed by the cat, the latter by the dog.

Oct. 25.—*No* new cases admitted. The floor of the room was covered by a considerable quantity of fæces; those of the dog being semisolid, and of a dark brownish-green colour; those of the cat more fluid, of a grayish tint on the surface, and brownish-yellow below. Under the microscope they presented the characters of healthy fæces. Both animals were lively and well, and anxious to escape, especially the cat.

In the morning gave the fever vomit, and in the afternoon the fever stools of Case I. Both animals exhibited a better appetite, especially the dog.

Oct. 26.—*No* new cases. Both well and lively. Fæces slightly more fluid and abundant; floor covered with a quantity of urine. Effluvia from the evacuations had a most disagreeable odour. Room cleaned, and animals turned loose for an hour, but allowed no other food.

[1] By this I mean no cases of genuine cholera. There were occasionally admitted patients labouring under simple, but excessive, or suspicious diarrhœa, as well as cases simulating cholera in some of their features, *e. g.*, colic, cramps, vomiting, and purging, induced by intoxication and exposure to cold.

In the morning gave the collapse vomit, and in the afternoon the collapse stools of Case II.

Oct. 27.—Cases IV. and IX. admitted in collapse. Both well and lively. Fæces still more fluid, partly from admixture with urine, but not otherwise altered. Dog ate with avidity, and consumed proportionally a much larger quantity than the cat. The stench in the room necessitated its being cleaned and ventilated during the day. The animals were let loose on the ground floor of the hospital.

In the morning gave the collapse stools of Case III.

Oct. 28.—Cases V. and VII. admitted in collapse; Cases IV. and IX. in reaction, passing into fever. Animals well, very anxious to escape.

In the morning gave the collapse vomit of Case III.

Oct. 29.—4 cases under treatment; 1 convalescent from cholerine, and 3 in the "consecutive fever" of cholera.

In the morning gave the collapse stools of Case IV.

Oct. 30.—Case VII. admitted. Animals *in statu quo*, and fæces still very fluid, but, along with urine, comparatively normal.

Gave collapse vomit of Case IV.

Oct. 31.—Case VIII. admitted in collapse. Case V. dismissed recovered. Animals continue well.

Gave collapse vomit of Case VI.

Nov. 1.—Case VIII. fatal in collapse. Four "consecutive fever" cases remain under treatment. Animals well; room cleaned, and animals let loose for the greater part of the day.

Gave collapse vomit of Case V.

Nov. 2.—*No* new cases admitted. Animals well.

Gave collapse stools of Case V.

Nov. 3.—*No* new cases. No perceptible change on the animals; fæces still fluid, and urine abundant.

Gave collapse vomit of Case VII.

Nov. 4.—Case V. fatal in typhoid stage. Three fever[1] cases remain under treatment. Animals well.

Gave collapse vomit of Case VIII.

Nov. 5—*No* new cases. Animals well; fæces frequently differed in colour and consistence; their usual fluidity indicated a slight diarrhœa.

Gave about a pint of blood, removed from the heart and great vessels of Case VIII. at the *post mortem* examination on 2d curt. It was almost wholly consumed by the dog.

[1] In using the words or terms "fever," "consecutive fever," "rice-water," "serous stools," &c., I do not wish to indicate a belief in any particular theory or view of the nature of cholera, or any resemblance which I can trace in the stools. I employ them merely as the phrases ordinarily made use of to signify certain stages or phenomena of the disease.

Nov. 6.—*No* new cases. Both animals very quiet, and evidently ill. Appear to have had severe diarrhœa; fæces of dog tawny in colour and consistence; urine abundant.

In morning, gave contents of *small* intestines of Case VIII., removed at the *sectio cadaveris*; they were chiefly swallowed by the dog.

In afternoon, gave contents of *large* intestines of Case VIII. This was followed by a continuance of severe purging; the fæces of the dog were very fluid, but of a brownish-green (biliary) tint; the microscopical characters did not essentially differ from those of more solid and normal evacuations. The substances detected by the microscope usually consisted of the matters contained in the evacuations on which the animals were fed, many of them passing rapidly through the system of the dog and cat apparently little altered. There was no appearance of mucous flocculi in even the most fluid of the evacuations.

About 11 P.M. ʒii. of the heart-blood of Case VIII., not defibrinated, and partially clotted—removed at the *sectio*—was *injected* by a common syringe into the cellular tissue of the thigh of each of the two animals. Immediately after the operation they scampered away apparently in good health.

Nov. 7.—*No* new cases; 3 cholera convalescents under treatment. Room cleaned and ventilated. In afternoon, dog No. 2 introduced; it was shy and timorous, refusing food, but withal lively and well.

Dog No. 1 and cat better; appeared in perfect health; the former gamboled very friskily, the latter as usual made repeated sly attempts to escape in search of more agreeable food, a warmer lodging, and a more pleasant companion. Floor covered with very liquid but dark coloured fæces, and with limpid urine; stench very strong.

Gave the urine of Cases IV., VII. and IX., concentrated nearly to dryness, mixed with milk and a little porridge.

Nov. 8.—*No* new cases. Milk and greater portion of the porridge had been consumed, but the urine-sediment had scarcely been touched. No appreciable difference in the animals. Gave no other food till the urine should be licked up. In the evening a little had been licked by the dogs, the cat having probably had the lion's share of the porridge and milk. By virtue of her claws, Grimalkin usually managed to have a selection of the viands, keeping the dogs in the background till she herself was served.

Nov. 9.—*No* new cases admitted. Diarrhœa continued in a mitigated form; animals almost *in statu quo*. Dog No. 2 had swallowed very little. Urine not consumed.

Gave the collapse stools of Case VIII., passed on 31st ult. and 1st curt.

Nov. 10.—*No* new cases. Dog No. 2 succeeded in making her escape; all animals apparently well.

Gave the filtered fluid portion of the fever stools of Case V., passed on 28th ult.

Nov. 11.—*No* new case. Animals *in statu quo*.

Gave about oj. of the fluid portion of the collapse stools of Case II., passed on 3d ult., evaporated to dryness, and mixed with some milk and porridge.

Nov. 12.—Case X. admitted. 3 convalescents still remain in hospital. Animals well.

Gave oj. of fluid portion of collapse vomit of Case II. evacuated on 3d and 4th ult., concentrated and mixed with milk.

Nov. 13.—Case XI. admitted, and *fatal* shortly after in collapse; 1 fever case and 3 convalescents remained under treatment. Animals still comparatively well; fæces still fluid.

Gave collapse stools of Case X.

Nov. 14.—Case X. *fatal* in consecutive fever; Case IX. dismissed well; 2 convalescents remain. Dog No. 1 made its escape yesterday afternoon, but was re-captured this morning.[1]

Gave collapse vomit of Case X.

Nov. 15.—*No* new cases.

Gave collapse stools of Case XI.

Nov. 16.—*No* new cases. Dog No. 2 re-captured and re-introduced; still sly and timorous. The semifluid, but still dark-coloured, fæces of the dog No. 1 were again examined by the microscope, and found to contain, in addition to vegetable débris, crystals of phosphates and chloride of sodium, grains of siliceous earth, and entozoon-ova (probably of an *ascaris*) in various stages of development.

Gave contents of the intestines of Case XI., removed at the *sectio* 2 days before.

Nov. 17.—Cases IV. and VII. discharged recovered. *No* cases remain in the hospital.

[1] When I do not specially mention the condition of the animals, or the characters of the fæces, it is to be understood that no appreciable or note-worthy alteration from the normal state was observed.

Gave about oj. of the heart-blood of Case XI., removed at the *sectio* 3 days before.

Nov. 18.—*No* new cases. Animals continued well. Fæces resembled soft soap in appearance, others resembled treacle, and were probably passed by dog No. 1, which appeared chiefly to have consumed the blood supplied yesterday. The microscope detected in the latter a considerable quantity of entozoon-ova, hairs, phosphates and chloride of sodium, vegetable débris, "annular bodies" such as occur in the cholera evacuations of man, and granular matter of a dark green colour. Most of the so-called "annular bodies," or "cholera corpuscles," appeared to suffer disintegration or great alteration by passing through the intestinal system of the dog and cat; others, however, were discharged unaltered, *e.g.* those I have for convenience sake denominated "gonidic."

Gave about ʒvi. of blood, taken at a *sectio* 2 days before from the heart and lungs of Case X.

In afternoon, gave contents of the intestines of the same case, also removed after death.

Nov. 19.—*No* new cases. Dog No. 3 introduced in good condition, quiet, shy; lay in a corner apart from the others; the cat also avoided canine society; but dogs 1 and 2 generally nestled together in some quiet corner; they all lived together, however, in comparative harmony.

Gave collapse vomit of Case XI., evacuated 6 days before.

Nov. 20.—*No* new cases. Microscopical examination of the fæces revealed no change. Animals all well.

Gave fever stools of Case X., passed 6 days before.

Nov. 21.—Case XII. admitted in collapse; no other cases in hospital. The body-clothes of this case were heaped up in a corner of the experiment room. Animals well; fæces still fluid, but otherwise normal.

In morning, gave about oj. of fluid portion of collapse vomit of Case VIII., concentrated by evaporation.

In afternoon, gave above ʒx. of the fluid portion of the collapse stools of Case XI, also concentrated by evaporation.

Nov. 22.—1 *fever* case in hospital (*i.e.*, a patient in the "consecutive fever," or typhoid *stage* of cholera). The animals were in the condition and positions already described.[1]

Gave reaction and fever stools of Case XII.

Nov. 23.—*No* new cases. Animals well.

[1] *Vide* page 289 *et seq.* of the Journal for the *special* experiments with the fomites, which ought to have been described at this stage of the *general* experiments.

Gave collapse vomit of Case XII.

Nov. 24.—Case XII.—*Fatal* in acme of consecutive fever. Body-clothes removed from experiment-room, and bed-clothes in which the patient had died, and which were saturated by her discharges, were substituted.[1]

Gave fever stools of Case XII.

Nov. 25.—*No* cases in hospital. Animals apparently in good health. Gave contents of the intestines of Case XII., removed at the *sectio* this afternoon. Room cleaned and ventilated during the day.

Nov. 26.—*No* new cases. Animals still well; confined all day. Gave about ʒx. blood, taken from the heart and great vessels of Case XII. at the *sectio* yesterday.

In afternoon, gave about oj. of the fluid portion of the fever stools of Case X., concentrated to one-tenth by evaporation.

Nov. 27.—Dog No. 2 died of cholera; remaining animals all apparently labouring under the disease in different degrees. Fæces, &c., as already described. As the matters supplied yesterday had not been consumed, I gave them no further food.

No new cases admitted.

Nov. 28.—Dog No. 3 died of cholera. Dog No. 1, and cat, still very ill. Cleaned and ventilated the room; turned loose the surviving animals, and gave them a supply of fresh milk and porridge.

Nov. 29.—Dog and cat still very much debilitated, and labouring under diarrhœa; allowed to run at large in the grounds surrounding the hospital, and fed on milk, porridge, bread, meat, &c. Dog No. 4 introduced.

Nov. 30.—*No* new cases. Animals all comparatively well and lively. Again confined them in the experiment-room, and gave them, as food, portions of the blood, fat, flesh, bones, and viscera of dogs Nos. 2 and 3, removed at the *post mortem* examinations on the 27th and 28th current; were chiefly consumed by dog No. 1. Dog No. 5 introduced.

Dec. 1.—*No* new cases. Dog No. 1 very lively; No. 4 trembling, quiet, and timorous; No. 5 lively, *whining and barking* from anxiety to escape. Cat very quiet; all well; none affected with diarrhœa. Most of the fæces were semisolid, and of a dark brownish-green colour; some were semifluid and tarry. Urine was abundant, and normal.

Experiments intermitted till 7th.

Dec. 6.—Cases XIII. and XIV. admitted in *collapse*. Animals in good health; had been running about in the hospital grounds daily, and fed on porridge, bread, &c. Dog No. 6 obtained.

[1] For characters of the fæces and urine of the animals, *vide* page 288.

Dec. 7.—*No* new cases. Dogs (not the cat) confined in experiment-room in the evening and during the night, and body-clothes of Cases XIII. and XIV. were thrown in a heap in a corner. Dogs Nos. 1 and 4 slept among these clothes, while Nos. 5 and 6 slept at door.

Dec. 8.—All well. Animals let loose during day, and fed on porridge and milk. Room ventilated and cleaned. Fæces and urine normal. Animals were shut up for the night in a damp, dark cellar of an untenanted house a few yards off.

Dec. 9.—*No* new cases. Animals well; confined for a few evenings in an under-ground room of the house above mentioned. In addition to some flesh, porridge, and milk, gave them collapse stools of Case XIII.

Dec. 10.—*No* new cases. All well. Stools had been consumed only partially, and that by dog No. 1. Dogs Nos. 7 and 8 introduced. Gave collapse vomit in the morning, and reaction and fever stools in the afternoon, of Case XIII.

Dec. 11.—Case XV. admitted in collapse; two patients in consecutive fever stage remained under treatment. Animals well; fed on porridge, meat, and general kitchen refuse, and allowed to run about in the open air all day; confined in the room during night, the body-clothes of Case XV. being thrown into a corner thereof.

Dec. 12.—Case XIII. fatal in consecutive fever; two fever cases were still under treatment. Animals well; bed-clothes of Case XIII., in which he died, and soiled by his evacuations, were introduced into the room at night.

Dec. 13.—Case XVI. admitted in collapse, and *fatal* 3½ hours after admission; 1 fever case, and 1 convalescent, remained under treatment. Animals well; body and bed clothes of Case XVI. introduced into room, along with the animals, at night.

Dec. 14.—*No* new cases. Animals well; running loose all day; No. 7 escaped; still feeding on porridge and milk, meat of various kinds, and other kitchen refuse.

Dec. 15.—Case XV. *fatal* in *consecutive fever;* no new cases in hospital. Animals well; bedding in which Case XV. died introduced at night.

Dec. 16.—Case XIV. dismissed recovered; no other cases in hospital. Animals well and lively. Floor covered with fæces; one specimen was perfectly formed, of the colour and consistence of the thicker plastic clay, and appeared as if it had been forcibly extruded through an unyielding sphincter;

a second specimen was also well formed, but of a brownish-yellow colour, soft, and easily broken down; a third was of the consistence of thick porridge, amorphous, of a deep brownish-yellow; it contained a considerable amount of potatoes and other vegetable matters, undigested or only partially so; a fourth was semifluid, amorphous, of a grayish-black colour, and mixed with sand and dust; a fifth was fluid, and of a dark grass-green colour. Their microscopic characters were characteristic of comparatively healthy fæces. Two specimens were agitated with water, and the fluid filtered; both had a greenish-yellow tint, no peculiar odour, and were alkaline and albuminous;—in one the reaction with heat and acid was brownish-red, which speedily changed into brownish-yellow, in the other it was pinkish,—thus showing the decidedly *biliary* nature of these reactions.

Dec. 17.—At this date the experiments were put a stop to by the cessation of the epidemic in Edinburgh. The surviving animals were therefore turned loose, and fed on the kitchen refuse. They continued to enjoy perfect health; some of them even appeared to have gained bulk and weight.

I cannot, in the present paper, make any remarks on the above experiments as bearing on the general question of the *contagion* of cholera; but I would simply beg, in the meanwhile, to call attention to the different effects of the fomites before and after the week between 21st and 28th November. Prior to this period, I endeavoured to concentrate in the animals all the predisposing causes of the disease as we know them in the human subject; subsequently I tried equally to disperse these, by giving the animals nutritious food, exposing them freely to the air, and giving them ample exercise.

Crichton Royal Institution, Dumfries,
April 1854.

HISTOLOGY

OF

THE CHOLERA EVACUATIONS

IN

MAN AND THE LOWER ANIMALS.

BY

W. LAUDER LINDSAY, M.D.,

PERTH.

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HISTOLOGY

OF

THE CHOLERA EVACUATIONS

The following notes are not intended to embrace the whole subject of the histology of the evacuations passed during life, or of the fluids and solids examined after death, in cases of cholera in man and the lower animals, but refer only to a limited number of observations made by myself in the City Cholera Hospital of Edinburgh, towards the close of the year 1853. These observations have been, in great measure, confirmed by the simultaneous or subsequent labours of various continental observers, who have, since the date above mentioned, published valuable monographs on some hitherto obscure points in the natural history of cholera. In a few minor details only do our respective results differ; but as my object here is simply to enumerate facts, I shall not attempt the explanation of discrepancies, or the solution of difficulties, in regard to the bearing of observations on theories of the nature or cause of cholera. The latter is a subject indeed for which I do not think we have yet obtained sufficient or requisite data whereon to speculate with exactitude or advantage to science. The symptomatology and pathology of the cases upon which the following observations were made, have been already published in various medical journals during the year 1854.[1] It will be convenient to divide the subject-matter into two sections:—

I.—The histology of the vomit, stools and urine passed during life, and of the thoracic and abdominal viscera and their contents, examined after death, in *man*; and

II.—The histology of the same discharges, viscera or fluids in the *dog* and *cat* to which the disease was communicated by artificial means.

I.—*Histology of the cholera evacuations, etc., in man.*

The chief histological elements observed may be shortly tabulated as follows:—

1. *Vomit*: A. Ejected during life.
   - *a. Mucus*: fibrillated bands without or with granular corpuscles.
   - *b. Granular corpuscles*: probably chiefly mucus corpuscles and epithelial nuclei.
   - *c. Epithelium*: tesselated, globular, and cylindrical: perfect, variously abortive, or altered in shape—transparent, hazy, or granular.
   - *d. Debris of food.* 1. *Vegetable* food.
     - *a.* Fragments of epidermis: potato, oats, barley, cabbage, carrot, turnip, onion, sugar-cane, etc.

[1] Chiefly in the *Association Medical Journal*, March 10, 1854, *et seq.*, *Edinburgh Medical and Surgical Journal*, April and October 1854, and *Edinburgh Monthly Medical Journal*, August 1854.

b. Fragments of parenchyma of several of same vegetables.
c. Isolated cells of epidermis and parenchyma, including, along with starch corpuscles, many of the "annular bodies," and "cholera fungi or corpuscles" of recent authors.
d. Epidermic hairs.
e. Spiral and porous vessels, and uncoiled spirals.
f. Chlorophylle grains—isolated and aggregated.
g. Starch corpuscles of the potato, oats, barley, wheaten bread, etc.

2. *Animal* food.
a. Muscular fibre of beef, etc.
b. Fish scales: Findon haddock, etc.

e. Oily and fatty matters:
a. Fluid: oil globules.
b. Concrete: fat vesicles.
c. Echiniform, or spiculated bodies.
d. Crystalline: filiform bundles—radiating masses.

f. Crystallizable matters, salts, etc.
a. Triple phosphates: chiefly in prismatic form.
b. Chloride of sodium: in broken cubes and irregular tabular masses, etc.

g. Fungi: *a.* Sarcina ventriculi: entire or partially disintegrated.
b. Mycelium and sporules of various kinds of mould.

h. Animalcules *a.* Vibrios.
b. Acari.

i. Pus.
k. Compound granular bodies.
l. Molecular and granular debris, variously coloured.

B. Post-mortem contents of stomach.
Epithelium: conoid, fusiform and fibroid, and other elements of disintegrated mucous membrane, in addition to several of the above substances.

II.—*Stools.* A. Passed during life.
a. Mucus: fibrillated bands always associated with granular corpuscles.
b. Granular corpuscles as in the vomit, but larger and more numerous: associated with much granular matter.
c. Epithelium: tesselated and globular, never cylindrical.
d. Debris of vegetable and animal food: as in the vomit, but to a much less extent, and in less variety.
e. Oily and fatty matters: fluid and concrete forms only.
f. Crystallizable matters, salts, etc.: in addition to phosphates and chloride of sodium, as in the vomit,
a. Urates and
b. Siliceous matters.
g. Fungi: Mycelium and sporules of various kind of mould.
h. Animalcules: Vibrios.
i. Compound granular bodies, variously coloured.
k. Blood corpuscles.
l. Hyaline globules: non-oleaginous.
m. Molecular and granular debris, variously coloured.

B. *Post-mortem* contents of intestines. In addition to several of the above histological elements,
a. Epithelium: cylindrical, isolated or in patches, and other disintegrated elements of mucous membrane.
b. Shreds or flocculi of fibrinous false membrane.

III.—*Urine.* A. Passed during life.
a. Mucus: more hyaline than in the vomit and stools, and in less quantity.
b. Granular corpuscles, as in preceding evacuations.
c. Epithelium: tesselated and globular: clavate, fusiform, or otherwise

altered in shape—hazy or granular: in its origin renal, vesical, urethral, vaginal, etc.
d. Fibrinous casts of renal tubuli: transparent, granular and oily.
e. Crystalline matters, salts, etc.:
a. Uric acid: in lozenges, rhombs, bundles or stellar masses of acicular crystals, etc.
b. Urates: amorphous granular form and in globular masses, with a crystalline radiating texture.
c. Phosphates: prismatic, plumose, stellar, cruciform, etc.
d. Oxalate of lime: octohedral and dumb-bell.
e. Chloride of sodium, in sparing quantity.
f. Compound granular bodies: the "exudation corpuscles" of some authors.
g. Pigmentary matters: blue and green.
h. Pus corpuscles.
i. Isolated vegetable cells and other accidental impurities of vegetable origin.
B.—*Post-mortem* contents of bladder. In addition to several of above histological elements, various results of disintegration or mechanical maceration of the mucous membrane of the bladder.

Of the histological elements above noted, several usually occurred in any given specimen of the respective evacuations; seldom or never all of them at once. Some were of very common occurrence, as mucus, granular corpuscles and epithelium; others very rare, as blood corpuscles. A few must be regarded in the light of accidental admixtures or impurities, as urates and siliceous matter in the stools; others were probably the result of incipient decomposition of the fluid evacuated, as vibrios and fungoid mycelium. Let us consider these elements more in detail.

I. *Vomit.*—Vomiting occurred chiefly during the collapse stage, or when collapse was passing into reaction—prior to the development of the fever stage. The fluid ejected varied greatly in quantity and colour. It exhibited various shades and combinations of brown, yellow, green, pink, red, and grey—frequently even in the same case, at different periods of the same day. These colour-changes are probably to be referred to the action of modified bile or blood—more frequently, I think, to the former than the latter. The specific gravity was generally low, varying from 1005 to 1010: the reaction with test-paper was generally acid, though sometimes neutral, or even alkaline in one or two cases; and the application of reagents showed that the fluid was sometimes albuminous—more frequently not. The specific gravity, acidity or alkalinity, odour, amount of sediment, and other characters, necessarily depended, in a great measure, on the nature and amount of the fluid and solid ingesta, and varied accordingly. In the first vomits, were usually discharged quantities of partially digested or undigested food—in some cases, masses or quantities of potatoes, cabbage, onions, barley, oatmeal, beef, herrings, and other articles of diet commonly used among the lower orders of the community, constituting the bulk thereof. In such cases, the sediment greatly preponderated over the fluid element of the vomit; but, in subsequent vomits, the sediment gradually decreased in quantity, becoming more homogeneous, —frequently resembling bran and water intermixed—until it was

present only as a flocculo-granular matter, or as mucous flocculi. In the latter case the fluid was whey-like, or of a very pale straw-yellow colour, the vomit closely resembling the ricy stools of the collapse stage: this, however, was very rare. The histological elements of such a ricy vomit were very similar to those of the ricy stools, with the exception, that in the vomit there was generally a considerable amount of pavement epithelium, and a larger quantity of food debris. The mucous flocculi consisted of hyaline bands or ribbons of mucus, colourless, and very delicately striated, bearing on their surface multitudes of small granular corpuscles, similar in general appearance to, but smaller than, pus corpuscles, and associated with a varying quantity of molecular and granular matter. Both in the vomit and stools—more frequently in the former than in the latter—the mucus occurred in delicate, almost transparent bands, devoid of the corpuscular or molecular element. This I have also observed in other diseases—as in the stools of chronic dysentery—illustrating the proposition, that the corpuscular element is not essential to the composition of mucus.

The granular corpuscles varied much in size. Their appearances under the action of water and acetic acid, separately or combined, also varied greatly. They sometimes remained unaffected, or the granularity was simply diminished, the size of the corpuscles being increased: a single or compound nucleus was sometimes developed, occupying a central or lateral position, the granularity of the cell-walls becoming diminished, or disappearing: or, lastly, a delicate transparent cell-wall became evident, more or less closely enclosing the corpuscles, which now assumed the characters of nuclei. Their histological characters varied to such a degree, as to render it impossible specifically to designate their true nature. In consideration of the reaction on them of acetic acid, they might be denominated mucoid or pyoid—these terms merely implying a general resemblance to mucus or pus corpuscles, without determining their nature or origin. A consideration of the circumstances under which they are developed, as well as of their microscopic characters, leads me to regard them as chiefly mucus corpuscles and epithelial nuclei. It must be remembered, however, that they are presented to us in a very young, or comparatively immature, condition: they are developed with great rapidity, and thrown off in infinite numbers, from the mucous surfaces of the intestinal canal, almost immediately after birth, or at least long prior to maturity. Hence they seldom present the characters of fully matured mucus corpuscles, or epithelial scales. They can be much better studied in the ricy stools than in the vomit of cholera, in consequence of the former frequently containing no other histological elements than mucus bands, and the granular corpuscles in question.

Epithelium was very common, and was most abundant in vomit containing little or no food debris, and having a scanty flocculent sediment. Of such a sediment it frequently constituted the bulk:

and it also occurred, sometimes to a considerable extent, in the frothy mucous scum. It often appeared more abundant in the later than the earlier vomits, and its quantity seemed, to a certain extent, proportionate to the intensity of the retching, or the expulsive or spasmodic efforts of the stomach and œsophagus. The epithelium was almost always of the pavement or tesselated form: it was frequently tinged greenish or brownish by biliary pigment, and it differed much in transparency, haziness, and granularity, being sometimes very dark and granular, or infiltrated to a considerable degree with oleo-albuminous globules. Its form also varied greatly, being sometimes very large, and irregularly hexagonal or polygonal, globular or ellipsoid; or smaller and variously elongated, fusiform or caudate. When small, shrunk, and elongated, it was frequently also hazy or granular. A great proportion of the epithelium was undoubtedly in a young or undeveloped condition. In only one case did I find cylindrical or conoid epithelium. Here the collapse was very severe and rapidly fatal, and the retching intense and protracted.

In the first vomits, I have already stated, that the nature of the food could often be easily distinguished by the naked eye. When the sediment became more homogeneous, and simply granular or granulo-flocculent, the microscope enabled me to detect fragments or isolated cells of the epidermis and parenchyma, epidermic hairs, spiral and porous vessels, and fragments of spirals from the fibrous tissues, starch corpuscles, and chlorophylle grains—the result of the partial disintegration of various culinary vegetables, or articles of diet of vegetable origin. These vegetables, or articles of diet, were chiefly potatoes, bread, oatmeal, and barley, cabbage, onions, carrots, turnips, or other ingredients of Scotch broth, besides sugar cane, and other less familiar substances. Associated with vegetable debris were usually fragments of muscular fibre from beef, mutton, or other forms of animal food; fish scales were found very rarely. Of all the vegetable tissues or cells above referred to, the most constant were the parenchymatous cells of the potato, and starch corpuscles, chiefly from the potato, oats, and barley. The former were large, irregularly globular, oval or oblong cells, usually full of delicate, but comparatively distinct, starch corpuscles: when empty, they appeared as delicate, shrivelled vesicles or sacs, having a slight brownish or greenish tinge. The starch corpuscles varied much in size: on the larger ones the striæ were usually distinct. When ruptured, or emptied of their contents, they frequently resembled entire or broken rings, the inner border having generally a lacerated edge. The peculiar reaction of iodine was observable in some, and not in others.

The isolated or disintegrated individual cells of the tissues above mentioned, probably include many, if not most, of the "annular bodies," the "cholera corpuscles or fungi," which so startled the histological and medical world during the cholera epidemic of 1848–9. At least, the ultimate elements of these tissues or substances, as observed by myself, correspond, in their characters, to those pub-

lished, as delineative of the bodies in question, by their original discoverers. I believe that potatoes, oatmeal, bread, and the vegetables of common broth, will furnish most of the forms of the once famed annular bodies; that they are not, therefore, fungoid in their nature or origin; and that they have no essential or causative relation to cholera. I have found them equally in other diseases—as in the stools of diarrhœa and dysentery. It is unnecessary to specify, in detail, the forms of these cellular bodies, observed by myself.[1] Many of them are simple globular cells, containing chlorophylle granules, aggregated regularly or irregularly round a central nucleus. This is the character of the gonidia of lichens—the green coloured cells lying immediately below the cortical layer of the thallus: hence, in former publications, in order to economise space and words, and to indicate their general appearance, I denominated these bodies *gonidiæ*. When emptied of their contents, they are delicate hyaline vesicles, and appear often as mere circles or rings, enclosing a free central area. Sometimes there is an inner dotted ring, probably produced by a puckering of the cell wall. This cell wall sometimes disappears, and the chlorophylle grains may then be found aggregated circularly round their nucleus, in regular masses, or free and intermixed with granular debris. Others of these bodies are of larger size, but very irregular shape: their walls are thick, and variously coloured, especially brown. Starch globules, partially broken up, are probably a common form of annular bodies: in this condition they frequently resemble the shrivelled sporangia of ferns. The non-action of iodine is not, I think, a sufficient disproof of such bodies being of an amyloid nature; for I have already mentioned that its usual reaction is sometimes absent, where the corpuscles otherwise bear indubitable marks of being starch. I have never seen annular bodies produced from spiral or annular vessels of plants, as was suggested in the report of the College of Physicians, London, in reference to the bodies described and figured by Brittan and Swayne of Bristol, though such vessels themselves are far from uncommon. Nor have I been able to trace any of them to medicines, for they occurred equally in cases where no medicines were given, or prior to their administration. Moreover, they were comparatively seldom found in the collapse stools, occurring chiefly in those of the reaction and fever stages; and their presence was coincident with the appearance of less equivocal forms of food debris. It will be evident, then, that I can see no satisfactory groundwork for the fungus theory of cholera, which I am not a little surprised to find still possesses powerful advocates.[2] We shall, hereafter, see that there is equally little foundation in fact for the animalcular and other theories of the cause or nature of cholera. We must evidently look elsewhere for a solution of the difficulty,—for as yet we are only on the threshold of the inquiry.

[1] Drawings and descriptions will be found in the *Association Med. Journal*, April 14, 1854.

[2] *Vide* Professor Daubeny of Oxford, "On the influence of the lower vegetable organisms in the production of epidemic diseases."—*Edin. New Philosoph. Journal*, July 1855.

On standing for a short time, a greasy scum usually formed, especially in the earlier vomits; this consisted of oil globules, associated with prismatic phosphates. Occasionally it contained small pellets or masses of concrete fat, resembling pieces of tallow or suet: under the microscope, these sometimes consisted of irregularly oval vesicles, marked by a central stellate radiating mass of delicate acicular or filiform crystals, at other times only oil globules and amorphous granular matter could be observed. The vesicles were also found separately, especially in the earlier vomits, containing a considerable quantity of food debris. The crystals were probably margaric acid, the result of the decomposition of fatty ingredients of the food.[1] Fatty matter also occurred in the earlier vomits containing a considerable amount of undigested food, in the form of irregularly globular, oval or oblong, dark or light, sometimes hazy or granular bodies, surrounded by or bristled over with divergent, acicular spiculæ or crystals, sometimes so delicate that they were scarcely visible. In some forms the spiculæ seemed few,

This engraving represents crystalline and other forms of fatty and oily matters occuring in the vomit of various cholera patients: the vomit having been chiefly ejected in the collapse stage.

short, and thick, and when broken or rubbed from the surface of the nucleus, which was generally light coloured, their roots, or points of origin, gave the latter the appearance of being covered over with

[1] Similar vesicles and crystals are figured in Hassall's "Microscopic anatomy of the human body," plate 19, fig. 5, which is labelled "Human fat vesicles, on the surface of which, crystals, supposed to be those of margaric acid, radiating from a centre, have appeared: their presence is to be regarded as an indication that decomposition has begun to affect the contents of the cells."

wart-like dark points. In other cases, the spiculæ were very numerous, barely visible as fine streaks, and appeared collapsed round or closely embracing the nucleus. The nuclei and crystals were sometimes separate, the latter occurring in irregular raphidian bundles, or in small confused groups. In the scum of the earlier vomits, concentrated by evaporation, the same crystalline matters occurred in the form of stellate radiating masses of thicker crystals, somewhat twisted or tortuous. The application of heat or ether caused the instantaneous disappearance of the crystals, and the agglomeration or fusion of the nuclei into oily masses. The nuclei often appeared compound, or as if made up of an aggregation of fatty globular masses. These curious spiculated bodies, which bear no inconsiderable resemblance to some microscopical insects, I have elsewhere described and figured as "echinus-like," or echiniform fatty bodies.[1] In cholera vomitings, they were frequently associated with sarcina. I have found them more abundantly in accidental vomitings in other diseases; indeed, so common did they appear to be, that I am led to regard them as the results of the natural or healthy digestion of fatty matters in the stomach or intestines. The

The engraving exhibits, by way of comparison, the same bodies as met with in the vomit of a patient labouring under an early stage of general paralysis of the insane. For particulars of the latter case, *vide Medical Times and Gazette*, August 5, 1854.

same or similar bodies have been described by other observers, who apparently regard the crystalline element as stearine—the result of the decomposition of the ordinary fatty matter of the food, by bile and the pancreatic juice, separately or combined.[2]

[1] "On the presence of certain crystalline fatty bodies in the vomit of cholera."—*Medical Times and Gazette*, July 1, 1854. And "Crystalline fats and sarcinæ ventriculi in vomited matters," with plates.—*Ibid.*, Aug. 5, 1854.

[2] Dr Leared of London appears to have produced similar bodies by the action of the pancreatic juice on fat out of the body, *vide* "On the pancreatic juice in relation to the digestion of fat."—*Med. Times and Gazette*, June 3, 1854; and report of the Physiological Society of London.—*Lancet*, May 20, 1854. Robin and Verdeuil also figure and describe similar crystals in their celebrated "Chimie anatomique."

On evaporating down the earlier vomits especially, two crystalline matters were generally found in varying proportions, viz., chloride of sodium and triple phosphates,—the former as irregular or broken cubes or tables, the latter usually as prisms of large size. Common salt was often present in considerable quantity; its source was obviously the ingesta swallowed shortly prior to the cholera seizure. On the other hand, the phosphates, though almost always present, were sometimes so in very small quantity. The same salts occurred, under similar circumstances, in the stools; but their relative proportion was reversed,—the phosphates being generally present in comparatively large quantity, the chlorides only to a small extent. Phosphates were frequently found in the scum or sediment on standing for a short time; but their numbers were greatly increased by evaporating down the fluid portion of the vomits. This process was usually necessary for the detection of the chlorides under the microscope.

In several cases, the vomit had a frothy, yeast-like, copious scum, of a pinkish or brownish red colour, precisely similar to the ejecta in the majority of cases of chronic vomiting, marked by the presence of sarcina. In all these cases, this entophyte was present in considerable numbers, sometimes entire, more frequently partially broken up or disintegrated, often very dark or granular. It was usually associated with epithelium, food debris, and fatty matter. But it also occurred in one or two cases where there was no such scum: in one case the vomit resembled coffee grounds in colour and consistence; in another, it was of a lead grey colour, and the sarcina was associated with undigested food, cylindrical and pavement epithelium, mucus and granular corpuscles. I have found it in five out of ten cases of cholera, where vomiting was a marked symptom; but in none of these cases could I ascertain that there had ever existed gastric symptoms of any kind; and in the fatal cases, where a necropsy was obtained, there was no dilatation of the stomach, or pyloric obstruction, as are stated by some authors almost invariably to exist in cases of sarcenaic vomiting.[1] I did not find it elsewhere than in the vomit. There are two reasons probably for its not being generally found in the stomach and intestines after death, firstly, because it is less seldom looked for in these localities; and, secondly, because it very speedily suffers disintegration. I have found it also in accidental vomitings in other diseases, and in cases where no prior gastric symptoms had existed, or did at the time exist; and I am inclined, therefore, to look upon it as an entophyte

[1] *Vide* "Clinical Lecture on disease of the Stomach and vomiting of Sarcinæ," by Dr Todd of King's College Hospital, London.—*Medical Times and Gazette*, July 1, 1854.

of common occurrence, in states of health deviating but slightly from the normal standard. I believe that it has been hitherto considered too much as a cause, and too little as an accidental result or concomitant of gastric disease; that it cannot be regarded in any other light than as indicative of a vitiated state of the system generally, or of the gastric mucous membrane especially, in virtue whereof the latter becomes the nidus of fungoid growths; and that its presence does not demand the exhibition of special remedies which ought rather to be directed to the amendment of the constitution. The mycelium and sporules of various species of fungi, constituting various forms of vegetable mould, were found in the scum of the vomit, as well as of the stools; but only at some stage of decomposition. They are found, however, under similar circumstances, in the vomit and stools of other diseases, and, indeed, in all decomposing animal fluids, and they are, therefore, far from peculiar to cholera.

The same remark may be made with regard to the presence of *vibrios*, which I have seen in thousands—in a state of very active movement—in vomit, very soon after it was ejected. But the fluid was in a state of incipient decomposition; these animalcules occur abundantly in all animal fluids, under similar circumstances, and their presence in the vomit and stools of cholera, therefore, can have no causal relation to the disease. They were more commonly met with in the stools than in the vomit, for the simple reason, I presume, that the former undergo decomposition more rapidly and readily. In the scum of one vomit, consisting in great measure of partially digested food, I found several acari and their fragments; their presence was evidently quite accidental.

In one case, pus was found in the vomit; but its origin was muco-purulent sputa from a bronchitic lung.

The contents of the stomach, as examined after death, were generally of a greenish, brownish, or chocolate colour, and viscid from copious admixture with mucus. However much mucus they contained, they never had a ricy appearance. In mucus scraped from the interior of the stomach, were found small quantities of the ultimate elements of various vegetable tissues,—in other words, forms of the "annular bodies," already referred to, along with epithelium, conoid, or elongated so as to possess a fusiform or fibroid appearance, generally very dark and granular; fragments of the gastric glands; mucus bands; granular corpuscles; compound granular bodies; and molecular debris coloured by the bile pigment.

II. *The Stools.*—During collapse, these were generally whey-like in colour and consistence, with a sediment of curdy mucous flocculi; sometimes they resembled "potato water," "or sage water," according to the description of the patients themselves. The sp. gr. was usually below 1010: they were alkaline, albuminous, and devoid of odour. As reaction and fever became successively developed, the stools gradually assumed a yellowish

colour, and thicker consistence, becoming like thin pea soup. The specific gravity rose from 1010 to 1015, fœtor was acquired, and a granular sediment occupied about one-third to one-half the bulk of the fluid evacuated. Albuminosity and mucus gradually disappeared,—fœtor, colour, and food debris, being substituted. In advanced fever, the stools became darker and more fœtid, frequently loam coloured, sometimes tarry in colour and consistence. The latter appearance has usually been attributed to an excess of vitiated bile; altered blood pigment, or the secretions of the intestinal canal, may also have a share in its production. In the progress of convalescence, a few scybalæ sometimes appeared, and the fæces gradually acquired their normal consistence and colour.

The mucous flocculi of the ricy or collapse stools consisted almost entirely of delicately striated, hyaline bands of mucus, associated with granular corpuscles, as in similar circumstances in the vomit. The fibrillated and corpuscular elements varied in their relative proportions, but they were always conjoined. Epithelium I very rarely found, unless in the form of epithelial nuclei, the granular corpuscles already described, when speaking of the vomit. In one or two cases, large, irregularly round, or angular scales of pavement epithelium occurred; but the patients were females, and their origin was probably leucorrhœal matter, for they were associated with pus.

Cylindrical epithelium I never observed. This is totally at variance with the statements or observations of Continental pathologists and histologists from Boehm to Professor Buhl of Munich, the latter of whom would appear to have found, in the cholera stools, sometimes "the entire uninjured covering of the intestinal villi."[1] This form of epithelium, however, I found abundant in the *post-mortem* contents of the intestines—especially those of the small intestines. Upon its presence or absence in the stools, hinges another of the fallacious and unstable, but specious and attractive, *theories* of cholera, viz., that its essential nature, or, at least, that an essential lesion is a denudation of the intestinal villi, or desquamation of the intestinal epithelium. Did we speculate on the characters of the contents of the intestines after death, without having regard to the histological elements of the stools passed during life, we should, undoubtedly, be led into the error of supposing that such denudation and desquamation really might or did exist during life; but I think that a dispassionate consideration of the facts of the case must lead to the conclusion, that these phenomena have no claim to be regarded as vital, but are merely the results of *post-mortem* or mechanical maceration. The same desquamation, and even further disintegration of the elements of the mucous membranes, occurs equally, after death, in the stomach and urinary and gall bladders.

Food debris was almost never met with in the collapse or ricy

[1] *Vide* Review in *Edin. Medical Journal*, Oct. 1855, of paper in *Henle's Zeitschrift für Rationelle Medicin, Band VI. Heft I.*

stools; but in those of the reaction and fever stages it was comparatively common, usually stained by altered or normal biliary pigment. I found, in all the cholera stools, including the ricy or whey-like evacuations of collapse, what I considered to be biliary reactions, which led me to regard the prevalent opinion that the bile is wholly deficient in such ricy stools, and that the function of the liver is, therefore, totally suspended during the collapse stage, as very fallacious and erroneous.[1] The histological elements of the food debris present in the stools, were the same in kind as those occurring in the vomit, differing, however, in degree. In the stools, fragments of epidermic and parenchymatous tissues were much less, while isolated and broken up ultimate cells and contents of these tissues were much more, frequent. The reason of this was obvious; in the stools, the food had undergone a much greater disintegration. Hence the forms of altered or partially broken up vegetable cells, which have been by some authors denominated annular bodies, were often more readily discoverable in the stools than in the vomit.

As in the vomit, also, a greasy scum usually formed on the surface of all the stools, on standing, consisting generally, when microscopically examined, of oil globules, prismatic phosphates, and molecular matter. In the fever stools—never in those of the collapse stage—fatty and oily matter occurred in two forms, viz., in pellets or masses of concrete fat, and as a scum of liquid oil. The former condition existed in several cases where the stools were dark and fœtid, resembling in appearance and consistence gruel or porridge. The masses varied from the size of a pea to that of an almond or bean, and floated on the surface of the stools. In some cases they had a very bright, greenish-yellow tinge, from the presence of biliary pigment, which, however, was easily removed by repeated washing in water; they were usually almond shaped masses, somewhat soft and blubber like. Under the microscope, they were resolved wholly into oil globules. Others were of much firmer consistence, and whiter colour; they were irregular in shape, resembled pieces of common suet, and consisted of an aggregation of globular or oval vesicles, each marked by the central radiating masses of filiform crystals, mentioned when speaking of similar fatty masses in the vomit. The interspaces between the individual vesicles seemed made up chiefly of oil globules.[2] In two cases, a scum of fluid oil—of a bright, greenish-yellow colour—occurred; in both cases the stools being those of convalescents from a protracted fever stage. Where fatty or oily matters did occur in the stools, they appeared to bear no relation to the intensity or type of the disease, or to the characters of the matters evacuated. Some authors are inclined to connect all cases of the appearance of fat or

[1] "Clinical notes on Cholera."—"Presence of bile in the cholera evacuations." *Association Med. Journal*, March 10, 1854.

[2] "Cases of cholera, illustrative of the presence of fat in the fæces, and of certain alterations of the blood."—*Edin. Monthly Med. Journal*, Aug. 1854.

oil in the fæces with the supposed necessary existence of organic disease of the pancreas; but I can neither see the necessity of, nor ground for, such an assumption.

The same salts, also, existed as in the vomit, though, as I have already mentioned, in different degree. Phosphates were almost invariably present, both in scum and sediment, immediately after being voided, increasing in quantity when the stools were allowed to stand, or were concentrated by evaporation. According to the freshness of the evacuations, the period of the disease at which they were voided, the degree of concentration, the rapidity of evaporation, natural or artificial, the nature of reagents added, and similar circumstances, their crystalline form varied considerably. Their most usual and natural form was the prism—perfect or broken, varying greatly in size; but they occurred in an infinity of plumose, stellar, and cruciform conditions. Similar changes in the conditions of examination produced similar modifications in the crystallization of the salts of the urine and vomit. I have found phosphates of similar forms in all fæces I have examined, both healthy and diseased, in similar states of incipient decomposition. Chloride of sodium was only detected on evaporating down the filtered liquid portion of the stools; and, though generally present, it varied greatly in amount in different cases, and in stools at different periods of the disease, in the same case. Its crystalline form appeared to vary in the same way as that of the phosphates, though to a much less degree. In one or two cases—in stools evaporated to dryness—I found large dark brown globular urates of ammonia, similar to those which frequently occurred in like circumstances in the urine; but the patients were females, and I attributed their presence to the accidental intermixture of a small quantity of urine. Siliceous crystalline matter I also found sparingly; but it was evidently present, as an impurity or accidental admixture, its source being probably dust from the floor of the wards.

Blood corpuscles were distinguished only in one case—in a stool passed at, or immediately prior to, death:—it contained a quantity of viscid mucus, deeply stained and streaked with blood, and resembled some of the bloody stools of advanced dysentery. Hyaline bodies, which resisted the action of heat, ether, and other reagents, were also present, in sparing quantity; they were usually considerably larger than pus corpuscles. Fungoid mycelium and vibrios occurred, under the same circumstances as in the vomit.

The *post-mortem* contents of the intestines were sometimes of nearly as low specific gravity as the stools, but they were generally much more viscid from admixture with mucus; had a deep colour, due to altered biliary matter; were alkaline and fœtid, and generally contained some proportion of fluid fæculent matter. They were never ricy, or closely resembling the characters of the whey-like collapse stools; and they were seldom simply mucoid. In one case only, the intestines were lined by pure mucus, no foreign intermix-

ture of any kind being detectable. This mucus, when scraped or washed from the surface of the mucous membrane, agitated with water, and allowed to stand, gave to the fluid quite the appearance of the ricy stools. The mucous flocculi, on being examined by the microscope, differed in this, however, that they contained quantities of cylindrical epithelium, isolated, or in patches of varying size—the apices of the cells being united by a delicate hyaline continuous membrane;—and very rarely a few blood corpuscles. Mucus, scraped from different parts of the canal, always contained cylindrical epithelium, often in process of disintegration; frequently portions of villi, which were always nude; and occasionally shreds of mucous membrane and blood discs. The latter, when present, were most abundant towards the termination of the large intestine, which was generally the chief seat of hyperæmia or ecchymosis, sometimes of a hæmorrhagic oozing. Cylindrical epithelium occurred in much greater quantity in the small than in the large intestines, whose contents also were generally darker and more fœtid. In some cases, flocculi of false membrane, or effused lymph, were found; in others, there appeared to have been an exuviation of the mucous membrane itself.[1] The above histological elements constituted the chief points of difference, microscopically, between the contents of the intestines after death and the evacuations passed during life. The important bearing of this difference on theories founded on the pathology of cholera, I have already pointed out.

III. *Urine* was never obtained during the true collapse stage: it was first passed during the reaction stage, or sometimes not until fever had fairly set in. During these periods, it sometimes became necessary to remove it by the catheter. Whether voided naturally, or artificially drawn off, the urine first passed was generally turbid, acid, pale amber-coloured, devoid of odour, of specific gravity varying from 1010 to 1015, and invariably albuminous: it was, moreover, small in quantity, and contained a scanty flocculent mucous sediment. At subsequent stages of the disease, the density also continued comparatively low, and sometimes the quantity remained small: the albuminosity and mucous sediment gradually disappeared, and gave way to phosphates, urates, and other salts. The histological elements varied greatly from day to day, even in the same case, and especially during the fever and convalescence.

Mucus bands and granular corpuscles, similar to those occurring in the vomit and stools, were frequently present in the mucous sediment of the urine first passed, associated generally with a considerable amount of epithelium. The mucous bands were much more delicate and indistinct—less seldom striated and fibrillated—than in the stools or vomit, and very frequently entangled various crystalline matters. The granular corpuscles were also less frequent, and less

1 *Vide* "Histology and Pathology of Cholera"—"Clinical Notes on Cholera."—*Association Med. Jour.*, April 14 and 21; May 12; and June 10, 1854.

distinct; their nature was also more frequently ambiguous, from their being associated with pus corpuscles: they were sometimes deeply tinged with various urine pigments.

Epithelium, in a multitude of transitionary forms, between the large globular or polygonal pavement cell, and the elongated conoid or fibriform one was generally present, in some shape or degree, in the earlier urine passed. It sometimes occurred in patches, of pentagonal or hexagonal scales, but more generally in the form of isolated scales, which were quite globular, very large, or slightly superior in size to pus corpuscles, or irregularly round, oval, or ellipsoid, presenting sometimes bulgings, occasionally caudate, irregularly square or angular, boat-shaped, crescent-shaped, fusiform, or fibroid. As in the vomit, these scales varied much in their degree of transparency, haziness, or granularity, being sometimes infiltrated with oleo-albuminous granules. The flocculent sediment of the first urine passed, sometimes consisted of little else than pavement epithelium, which had been thrown off abundantly from the surface of the urinary apparatus or passages. Globular epithelium, from the renal tubuli, was generally intermixed with pavement epithelium from the bladder, but to a proportionally small extent—not such, in my opinion, as to justify the idea that desquamation of the epithelial lining of the renal tubuli is a characteristic feature of cholera.

Frequently associated with epithelium, in the first urines, were fibrinous casts or cylinders; sometimes of considerable length, convolute or twisted, and transparent, or faintly striated; at other times in more or less straight fragments, dark or granular, or covered by, or entangling, granular corpuscles, oil globules, and molecular debris. They existed in four out of twelve cases. They gradually disappeared with the epithelium in the later urine, or that passed during fever and convalescence. The histological elements of the first urine, so far as they have been above described, therefore, resemble those of the urine in some cases of Bright's disease; and some authors regard the presence of the large amount of vesical pavement epithelium as the only point of histological diagnosis. The presence of mucus, epithelium, and pus, again cause a manifest similarity to many cases of vesical catarrh.

Of the crystalline ingredients of the urine, uric acid was present in two or three cases, chiefly in the earlier urine passed. In some specimens of the urine, it formed a sparkling crystalline scum; in a few, it covered the sides of the urine jar with a granular coating; in others, it occurred in the sediment entangled among mucus, or associated with other crystalline matters. Its crystalline form varied greatly; most frequently it assumed the shape of lozenges or cubes; sometimes it occurred in oblong bundles or stellate masses of acicular crystals, or as small delicate prisms. The size of the crystals also varied much, and the colour presented various shades of brownish yellow, red, and crimson. In one case, it appeared in the first urine evacuated on the third day of the disease, which was very

mild, terminating in recovery without the intervention of a distinct fever stage. The urine was acid, albuminous, and of specific gravity 1020; containing, in addition, mucus, granular corpuscles, fibrinous casts, epithelium, urates, and dumb-bell oxalates. It occurred either in the scum, sediment, or on the sides of the jar, and in different crystalline forms, for several days. In another case, it appeared during the fever stage, and was coincident with the acmé of an exanthematous eruption on the body of the patient, accompanied by symptomatic fever, on the eighth day of a severe case. The urine was still slightly albuminous, and of sp. gr. 1010. It occurred also in the urine of the ninth day: on allowing this to stand for two days, the whole uric acid was found converted into urate of ammonia. On the 10th day uric acid also appeared in sparing quantity; on the 12th there was no trace; on the 14th it reappeared, along with pavement epithelium. Urate of ammonia was frequently present in its usual amorphous form. When the urine was concentrated by evaporation, however, it assumed the appearance of large irregularly globular masses, of a light or dark brown colour, having a centrifugally radiating crystalline texture, and herein resembling the fibrous concretions of iron pyrites, often found in clay ironstone. When of large size, these masses were very fragile, and hence were frequently found in fragments. In studying the forms of the various crystallizable or saline constituents of the urine, I was in the habit of allowing certain specimens to undergo spontaneous evaporation and decomposition, to limited degrees: other specimens were concentrated by heat, and various reagents applied. The result was, as might be expected, a great variety of crystalline forms—greatest in the case of the phosphates. The triple phosphate occurred frequently—chiefly in the forms mentioned when speaking of their presence in the stools; sometimes alternating with the urates. Oxalate of lime was present in rare cases, but, both in the dumb-bell and octahedral form, chiefly in the earlier urine. In one case, octahedral oxalates were present in the first urine, passed on the third day of the disease, which was mild, terminating speedily in recovery without the intervention of a specific febrile stage. The urine was acid, of sp. gr. 1022, and loaded with urates. The sediment contained, in addition, epithelium, fibrinous casts, pus, and exudation or compound granular corpuscles. In another case, dumb-bell oxalates appeared in the first urine, passed on the third day of the disease, which was very severe. The urine was turbid, acid, albuminous, and of sp. gr. 1015. The next urine passed by the patient, on the sixth day, was phosphatic; while that passed on the eighth day contained a uric acid sediment. Both patients were young females. In a third case—a male—dumb-bell oxalates were associated with uric acid, fibrinous casts, epithelium, and mucus, in the first urine, passed on the third day. These crystalline sediments, as we have seen, frequently alternated with each other, or suddenly appeared and disappeared. One day the urine might be high-

coloured, containing beautiful sparkling crystals of uric acid; on another, it was perhaps turbid, from supersaturation with urates; on a third, it might contain a copious sediment of phosphates or pus; and, on a fourth, only a slight mucous sediment, entangling a few oxalates. Again, epithelium, casts and mucus, on the one hand, and phosphates and urates on the other, appeared sometimes to bear a relation to each other; where the one group was common, the other was rare. The former were met with, chiefly or only in the first specimens of urine passed; the latter, in the urine of fever and convalescence. As in other febrile diseases, there was sometimes observed a sudden and peculiar change in the histological characters of the urine—more particularly its crystallizable or saline constituents—coincident with a crisis, exacerbation, or remission, in the symptoms of the disease—especially during the typhoid or true fever stage. Such changes, however, were not regular or constant; or, in other words, did not bear a specific relation to the phenomena or phases of the disease. Hence, they were of little or no diagnostic value. They consisted most frequently in the appearance, reappearance, and disappearance of the urates, and of uric acid.

In a few exceptional cases, a beautiful Prussian-blue colouring matter was developed in the first urine passed, either after the addition of nitric acid, or simply on concentration by rapid evaporation. In one case the urine was acid, albuminous, of sp. gr. 1015, and contained casts, epithelium, mucus, oxalates, urates, uric acid, and compound granular bodies. Nitric acid, without the previous application of heat, caused the urine to assume a brownish-red colour, and also produced a brownish flocculent precipitate, which, on standing in a test tube, became granular, and of an earth-brown tint. The sides of the tube were at the same time coated sparingly with a greenish-blue granular deposit, which, under the microscope, appeared to consist wholly of bluish granular matter entangled among mucus fibrillæ. Hydrochloric acid produced a similar brownish discoloration and precipitate. Aqua potassæ developed a greenish colour; while aqua ammoniæ produced no change in colour. On standing for some time after the application of heat, and the addition of nitric acid, a yellowish-green colouring matter was found to have tinged certain granular corpuscles, and a cobalt blue pigment certain other corpuscles, along with epithelium scales, cotton tubes, and other debris, occurring in the urine sediment. Some corpuscles were dark-bluish-green and granular; others were light-green, and semi-transparent; while others were also non-granular, and of a fine cobalt blue. In the latter case, the corpuscles were frequently elongated at one extremity or caudate. Within certain limits, the intensity of the colouring matter increased in proportion to the period that had elapsed since the application of heat and nitric acid. It appeared to have a strong affinity for textile fabrics or other vegetable fibre; for I have seen it attached to linen fibres or cotton tubes, when it did not exhibit its presence elsewhere in the urine-

scum, or sediment. On another occasion, in evaporating down several specimens of first urines, when the liquid had arrived at a certain stage of concentration, the scum assumed a deep Prussian-blue colour. These specimens were acid, albuminous, and of sp. gr. 1015 to 1020; nitric acid produced an orange-red or pink reaction, according as heat was previously applied or not. All the specimens were in stages of decomposition, having stood exposed to the air for some days after they were voided. Under the microscope, the scum, when cool, was found to consist chiefly of globular urates, all having a bluish tinge.[1] In all the above cases, the urine in which the discoloration or colouring matter was found, was that first passed after collapse; none were discovered in subsequent urines. Other observers have noticed a violet discoloration, followed by a deposit of a blue colouring matter, after the addition of nitric acid in the urine of convalescence also.[2]

Associated with the epithelium, were sometimes found large roundish or oval cells resembling large epithelium scales, but having nucleolated nuclei, more distinct in outline, and usually darker and more granular. With these, were generally intermixed pus corpuscles, sometimes in great quantity,—the urine sediment being distinctly purulent. Both of these elements occurred in females, and their origin was probably leucorrhœal matter accidentally intermixed with the urine. Compound granular bodies, varying in size, granularity, and colour, frequently resembling the bodies described as "exudation corpuscles," by some authors, were also frequently present, associated sometimes with oily matter in the form of oil globules. Isolated vegetable cells, similar to those already described as occurring in the vomit and stools, likewise existed in a few cases; but they were evidently to be regarded as impurities or accidental admixtures.

The *post-mortem* contents of the bladder, or the urine drawn off by catheter after death, generally resembled that last evacuated prior to death, with the exception that it was always turbid, and contained a more or less copious flocculent sediment. This was made up chiefly of the disintegrated elements of the mucous lining of the urinary bladder and passages,—the result apparently of *post-mortem* or mechanical maceration, as in the analogous cases of the stomach and intestines. It frequently contained greatly elongated forms of epithelium, fusiform, fibroid, and conoidal; the latter sometimes presented two nuclei, the cell wall having a ventricose bulging opposite each. Caudate or horned forms of globular or pavement epithelium were also met with. Associated with epithelium and mucus, oil globules, and non-oleaginous hyaline globules, were also sometimes detected.

[1] "The development of a blue colouring matter in the urine of Cholera."—*Med. Times and Gazette*, May 12, 1855.

[2] Paper by Mr Osborn on the same subject.—*Med. Times and Gazette*, March 31, 1855.

I have still a few remarks to make on the histological characters of the blood, and of some of the abdominal and thoracic viscera. In one case the white corpuscles of the blood appeared to be present in excess. I have found a similar leucocythemic condition in a considerable variety of constitutional affections, especially among the insane;[1] and, I believe it to be a state or symptom—certainly not an independent disease—of comparatively common occurrence. In a few cases, the red discs were hazy or granular. In one case they presented peculiar characters, some having the appearance of a delicate cell wall developed round a single or compound nucleus,—others having the cell wall developed in such a direction as to give the corpuscles caudate or elongated characters, while in others the form was still more irregular. Water rendered these appearances somewhat more distinct, but the subsequent addition of acetic acid made little change.[2] The red discs also sometimes showed a proneness to form rouleaux. Similar structural alterations, or exceptional conditions of the blood have been described by other observers as occurring in cholera. But they are rare—are not peculiar to cholera—have probably existed prior to the development of that disease, and are, therefore, unconnected in a causal relation therewith. Blood changes in cholera, where they exist, are more probably due simply to exosmose, or to pre-existing constitutional conditions, than to structural alterations or morbid conditions essentially related to the disease itself. Some prevalent views on the pathology of the blood in cholera, appear to me to be very limited or very erroneous. I have never seen the tarry viscid condition, formerly so frequently described. I have certainly found it sometimes dull in tint, dark in colour, grumous, and very fluid, showing no tendency to coagulation; but, on the other hand, it more frequently contained abundance of decolorized clots. Both conditions, according to my own limited experience, are equally common in other diseases. I have been able to discover no difference between the blood in cholera, and that in a multitude of affections where pulmonary obstructions of any kind have prevented its due or ordinary oxygenation. The lungs, in cholera, participate in the general hyperæmia of the internal viscera; and, as a consequence, œdema is not an unfrequent pulmonary lesion.

The renal epithelium was sometimes infiltrated with oily globules and granules; but, I do not think that this is sufficient to warrant the conclusion at which some observers have arrived, that the kidneys are hence the seat of fatty degeneration. I think it probable, that had the disease, in some of these cases, progressed towards a favourable, instead of a fatal, termination, this infiltration of oily matter would gradually have disappeared; and that, where it was a permanent state of the epithelium, it probably depended on pre-existing organic disease

[1] "Histology of the Blood in the Insane."—*Journal of Psychological Medicine*, Jan. 1855.

[2] *Edinburgh Monthly Medical Journal*, Aug. 1854, *loc. ol. cit.*

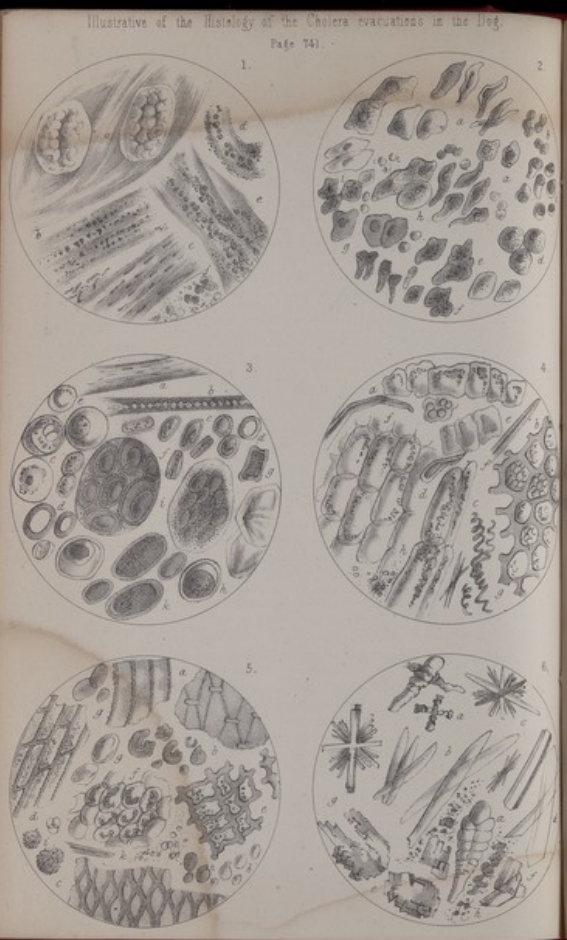
of the kidney, which might have been aggravated, but was not originated or developed, by cholera. In some cases, I found the kidneys presenting the characters of various features or stages of Bright's disease.[1] The epithelium of the liver was also frequently, and that of the gall bladder and bronchi sometimes, infiltrated in a similar way with oily globules and granules; and, we have already seen that the epithelium thrown off from the surface of the gastro-intestinal mucous membrane, and of the urinary bladder and passages, frequently presented like appearances. The hepatic epithelium was sometimes hazy or granular, without exhibiting distinct oil globules. The gall bladder was usually coated with inspissated mucus, abounding in cylindrical epithelium of a very bright greenish-yellow colour, associated sometimes with oil globules and granules. It was usually distended with bile, which appeared healthy, or at least exhibited no marked departure from the normal state. This statement is quite opposed to generally recognised opinions on the conditions of the bile in cholera. I have elsewhere—and in some detail—stated the grounds upon which my own convictions are founded.[2] The very term *cholera* appears to me a misnomer, founded on mistaken views on the pathology of the disease. The presence of cylindrical epithelium in the gall bladder, has given rise to the opinion that here also there has been a vital desquamation of the epithelial lining; but, as in the analogous case of the intestines, the phenomenon is probably a *post-mortem* one. The bloody juice squeezed from a lung in a state of pneumonic consolidation, contained pus corpuscles, but no exudation or compound granular bodies. The cream-like contents of a tubercular cyst about the size of a sixpence situated in the apex of a lung, contained pus and exudation corpuscles, cylindrical ciliated epithelium, and the vague entities denominated by many observers, "Tubercle corpuscles," besides molecular debris—the result of the disintegration of pus.

Before leaving this subject I would remark, that none of the above histological appearances are peculiar to cholera, but, on the contrary, most of them are common in a great variety of diseases. The intestinal evacuations are generally regarded as the most peculiar features of cholera: histologically they are not so, but closely resemble the stools of dysentery and diarrhœa. In ordinary physical characters the difference is generally great; sometimes, however, imperceptible. On the points of resemblance and distinction, however, I cannot here enter.[3] In connection with this subject, I have examined the stools in many cases of simple, acute and chronic diarrhœa, of the diarrhœa of phthisis and of chronic dysentery; and

[1] Pathology of Cholera—"Clinical Notes on Cholera."—*Association Medical Journal*, May 12, 1854.

[2] *Association Med Journal*, March 10, 1854. *Ol. cit.*

[3] They will be found somewhat fully detailed in a paper on the "Histology of Mucoid Evacuations in Diarrhœa, Dysentery, and Cholera."—*Association Medical Journal*, March 16, 1855.



I have found the same albuminosity, the same histological elements, the same excess of salts and of water, or serum; the latter, however, never to the same extent. The stools of the premonitory diarrhœa, and of the reaction and fever stages, closely resembled those of common diarrhœa in ordinary physical, as well as histological and chemical characters. Between those of the collapse stage, the ricy stools, and the purely mucous discharges of dysentery, the resemblance was less close. In rare cases, however, the bloody stools bore an intimate resemblance to those of advanced dysentery. In the latter disease, the proportion of serum is greatly less, and the mucus is more cohesive, viscid, and stringy; when mechanically broken up, however, and agitated in water, a ricy fluid, exactly resembling the rice water discharges of cholera, was produced. The corpuscular element of the mucus in dysentery is generally larger, more distinct, less abundant, and more frequently showing nuclei and nucleoli under the influence of acetic acid; moreover, it is sometimes altogether absent, as it was in one case where the stool was of an apple-green colour and jelly-like. In the same disease, also, pus and blood corpuscles are of frequent occurrence, the reverse being the case in cholera.

II. *Histology of the Cholera Evacuations, etc., in the Dog and Cat.*[1]

To exhibit at once the chief points of resemblance and distinction between the histological characters of the cholera evacuations in man and in the dog and cat, I shall here also shortly tabulate the principal appearances. But, in so doing, it is necessary to state that I had not the same facilities or opportunities for making separate microscopical and chemical examinations of the several evacuations in these animals as in man. The vomit and urine were generally so mixed up with the fæces that I was very seldom able to separate them for examination. The histology of the solids and fluids, after death, was much more fully and satisfactorily examined than that of the evacuations voided during life, the reverse being usually the case in the human subject. Hence the table can only be considered approximative, but it is probably sufficiently accurate to point out the close resemblance between the microscopical characters of the evacuations and the blood and viscera in the cholera of man, and in that of the animals which were the subjects of my experiments. To a certain extent, this resemblance was due to the mode of experiment, for instance, in regard to the nature of the food debris occurring in the stools, the animals having been fed on the evacuations of patients labouring under different stages of

[1] For the circumstances under which these animals were affected with cholera, I must refer the reader to the *Edinburgh Medical and Surgical Journal*, "On the Communicability of Cholera to the Lower Animals," April and October 1854; *Association Medical Journal*, "Clinical Notes on Cholera," September 15 and December 15, 1854; and *Gazette hebdomadaire de Médicine et de Chirurgie* (Paris), November 24, 1854.

cholera, evacuations containing the elements of food which I have already detailed in speaking of the cholera vomit in man. Most of the elements therein mentioned occurred in the stools of the dogs, many of them having passed through their intestinal canal unaltered, others having undergone only further disintegration. The most common of these were the parenchymatous cells of the potato, portions of the epidermis of barley grains, starch corpuscles from the oat and potato, many of the forms of isolated vegetable cells which I have already spoken of as gonidic and annular bodies, with a few hairs and fragments of spirals.

I. *Vomit.*
- *a. Mucus*—fibrillated bands, with or without granular corpuscles.
- *b. Granular corpuscles*—probably chiefly epithelial nuclei.
- *c. Epithelium*—pavement, seldom perfect in form or transparency, more frequently variously elongated or otherwise altered, and hazy, dark, or granular.

II. *Stools.*
- *a. Mucus*—fibrillated bands, with or without granular corpuscles.
- *b. Granular corpuscles*—generally larger and more distinct than in man, probably from earlier period at which they were examined after evacuation or death.
- *c. Debris of vegetable food*—Similar in kind to that mentioned under head of cholera vomit in man.
- *d. Oily matter*—only in form of oil globules.
- *e. Crystalline matters or salts*—phosphates and chloride of sodium, as in man, the former in a less variety of crystalline forms.
- *f. Fungi*—mycelium of various kinds of mould, as in man.
- *g. Ova of intestinal entozoa*—in various stages of development.
- *h. Compound granular bodies*—variously coloured, most frequently greenish-yellow.
- *i. Molecular matter*—similarly coloured.
- *k. Impurities*—
  1. Licked from the surface of their own bodies—
     Hairs.
  2. Licked from the floor—
     Siliceous crystalline matter and other elements of dust.

III. *Urine.*
- *a. Mucus.*
- *b. Granular corpuscles.*
- *c. Crystalline matters or salts*—
  Phosphates.
  Chlorides.
  Urates.
- *d. Oily matter*—only in form of oil globules.

The vomit, when distinguishable from the fæces, was a somewhat viscid, frothy matter, of a pale greenish-yellow tinge, consisting almost entirely of epithelium and granular corpuscles, both having a similar tinge, especially the latter. The epithelium was seldom rounded or broad, but more generally narrow and elongated—fusiform or navicular—sometimes very dark and granular. On the addition of acetic acid, a delicate cell wall became visible round most of the granular corpuscles, whose granularity was sometimes diminished; they never became compound, nor exhibited nuclei or nucleoli under the action of the acid.

During the earlier stages of the diarrhœa, produced by the nature of the animals' food, the stools were somewhat viscid, sometimes tarry in appearance, more generally of a brownish-green colour, containing much biliary pigment, a considerable amount of food debris, and frequently entozoon ova and phosphates, but little or no mucus. As cholera was developed, the stools became much more copious and watery, but still generally of a brownish-green tint. On minute inspection, however, they were found to abound in small flocculi of mucus. The disease proved fatal prior to the discharge of purely ricy or mucous matter, for the intestines were found, on *post-mortem* examination, lined or loaded with flocculent mucus, sometimes almost pure and colourless, or variously covered by or intermixed with greenish semi-fæculent matter. When the mucus of the intestines, freed from fæculent admixture, where it existed, was washed out by a stream of water, or was scraped from the surface of the mucous membrane, agitated with water, and the fluid allowed to stand, the mixture resulting could not be distinguished by the naked eye from the ricy stools of the collapse stage of human cholera. The flocculi, under the microscope, sometimes presented the appearance of delicately fibrillated bands of mucus, without being associated with or covered by granular corpuscles; the latter, however, were generally present, and varied from the size of pus corpuscles to about half that size. Under the action of acetic acid, alone or aided by boiling, some of these bodies became less granular, others were unaffected; some exhibited one or more nuclei, while around others a delicate cell wall was developed. They were frequently of a greenish-yellow tinge, as were sometimes also the mucus bands themselves. The mucus scraped from the surface of the stomach and intestines consisted sometimes solely of bands of mucus, so delicately striated as to be almost invisible; frequently isolated vegetable cells were adherent or intermixed in more or less abundance. Mucus from the stomach contained cylindrical and pavement epithelium, very dark and granular in one case; that from the œsophagus also contained granular epithelium and a number of isolated vegetable cells.

The urine could not be fully examined. It was generally turbid, having a whitish granulo-flocculent sediment. The vesical epithelium was only hazy; the renal epithelium was infiltrated with oil globules. The blood corpuscles appeared normal. Tracheal mucus contained normal cylindrical epithelium. Fluid squeezed from the lungs and bronchi contained epithelium, sometimes dark and granular, associated with oily granules, and a number of hyaline globules. The hepatic epithelium was slightly filled with oily granules and globules. The mucus of the gall-bladder contained cylindrical epithelium, isolated and in patches, besides a large number of granular corpuscles and oil globules, all of a very bright greenish-yellow colour.

The epithelium of the interior of the vagina, uterus, and fallopian tubes appeared healthy.

EXPLANATION OF PLATES.

Plate I.

Fig. 1. Mucus-fibrillæ and mucus-corpuscles, etc.
- *a.* From collapse vomit, ejected on first day: fibrillæ unassociated with corpuscles: case recovered.
- *b.* From collapse (rice-water) stools, evacuated on second day: case recovered: fibrillæ entangling hyaline globules.
- *c.* From collapse stools (muco-granular sediment of a dark lead grey colour) passed on first day in same case.
- *d. f.* From collapse (rice-water) stools passed immediately before death: case fatal in eight hours.
- *g.* From same stools: mucus-corpuscles, showing effects of acetic acid.
- *e.* From collapse vomit: case fatal in eighteen and a half hours.

Fig. 2. Forms of epitheljum—tesselated, cylindrical, fusiform, caudate, etc., in vomit.
- *a.* From collapse vomit: case recovered.
- *b.* „ „ case fatal in eight hours.
- *h. i.* „ „ in same case: *i.* shows effect of acetic acid.
- *c. k.* „ „ case recovered.
- *d.* „ „ cholerine: case recovered.
- *e.* „ „ case fatal in typhoid stage.
- *f.* „ „ case fatal in eighteen and a half hours.
- *g.* „ „ case fatal in typhoid stage.
- *l.* „ „ case fatal in acmé of consecutive fever.

Fig. 3. Forms of epithelium, etc., in urine.
- *a. c. g. h. i. o.* From urine first passed (in fever stage): sediment purulent (probably leucorrhœal): case recovered: *i. o.* show effect of acetic acid.
- *p.* Compound granular bodies from same urine.
- *b.* From collapse urine (first passed): case recovered.
- *d. f. l.* „ „ case fatal in acmé of consecutive fever.
- *e. g.* From urine first passed (in reaction stage): case recovered.
- *k.* From urine of advanced fever stage: case marked by an urticario-rubeoloïd exanthem: recovered.
- *m.* From urine first passed (in reaction stage): cholerine: case recovered.
- *n.* From urine passed during convalescence in same case.

Fig. 4. Forms of epithelium, etc., in urine.
- *a.* From urine drawn by catheter at the autopsy made the day after death: case fatal in typhoid stage.
- *b. c.* From urine passed in reaction stage: cholerine: case recovered. (*Vide* Fig. 3. *m. n.*, and Fig. 5. *c. d.*) *e.* Fibrine cylinders, transparent or granular.
- *c.* From urine passed in collapse: cholerine: case recovered.
- *d.* From urine first passed (in fever stage): case recovered.
- *f.* From urine passed in advanced consecutive fever: case recovered.
- *g.* From urine drawn off by catheter two days after death: case fatal in typhoid stage.

Illustrative of the Histology of the Cholera evacuations in man.

Fig. 5.

a. In fluid removed from stomach at autopsy: case fatal in three and a half hours.

b. In mucous contents of intestines removed at autopsy made the day after death: case fatal in eighteen and a half hours.

c. d. From urine first passed (in reaction): cholerine: case recovered: after addition of nitric acid, and standing for a day, the corpuscles at *c.* assumed a deep cobalt blue, and those at *d.* a Scheele's green colour.

e. Cylindrical ciliated epithelium in juice expressed from a condensed lung at autopsy made day after death: case fatal in consecutive fever.

f. Hyaline bodies in urine, drawn off by catheter at autopsy: case fatal in typhoid stage.

g. Pavement epithelium from collapse (rice-water) stools: case fatal in eight hours: probably from leucorrhœal matter.

Fig. 6

a. Altered blood corpuscles, in bloody juice expressed from a condensed lung at an autopsy made the day after death: case fatal in consecutive fever: action of acetic acid is exhibited on some.

b. In bloody juice squeezed from the bronchi at the autopsy in the same case.

c. e. h. n. o. Isolated cells of the tissues of various vegetables consumed as food, from collapse (rice-water) stools: case recovered.

d. From stool passed in fever stage: case recovered.

f. From collapse vomit and stools: case fatal in typhoid stage.

g. From collapse (rice-water) stools: case fatal in eight hours.

k. From reaction stools: case recovered.

l. m. „ case fatal in acmé of consecutive fever.

p. From collapse vomit: cholerine: case recovered.

* These cellular bodies probably represent the chief forms of the "cholera corpuscles," "cholera fungi," and "annular bodies" of observers during the epidemic of 1848–9. Some of the forms,—especially *c. k. l. m. n.*—I have elsewhere denominated "gonidic" bodies.

Plate II.

Fig. 1. Parenchymatous cells and starch corpuscles from the potato, oatmeal, barley, etc.

a. Parenchymatous cells of the potato from collapse stools: case recovered.

b. k. Potato cells from collapse stools: case fatal in consecutive fever: at *b.* the starch corpuscles are coloured by iodine.

c. k. Starch corpuscles in collapse stools of same case: *k.* coloured by iodine.

e. g. e. f. l. Starch corpuscles in collapse vomit: case recovered: *e. f.* coloured by iodine.

d. Empty potato cells in collapse vomit: case fatal in eighteen and a half hours.

i. Potato cells, with inclosed starch corpuscles, in collapse vomit of same case.

Fig. 2. Disintegrated elements of the tissues of various vegetables used as food; sarcina; muscular fibre, etc.

a. Sarcina ventriculi (entire) in collapse vomit: case fatal in eighteen and a half hours.

c. d. Spiral and annular vessels and isolated spirals, in same vomit.

f. Compound granular bodies in do.

b. Fragment of muscular fibre in collapse vomit: case fatal in consecutive fever.
c. Porous vessels in collapse vomit: case fatal in eight hours.
g. Sarcina ventriculi (broken up) in collapse vomit: case fatal in typhoid stage.
h. In collapse vomit: fatal in consecutive fever.
i. In collapse stool of same case.
k. *l*. In consecutive fever (pea-soup) stools: case recovered.
m. In collapse vomit: cholerine: case recovered.

Fig. 3. Fragments of the parenchyma, epidermis, and fibrous tissue of various vegetables used as food.
a. In fever stools: case recovered.
b. *c*. In collapse vomit: case fatal in eighteen and a half hours: *c*. is a portion of the fibrous tissue of the turnip.
d. *f*. Vegetable hairs in collapse vomit: case recovered.
e. *h*. In reaction stools of same case.
g. In collapse stool: case fatal in eight hours.

Fig. 4. Crystals occurring in the urine, etc.
a. *m*. Uric acid in sediment of urine first passed (in reaction, on first day) after nitric acid and standing: cholerine: case recovered.
b. Uric acid in scum of same urine.
c. ,, in sediment of same urine: after heat, nitric acid and standing.
e. Dumb-bell oxalates in sediment of same urine.
d. Octohedral oxalates in sediment of urine passed in convalescence: cholerine: case recovered.
f. Uric acid in fever urine (coincident with appearance of cholera exanthem): case recovered.
g. Uric acid in same urine: after concentration and hydrochloric acid.
h. *i*. In collapse stools, after concentration to dryness, the formation of an alcoholic extract and then a watery solution, the addition of ammonia and standing: case recovered.
k. In urine of convalescence: after concentration and the addition of a saturated solution of oxalic acid: case same as *h*. *i*.

l. In collapse vomit after concentration and addition of ammonia: cholerine: case recovered.

Fig. 5. Forms of fatty and oily matters in vomit and stools.
a. Concrete fat, floating in pellets, in stools passed during consecutive fever: case fatal.
g. From last stool passed before death in same case (brought away by injection).
b. Spiculated fatty bodies in sediment of collapse vomit (on standing): case fatal in eighteen and a half hours.
c. From collapse vomit: cholerine: case recovered.
d. From contents of stomach removed at autopsy: case fatal in typhoid stage.
e. Fat-crystals in collapse vomit concentrated to dryness: case recovered.
f. In collapse vomit: case fatal in eight hours.

Fig. 6. Crystalline matters in vomit, stools, and urine.
a. *q*. *s*. Plumose and cruciform phosphates from urine passed in advanced fever stage: case recovered: urine concentrated by evaporation and ammonia added.
b. Stellate group of prismatic phosphates, from stools passed in beginning of consecutive fever: same case: body marked by a scarlatinoid exanthem: stools loam-coloured and somewhat feculent.
c. In urine passed during convalescence from consecutive fever: same case: urine concentrated by evaporation.

d. From urine passed during advanced fever: same case: after concentration, addition of ammonia and standing two days.
g. *o*. *u*. From urine passed during consecutive fever: same case: after boiling, addition of nitric acid, and standing two days.
l. *r*. From the first and second urine passed at beginning of consecutive fever: same case: after concentration by evaporation on sand bath.
e. *f*. Urates from collapse urine: case fatal in eight hours: after concentration and standing.
k. From fever (pea-soup) stools on standing: case recovered.
p. From urine passed during consecutive fever: same case: body marked by an exanthem: after concentration and addition of ammonia.
i. From urine passed at beginning of consecutive fever: case recovered: after concentration and addition of ammonia.
n. *t*. From urine passed during convalescence from consecutive fever: same case: after concentration and addition of aqua potassæ.
k. In collapse vomit after concentration nearly to dryness: case fatal in eight hours.
m. From sediment of urine passed during reaction: cholerine: case recovered.

Plate III.

Illustrative of the Histology of the Cholera Evacuations in the Dog.

Fig. 1. *a*. Mucus, in delicately striated bands, unassociated with granular corpuscles, entangling the parenchymatous cells of the potato, which are full of starch corpuscles. In the cholera stools of man the corpuscular element of mucus is generally present: in the vomit it is sometimes absent.
b. *c*. Flocculi of mucus, removed from the duodenum after death, boiled in weak acetic acid.
d. Flocculi of mucus from the contents of the large intestines, removed after death, boiled in water. The granular corpuscles and molecular matter were tinged of a greenish-yellow colour from biliary pigment.
e. Ordinary appearance of the mucus flocculi contained in the stools or in the intestines, showing the striated bands of mucus covered by or entangling granular corpuscles and molecular matter. This is also the most usual character of the rice flocculi in the collapse discharges in man.
f. The corpuscular element separated.

Fig. 2. *a*. Various forms of pavement epithelium from the vomit, all having a light greenish-yellow tinge from biliary colouring matter. The epithelial sediment of the cholera vomit in man generally consists of precisely similar elements, the scales being comparatively seldom perfect in size, form, or transparency, but frequently becoming elongated, navicular, fusiform, fibriform, and dark, hazy, or granular.
b. Granular corpuscles associated with the above and similarly tinged.
c. Action of acetic acid on them, showing the development of a delicate cell wall.
d. In mucus scraped from the gall bladder.
e. Hepatic epithelium, varying in darkness and granularity.
f. Cylindrical and pavement epithelium in mucus scraped from the stomach, very dark and granular.
g. Epithelium and granular corpuscles in mucus scraped from the inner surface of the urinary bladder.
k. Various forms of pavement epithelium in mucus scraped from the lining membrane of the urinary bladder in another dog. It is altered in a similar way to that contained in the vomit. Precisely the same appearances occur in the epithelial sediment of the first urines passed in the cholera of man.
The frequent haziness or granularity of the epithelium in different parts of

the body, and its infiltration with oily globules or granules, are equally common in the cholera of man.

i. In the mucous juice scraped from a section of the lungs.

Fig. 3. *a. b.* Animal hairs, probably accidentally swallowed by the animals, and occurring in the intestinal evacuations.

c. d. e. f. g. Various forms of isolated or partially disintegrated cells of vegetable tissues consumed as food by human cholera patients, and contained in their vomit and stools, especially the former. On these evacuations the dogs were fed. A diarrhœa was induced, during which the vegetable elements of their food frequently passed through the system of the animals almost unchanged. They include various forms of the so-called "annular bodies," "cholera fungi," or "corpuscles" of some authors, as is shown at *d*. *e*. Exhibits the forms which I have elsewhere denominated "gonidie." *f*. Were scraped from the surface of the duodenum, to which they were adherent. Some of these cells were generally to be found adherent to or entangled in the mucus coating of every part of the intestinal tract. The same bodies occur, but in greater abundance and variety, in the stools and intestinal coating in the human subject, not only in cholera, but in other diseases. Their contents possess various tints of yellow or green.

h. Collapsed and empty parenchymatous cells of the potato, of a light brownish colour. They become blue under the action of iodine.

i. k. Entozoon ova in various stages of development, aggregated or free; their colour consists of various shades of brown or brownish-green. In connection with their frequent occurrence in the intestinal evacuations, it may be mentioned that a tænia was found in the discharges of one dog and a lumbricus in the intestines of another.

Fig 4. *a. b. c. d. e.* Fragments of the parenchyma of various vegetables consumed as food, chiefly in the form of broth, and contained in the first vomits of human cholera patients. On these evacuations the animals had been fed, and the contained vegetable tissues had passed almost unaltered through their intestinal system. Most of the cells will be seen to be full of chlorophylle grains or starch corpuscles; at *e* they are empty.

f. Fragments of epidermic hairs of various vegetables generally used as food by man. Such hairs occur in ordinary wheaten bread.

g. Uncoiled spirals from the fibrous tissue of vegetables, also used as food by man, such as the turnip and carrot.

h. Isolated chlorophylle grains which have escaped by the rupture or disintegration of the parenchymatous cells above figured and described.

Fig. 5. *a. b. c. d.* Fragments of the epidermis of various vegetables generally used as food by man, chiefly the ingredients of broth; they possess various shades of green. Fragments like *a* occur in coarse bread.

e. f. Fragments of the parenchyma of similar vegetables, similarly coloured.

g. Starch corpuscles, entire, variously ruptured or otherwise altered, from the contents of the large intestines. They strike no blue colour on the addition of iodine, even with the aid of mineral acids. Hence the non-action of iodine cannot be considered a sufficient proof of the non-amylaceous nature of such corpuscles or cells. In other cases, the same corpuscles, and apparently under the same circumstances, exhibit the usual reaction of starch with iodine. I have noticed the same phenomenon in the cholera of man. When partially broken up, starch corpuscles probably include some of the forms of "annular bodies" already alluded to.

h. Starch corpuscles adherent to the mucous membrane of the intestines or intermixed with the mucus thereof.

i. Compound granular bodies of a deep greenish-yellow colour.

k. Isolated chlorophylle grains and molecular debris of a similar tinge.

The histological elements exhibited in Figures 3, 4, and 5, occurred in the

intestinal discharges and *post-mortem* contents in the cholera of the dog. The vegetable tissues, and their disintegrated cells, are the same as those occurring in the cholera stools of man. The animal hairs and entozoic ova are alone peculiar to the dog.

Fig. 6. *a. b. c. d.* Crystals contained in various specimens of the urine, when concentrated by evaporation.

e. In the semi-fæculent contents of the large intestines.

f. Ordinary prismatic phosphates appearing in the scum of the intestinal evacuations on standing for a short time. Along with and in similar circumstances to the phosphates, chloride of sodium occurred sparingly in broken tables.

g. Stellate groups of acicular phosphates occurring in similar circumstances.

h. Siliceous crystalline and granular matter, probably licked by the dogs from the floor of the room in which they were confined.

The crystallizable constituents of the urine were fewer in number, and presented less variety in crystalline form than those of the cholera urine in man.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

Dr E. Wilkes
With Author's compts.

CASE OF CHOLERA,

IN WHICH

THE BLOOD WAS REMARKABLY ALTERED.

By JAMES M. COWAN, M.D.,
EDINBURGH.

[*Reprinted from the Monthly Journal of Medical Science for March* 1854.]

Since the researches of Bennett and Virchow, on the structural alterations of the blood, every observation connected with that subject has acquired a degree of interest hitherto unknown. Hence the following case, though an isolated one, may not be undeserving attention :—

Mrs G., æt. 51, wife of a shoemaker, living in No. 48, Candlemaker Row, was admitted into the Edinburgh Cholera Hospital at 1.5 P.M. of the 8th of February 1854.

Immediately thereafter I found her pulseless at the wrist, complaining of cramps in the calves of both legs, recurring every four or five minutes, and when present, giving rise to great pain. She had also, almost incessant purging and vomiting. The stools presented the usual appearances of rice-water discharges, and the matter vomited seemed to contain some bile. The surface of the body generally felt cold, the skin doughy, the tongue and breath were also cold, the former being coated with a thick brown fur. She had a peculiar sunken expression ; countenance of a deep sallow tint; eyes much depressed in the orbits, and surrounded by a very pronounced dark areola. The voice was very weak, almost whispering.

At 10 o'clock P.M. of same day, the heat of surface is reported as good ; the pulse barely perceptible at the wrists ; occasional vomiting; excessive thirst ; purging almost incessant. She has made no water. It is doubtful when urine was last passed—according to her own account, not since the day of seizure, four days before admission.

Feb. 9*th*, 10 A.M.—Had slept during the night ; vomiting is abated ; purging continues much the same. Pulse small and thready, but quite perceptible ; temperature good; thirst not so urgent. 2 P.M.—Pulse evidently rising in strength. I drew off by catheter about 5ij. of turbid dark-coloured urine, which was found highly albuminous, and presented the same reaction on the addition of nitric acid, as Dr Parkes has pointed out in the urine of cholera. 11 P.M.—Pulse is described as of natural strength, 96 per minute. Within the last two and a half hours has had three stools, chiefly fluid of a dark green colour, and containing a very small proportion of solid flocculent matter. Other symptoms have disappeared.

Feb. 10*th*, 2.30 A.M.—I was sent for very hurriedly, being told a great change had almost instantaneously come over this patient. On arriving at her bedside, I found her face of a death-like paleness; respiration very slow and tranquil; pulse quite imperceptible, the pulsations of the heart even being scarcely recognised by the hand applied over the pericardial region. The extremities were cold; she had no vomiting. After some doses of diffusible stimulants and dry frictions steadily applied to the chest and extremities, she somewhat rallied, and at the end of an hour, heat having returned to the extremities, the pulse being quite perceptible at the wrists, and the expression of extreme debility partially gone, I left her. Another half hour had scarcely elapsed when the nurse returned saying, "the patient was dying." I again found the same alarming phenomena as before. In addition, there were now an occasional rattle in the chest, a glazed appearance of the cornæ, and the eye-balls were turned upwards. She expressed a fear of impending dissolution. Through the œsophagus tube (for swallowing could be effected but with great difficulty) I poured some brandy and beef-tea. It, however, had no effect in rousing the depressed energies of the system, and at 4 A.M. of the 10th she quietly sank.

Treatment.—This consisted of the hot-bath soon after admission, frictions with anodyne liniments, to allay cramp, and one of Dr Steven's saline powders every half hour; the first six were rejected, the rest were retained. She had also occasionally turpentine and saline enemata.

Preceding history.—The most of the following particulars I learned from her daughter. Mrs G. has had no children since her 28th year. During the last six years she has led a very dissipated life, being in the habit of spending the greater portion of her husband's wages in procuring whisky; she has enjoyed but very scanty fare. Her daughter described some kind of fits to which her mother, when very intoxicated, was subject; the description was rather vague, but somewhat analogous to that of epilepsy. She never had attacks of syncope. Her legs and ancles sometimes became swollen to such an extent as, to allow of the entrance of her feet, the shoes had to be cut. For the last eighteen months she has been subject to attacks of diarrhœa and nausea, being so affected at least twice or thrice every month. She never complained of palpitation at the heart. Eight days before admission into the hospital, she and her husband, both at the time suffering from diarrhœa, went down to Leith to attend a son-in-law, who, they were told, lay ill of cholera. They remained by their son-in-law's bed four days and nights, at the end of which time, convalescence appearing established, they came home to Edinburgh. Soon after their arrival the husband was seized with the symptoms of severe Asiatic cholera; and next day Mrs G. was attacked with cramps, vomiting, purging, etc. In the flat at Leith above the son-in-law's house, two men died of cholera the week previous to the illness of their relation. This is authenticated by a medical gentleman of Leith.

Sectio cadaveris—Nine hours after death.

Rigor mortis inconsiderable; peculiar sallow tinge of body; little posterior lividity; one and a half inches of fat in the abdominal parietes.

Head.—Considerable congestion of posterior portions of hemisphere of brain; considerable amount of sub-arachnoid effusion not coagulated.

Chest.—Pleuræ moist; lungs highly emphysematous, and very anæmic; both together weighed not more than 1 lb.; bronchi of both contained some frothy mucus; their lining membrane appeared of a rosy tint.

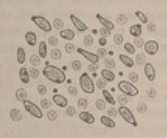
Pericardium contained a little fluid quite clear; one small spot of ecchymosis not exceeding the size of a pin's head was seen at the base of the right ventricle.

Right side of heart fully distended; left comparatively empty; cavities contained dark thick blood, semi-coagulated; some of the coagula were partially decolorized, and extended into the larger vessels; valves healthy; heart when emptied seemed small, especially when contrasted with the size and condition of the patient; its fibre was not fatty.

Abdomen.—Liver weighed 2 lb. 11 oz.; it was not at all congested, of a rather fawn colour, and of softer consistence than normally; gall bladder contained about two ounces of a thin green bile; spleen weighed 4½ oz.; it appeared of normal size, but felt softer than in a state of health; on section it presented a homogeneous mass, of a colour which can only be described as deep brown black, with a shade of vermilion (mahogany colour); not a trace of the healthy structure of the spleen; no malpighian bodies could be observed; stomach and intestines healthy; mucous membrane very pale, nowhere injected, except a small portion of the lower end of the descending colon (consequence of enemata).

Kidneys together weighed 9¼ oz.; right kidney was congested; external surface slightly irregular, presented portions of atrophied texture and a few small cysts; tubular character of cortical substance not very distinct; the left, on section, showed some cysts; consistence firm; otherwise healthy. Urinary bladder firmly contracted; mucous membrane injected and thrown into rugæ; external surface of cervix uteri showed a rough, irregular ulcer, of an oval shape, extending nearly all round the os; no thickening of the adjacent tissues; on pressure some gelatinous mucus escaped from the os; lining membrane of uterus, near fundus, injected; ovaries normal.

Microscopic Examination.—On examining a drop of the blood under a power of 240 linear diameters, the red corpuscles appeared very faint; seemed to contain little, if any, colouring matter, and did not present, as is their wont, the characteristic appearance "of rouleaux's of coin." The white corpuscles seemed to bear a normal proportion to the red. A few granules were seen here and there in the field of the microscope. In addition to these, however, were numerous other bodies, which could not fail to attract notice—generally circular in shape; some, however, oviform; a few caudate, and composed of a well defined membrane, not at all puckered, enclosing one or two distinct granules; these were very small, quite round in form, and possessed of clear centres; they appeared to be attached in general to one of the extremities of the circumference of the corpuscle; in some cases it was difficult to say whether they were adherent to its interior or exterior. When observed in motion some of these bodies appeared as if flattened on either side. They seemed to bear the proportion of one to seven or eight of the red blood corpuscles. The long diameter of the corpuscles measured about the 100th of a millimetre; their transverse 150th of a milimetre.



The corpuscles observed in the blood. The fainter bodies, which are the normal corpuscles partially dissolved, have been drawn by the wood-cutter rather too small—240 diam. lin.

On the addition of acetic acid they gradually swelled up, their external wall becoming fainter and fainter, until at last it appeared to rupture, and the included granules were set free. The acid, with the exception of rendering the granules more faint, had no other effect. A strong solution of muriate of soda diminished the size of the corpuscles, rendering them more distinct. On adding sq. potasse the corpuscles increased in size, their external wall becoming fainter. In the tissue of the liver the same bodies were seen, identical in appearance and behaviour under chemical reagents; the true hepatic cells were healthy. In the tissue of the heart, amid the muscular fibrillæ, similar bodies were observed. The same bodies, with the exception of a few true spleen corpuscles, were seen to constitute the whole mass of the spleen. In this instance they appeared rather larger in size than in the blood, and presented a much greater diversity of shape—the caudate being the most predominant form—at the extremity of whose tail-like projection, a nucleus could be observed as if pushing the cell wall before it.

Remarks.—It was observed at the time of the examination of the

body of this woman, "there was not a single morbid appearance which could be held as accounting for the cause of death." And it cannot fail to attract notice, that although we had all the most characteristic phenomena of true Asiatic cholera developed during the lifetime of the patient, we did not discover after death one of the usual morbid appearances, such as they are, met with in the bodies of individuals, the subjects of malignant cholera.

Various questions of great interest arise from the study of the case, which, I regret to say, are difficult of solution. In the first place, what led to these bodies? How is their presence to be explained?

Only three hypotheses are admissible—1st, This diseased state existed antecedent to the attack of the fatal disease; 2dly, It was the result of the morbific influence of the cholera itself; 3dly, It was consequent on the treatment employed. Now, it seems to me the first of these is the most probable explanation. This becomes apparent when we pass in review the preceding history of the patient—her delicate state of health, irregular habits of life, liability to diarrhœa and sickness; when we consider the peculiar appearance presented by the spleen, quite unusual in Asiatic cholera, and the mode of death, which also is comparatively rare, we can come to no other conclusion, but that these bodies, found in the blood and tissues, unconnected with the essential disease "cholera," nevertheless had a powerful influence on the disastrous issue of the case. The other hypotheses, I regret to say, from want of data, I can neither support nor refute. They form proper subjects of inquiry.

In the second place, what are these bodies? That they are, in some mode or another, connected with the blood is incontestible, from their being found in that fluid, and so universally amid the other tissues of the body, as can be explained on no other hypothesis. Are they, then, a modification of the red corpuscle? Are they an early or retrograde stage in the development or decay of the white blood corpuscle? Or have they any other relation to these bodies? I regret to say I have no answer to give these questions. All I can do in the meantime is merely to chronicle the fact of such bodies as described having been found in the blood and tissues of a woman who died presenting the true Asiatic type of cholera, and concurrent with a peculiar state of the spleen.

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ON THE

SO-CALLED FATTY DEGENERATION

OF THE

PLACENTA.

BY

JAMES M. COWAN, M.D.,

EDINBURGH.

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MDCCCLIV.

On the so-called Fatty Degeneration of the Placenta.

By James M. Cowan, M.D., Edinburgh.

The subject of fatty degeneration, ever since the microscope came to be extensively employed in pathological investigations, is one which has much engaged the attention of the morbid anatomist. We have had described, and no doubt very properly, fatty degeneration of the heart, kidneys, and liver; not to speak of those instances met with in the arteries, effused lymph, and extravasated blood, with which every pathologist must be familiar. At one time, indeed, there seemed to be a general tendency to refer every lesion displayed by the scalpel of the anatomist to the category of fatty degeneration—so much did this seem to be the case, that the phrase became a sort of by-word, and served as a synonym for every morbid condition that seemed mysterious, or was found difficult of explanation.

In the course of time, however, as a result of the conjoined labours of the microscopist and chemist, certain tests were discovered easy in their application, and, as some imagine, of unimpeachable accuracy, the mere application of which, if followed by certain effects, was held to determine whether the lesion under dispute should be regarded as an instance of fatty degeneration or not.

Thus the microscopist detected the highly light-refracting quality of the fatty granule, and its invariable simplicity of form and structure—characters, deemed by him almost sufficient of themselves to demonstrate its pathological significance. To these, however, the chemist added a third, which the former by way of confirmation almost habitually employs, although some—and it would seem with reason—have doubts as to its real importance and value. I of course allude to the solvent action of sulphuric ether on all bodies into

whose composition fat enters as an important element. It may be added that if the mere appearance presented to the naked eye be trusted to as a criterion of the existence of this lesion, it will, with very few exceptions, even in the hands of experienced pathologists, almost infallibly lead into error. Fat, as is well known, is met with in the human system, both as a normal and abnormal element. In the shape of adipose tissue it is extensively diffused throughout the body, and serves many most important purposes. This amount of natural fat must not, however, exceed certain limits—if it be excessive, or present itself in situations where normally comparatively little fatty tissue is to be found, then it becomes a diseased condition, known to pathologists under the title of "fatty deposition." Of this we have a striking example in the case of the heart. Again, certain of the tissues of the healthy body, as muscular fibre, sometimes undergo a peculiar transformation, in whose ultimate composition fat can be recognised both microscopically and chemically. Seeing this transformation is attended by certain morbid phenomena, it is not unreasonable to consider it as a degeneration; hence the phrase "fatty degeneration." Other distinctions exist between these, somewhat allied and often confounded conditions; thus the one can be detected by the naked eye, the other needs for its demonstration the assistance of the microscope—the one presents an arrangement of minute vesicles lodged in the meshes of areolar tissue, the other an aggregation of corpuscular forms, not individually defined. It is necessary to bear these distinctions in memory, for the two phrases have been used synonymously;[1] and as the placenta is an organ in whose intimate composition fat cannot be detected, even by the refined processes of chemical analysis,[2] we perceive at once it cannot be the subject of fatty deposition, provided the limitation just stated to that phrase be admitted as correct.

One other remark I wish to preface, which seems to be called for by the vagueness with which some pathologists express their ideas, and it is this,—that in applying the phrase "fatty degeneration," to a morbid condition of any organ, it is intended to represent that the true and essential component structures of that organ and not the product of any morbid action that may have been accidentally excited, as exudation or extravasation, have been transformed into fat. In saying the heart and bloodvessels are in such a state, it is usually understood the muscular fibrillæ in the one, and the true component tissues of the walls of the other, and not the blood or nerves supplied to those organs, have undergone such a degeneration. So, in speaking of "fatty degeneration of the placenta," it is to be presumed to signify, the villi, that is the true and essential structures of the organ, have undergone such a morbid process.

[1] See Report of London Medico-Chirurgical Society (*Lancet*, May 24, 1851.)
[2] According to Mr Hassall's statement.

It is with a view of precision in thought and expression, to avoid misunderstanding, I have entered into the above few explanations, and shall now proceed to consider the so-called fatty degeneration of the placenta, with the object of pointing out what seems to me to be its true pathology. Having had occasion two years ago to peruse a communication read by Dr Barnes to the London Medico-Chirurgical Society on this subject, my attention has been for some time applied to its investigation; and as, after a rather extensive examination of specimens, and careful inquiry into the general facts of each case, I have been led to a different conclusion from that arrived at by Dr Barnes,—and the subject seems invested with a great pathological, not to speak of therapeutic importance,—I venture to lay before the profession the results of my investigation.

In Dr Barnes' paper, as reported in the *Lancet*[1] (May 24, 1851), two cases of this lesion are given, with an account of the symptoms experienced by the mother during utero-gestation, and of the examination of the diseased placentæ after they had been expelled from the uterus. Dr Barnes then, in some comments upon the cases, goes on to state "that he regards the occurrence of this change of structure in the placenta as highly interesting, both to the pathologist and obstetric practitioner. . . . . The conversion of portions of placentæ into solid unyielding structure, and the consequent imperfect attachment of these portions and the surrounding healthy structure to the womb, give rise to hemorrhage, and premature labour may occur possibly during the life of the child." From this statement we are led to infer that Dr Barnes regards the essence of this lesion to consist in a degeneration of the proper tissues of the placenta; that is, of the villi into a fatty abnormal matter. What I wish to point out is, that the appearances presented by this morbid condition, and described by Dr Barnes, in most respects accurately, are capable of a totally different explanation; in short, it is not the villi of the placenta, but some of the constituents of blood which have become extravasated into its substance, that have undergone this so-called fatty degeneration; and

[1] This Paper is now published in the Society's Transactions, vol. xxxiv.

that, consequently, we have to do with the results of a hemorrhage into the placenta.

The facts upon which this opinion is based will be found to be derived from two distinct sources:—

1*st*, From the general and minute examination of the diseased structure; and,

2*d*, From a consideration of the causes of the lesion, and the symptoms experienced by the mother previous to the expulsion of the placenta from the uterus.

It may be here stated I have heard it objected to accepting such evidence as the narration of symptoms experienced by a patient, and the account of her general state as certified by a physician, in support of a pathological question. But I need hardly say, considering the intimate and interesting relation the placenta stands in to the female economy, it would be a most anomalous circumstance indeed in the history of medicine, to find any great disease of this organ without some corresponding constitutional derangement.

3*d*, After stating the facts under each of these divisions, I shall discuss the views that have been taken of the pathology of this lesion, and hope to shew that that of the fatty degeneration of the constituents of extravasated blood is the only explanation comprehensive enough to account for the whole phenomena, and as such is entitled to our support.

I.—*The general and minute examination of the diseased structure.*

In my own experience I have met with ten cases of this lesion. If to these be added the two reported by Dr Barnes, and one mentioned by Professor Kilian of Bonn, an abstract of which appeared in the *Medico-Chirurgical Review* for 1851, we collect together thirteen cases.[1]

Let us first examine a placenta which affords a well-marked and advanced specimen of this lesion. The first glance of it will suffice to shew two very distinguishing characters,—a diminution in size, as compared with the organ in a healthy state, and an excessively bloodless appearance. The former holds true both as regards the thickness and breadth of tissue. I have seen specimens which did not exceed in size the half of the normal placenta, and which were expelled from the uterus at the full term of utero-gestation. It not only, however, appears of diminished proportions, but

[1] Ten additional cases have been cited in a recent paper by Dr Barnes, in the 30th vol. of *London Medico-Chirurgical Society's Transactions*.

also much more compact than normally; in place of its spongy, loose, and soft appearance in health, it seems contracted and condensed in bulk, as if atrophied. When taken into the hand these characters are more distinctly brought out. In some points it feels very condensed and indurated, like a piece of boiled liver—to use the simile employed by Ruysch;—hence from the anatomists of the last century it received the appellation of "scirrhous." Its colour is yellowish-white; not a trace of the usual dark red, congested appearance of the healthy organ is visible. From this it is evident very little blood can have circulated or been contained within its vessels. Both the fœtal and maternal aspects of the organ present an unevenness of surface, and appear studded over with little projections, separated by sulci; some flattened, others more or less conical,—perhaps most distinctly so underneath the amnion; hence it has been described as "tuberculated." If the disease be principally situated between the chorion and decidua—for it is met with in one or both of two positions, either amid the true tissue of the placenta, or between the chorion and decidua—then the uterine surface will be comparatively smooth, for the blood coagulating has filled up the sulci. The characters of condensation and induration I consider owing to the extravasated blood having undergone coagulation, and the anæmic appearance to the effect of these coagula pressing upon the bloodvessels, and so obliterating the circulation. The degree of condensation and anæmia will be in proportion to the extent of the extravasation. I have seen it confined to one spot not exceeding one inch in diameter, and again so extensively diffused that it became impossible to find a remnant of structure presenting the usual colour and degree of consistence of the healthy tissue of the placenta. It is likewise to be anticipated, that the longer the placenta has remained within the uterus, after the occurrence of extravasation, the greater will be the degree of induration, for the farther advanced is the process of coagulation. Frequently, and most especially in those cases in which more than one extravasation has happened, we find the uterine surface covered with thin layers of coagulated blood, which can be easily separated the one from the other, like the coats of an onion. When a section is made through a thick deposit of these layers, the same appearance is presented as in a section of any aneurismal tumour in which some coagulation has taken place. These laminæ are found to extend even between the membranes and internal surface of the uterus, particularly in

those cases in which hemorrhage *per vaginam* has occurred during utero-gestation. It is found impossible to inject placentæ so affected; the bloodvessels are so compressed they cannot allow of the penetration of the matter of the injection, and on the employment of the slightest amount of force their walls speedily rupture.

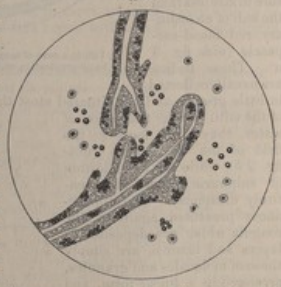
On making a vertical section through the diseased portion, a sensation is imparted to the hand as if the knife were being made to penetrate something hard and resisting like a fibrous tumour. On looking at the surface of the section with the naked eye, we perceive here and there spots of a yellow colour and granular in appearance, by scraping which gently with the point of the knife we disclose the villi of the placenta, very much condensed and altered in appearance, but still preserving their true form, and not at all broken up. In the neighbourhood of these spots we find little deposits of a much harder consistence, not very accurately defined in outline, of a pale yellow colour, and not at all granular, but smooth in aspect. On breaking up one of these masses, we find in the centre bodies presenting quite the appearance of those already described,—only the villi are in much fewer numbers, present their usual form, and appear even more compressed than in the former situation. Offshoots are sent from these deposits in all directions, surrounding and compressing the villi. In certain specimens we are enabled to trace the gradual steps of coagulation the blood has gone through from a recent fluid state, presenting its usual dark scarlet colour, up to the transformation into a yellow deposit of no inconsiderable consistence. In one part of a placenta we meet with a deposit of quite recent fluid blood; in another some of the colouring matter has been absorbed, but it is still soft and not much altered in appearance; in a third we find a kind of encysted clot consisting of a nucleus of yellow fibrine surrounded by clear serous fluid; while lastly—and this is the most advanced stage in the process—we find a hard yellow mass of semi-cartilaginous density. It is rare to meet with true encysted coagula; according to my experience they are seen in one situation only, that is, between the chorion and decidua: the clot had so pressed upon the villi as to form a little cup-shaped depression; in which it was deposited and lined by an apparently serous structure. In some cases of very partial occurrence of this lesion, we meet with small rounded masses imbedded in the substance of the organ, quite hard and isolated to touch, surrounded by healthy soft compressible tissues, which on section present no cyst-wall, but still shew a distinct and

pointed line of demarcation separating them from the rest of the placenta. The tissue of the organ is so lacerable and easy of distension, that the blood, when extravasated, finds no difficulty in insinuating itself amid the intra-villous spaces. This anatomical condition seems a sufficient explanation of the rarity of encysted coagula as a result of hemorrhage into the placenta.

Another appearance sometimes seen in placentæ affected with this lesion, and which occurs in connection with the other appearances already described, is that of a rounded melanotic mass, quite granular in section, and of the same consistence as the contents of a steatomatous tumour. We have twice seen this degeneration, never however very extensive. In the Edinburgh University Museum[1] is a preparation shewing little rounded masses of a melanotic aspect, scattered over the uterine surface of a placenta—no particular description however is given. Dr Simpson[2] regards this as a regular step in the process before the stage of yellow hepatization—if we may be permitted the expression—is reached; but from the rarity of its occurrence I am inclined to regard it as due to some special conditions not existing in every case.

The microscopic appearances presented by well-marked specimens of this lesion are very distinct and highly characteristic—indeed they are sufficiently diagnostic to distinguish it from all other diseased conditions to which the placenta is liable. Under a power of 240 linear diameters, the villi do not appear much altered from their normal condition. What has been remarked with the naked eye, in respect of the bloodlessness of the tissue is

Fig. 1.



Appearances presented by the villi and bloodvessels in a case of simple sanguineous extravasation.—(Case "Kelly.") Mag. 240 lin. diam.

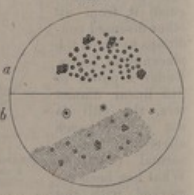
[1] *Vide* Catalogue of Museum, p. 256.
[2] Edinburgh Medical and Surgical Journal, 1836, vol. xlv.

now quite confirmed; scarcely a red blood-corpuscle can be distinguished within the walls of a vessel—the villi in the vicinity of the extravasation seem compressed, and do not present their usual extent of surface—with the exception of a slightly more defined, because thickened external wall, their organization shews nothing abnormal. Those villi which are found imbedded amid the coagulated blood do not only present a compressed appearance with thickening of the external wall, but are also covered with numerous rounded granules which strongly refract light and contain no nuclei (*vide* fig. 1). They measure in size less than the half of an ordinary red blood-corpuscle (*vide* fig. 3 *a*). These bodies are usually collected in little groups, and are situated most thickly near the bases of the villi. Under the action of water, they swim loose in the field of the microscope (*vide* fig. 2 *b*), while on the addition of sulphuric ether they entirely disappear. The appearances presented again by the coagula which are deposited in layers and laminæ, are simply those of molecules and granules, arranged in a linear form, and which present the same behaviour on the addition of chemical agents as those bodies already described (*vide* fig. 3 *b*). The dark melanotic masses present nothing under the microscope but a collection of

Fig. 2.

a. Villi in a case of sanguineous extravasation.
b. Same preparation when water has been added.

Fig. 3.



a. Appearance presented by coagula when situated amid the tissue of the placenta or sub-amniotic. (Case "Kelly.")
b. Same when in contact with uterus or surface of chorion.

amorphous granules, which on the addition of nitric acid are completely dissolved. In the specimens, again, of this lesion in which we can trace with the naked eye the changes the blood undergoes, and of which a delineation has been given by Cruveilhier,[1] the microscope in one part detects the ordinary red blood-corpuscle of its usual form and size; in another spot these become less numerous, and shew a more corrugated external wall, and are mixed up with other cellular bodies of an irregular form, containing neither nuclei nor colouring matter; in a third place, these last are seen occurring in connection with oleo-albuminous granules, which, in a fourth portion of the diseased structure, form the only object in the field of the microscope.

Such is the result of the general and minute examination of placentæ affected with this lesion. From it I would deduce the following conclusions:—

1*st*, That this is a morbid condition denoted by a peculiar condensation and consequent atrophy of the true placental tissue; 2*d*, By a degree of anæmia—characters explicable on mechanical grounds. 3*d*, Under the microscope certain bodies are seen which can be traced as transformations of certain of the constituents of extravasated blood, and which present a characteristic behaviour under the action of chemical tests,—oleo-albuminous granules, in short, which are met with in no other morbid state of this organ with which the pathologist is as yet familiar.

II.—*The causes of this diseased condition, and the symptoms observed during utero-gestation.*

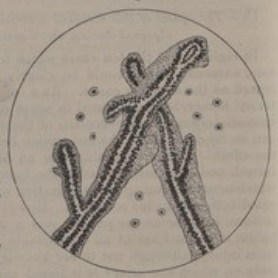
In eight out of the ten cases which fell under my observation, external injury could be clearly, with one exception, traced as the exciting cause. As a consequence of this, external hemorrhage happened in seven of these cases almost immediately upon the reception of the injury, so that there can be no doubt as to the connection between this cause and the effect. In the remaining one no direct injury could be traced, and it seemed probable the hemorrhage *per vaginam* which occurred during utero-gestation was due rather to some constitutional morbid condition. However it is matter of conjecture, because as the patient was a woman of abandoned habits it seemed not at all improbable that she did sustain such injury, although not to her knowledge. The part of the body on which the injury is inflicted may vary in each case. In four the injury was direct, that is to say, inflicted on the abdominal region. In three it was indirect, that is, received on other parts of the body not in immediate relation with the

[1] Anatomie Pathologique, tom i., liv. 16.

uterus. The effect of the injury is to produce a sudden and abrupt contraction of the uterine fibres, which causes a rupture of those vessels which form the bonds of connection between the uterus and placenta; the hemorrhage of course will be in proportion to the extent of the laceration. If this explanation be admitted we at once get rid of the difficulty Dr Simpson experienced in assigning the particular source of hemorrhage in what Cruvelhier has called "apoplexy of the placenta." Such external injury, if this be its mode of operation, can have no effect on the umbilical vessels.[1] In the normal contractions of the uterus the pains gradually increase in intensity up to a certain point, at which they attain a maximum, and from which they gradually decline. I apprehend it is in consequence of this arrangement that in natural labour laceration of the utero-placental vessels takes place so rarely. That such an occurrence does sometimes however occur is undoubted. In the North of England Medical and Surgical Journal is reported a case in which a woman died suddenly during parturition, and on the *post-mortem* examination a coagulum of blood was found between the placenta and the fundus of the uterus, which weighed 18 oz.

In the remaining two cases of the ten the lesion seemed to owe its existence to a condition of the bloodvessels ramifying in the villi of the placenta, which has never yet been, so far as I know, described, and which is identical in appearance with that observed in the vessels of the pia mater in some cases of apoplectic extravasation. The bloodvessels were completely and equally coated over all their extent with oleo-albuminous granules (*vide* figs. 4 and 5). That these granules were part and parcel of the walls of the vessels, and did not result from any exudation exterior to the villi, is proved by their remaining unaffected on the addition of water, and not dissolving when sulphuric ether was added. In both cases the patients suffered previous miscarriages, the one two, the other three. The exact appearance presented by the placenta on each of these occasions could not for obvious reasons be ascertained. Most probably, however, as the patients were of a leucophlegmatic habit of body, the cause of abortion was the same in each particular pregnancy. In neither did external hemorrhage occur previous to labour, and in both the extravasation was limited, and in some parts quite recent.

Fig. 4.



Appearance of simple fatty degeneration of the vessels of the villi.—(Case "Henderson.")

[1] M. Murat, in the *Dict. de Médecine*, states, laceration of the placenta may be produced by external violence or shocks communicated to the system of the mother, and of this lesion hemorrhage is a frequent occurrence.

Fig. 5.

Appearance of villi in a specimen of both lesions of sanguineous extravasation and fatty degeneration.—(Case "Wilkinson.") Mag. 200 lin. diam.

In addition to an exciting cause, there must be also some pre-disposition, for it would be too much to say every woman who sustained an injury during pregnancy would suffer from this lesion. It is very liable to occur in women who have previously aborted,—like the fœtus, the placenta has a strong tendency to the perpetuation of a particular type. In four of the cases that came under my observation the patients had miscarried more than once previously; and, it is to be presumed, from the same cause,—although from the circumstance of dispensary patients being rarely attended in two accouchements by the same medical man, and not unfrequently by women only,—it becomes impossible to ascertain this point with any degree of accuracy. Dr Lee of London mentioned, in the course of the debate which followed the reading of Dr Barnes' paper, that he had attended a lady who sustained eight abortions in succession, the pathological cause of which, according to his statement, was, "a change in the villi of the chorion, which had the effect of obliterating the circulation." Schulzius met with a woman who had twenty successive miscarriages—the state of the placenta he does not particularly mention. Dr Williams thinks an altered state of the blood, or some defect in nutrition, may have some in-

fluence in producing fatty degeneration of the placenta. This lesion, again, Dr Quain is inclined to regard as having its origin in some modification of the nutritive and circulatory apparatus of the organ by which the albuminous and fibrinous elements of the blood are retained within the vessels, or extravasated, and then subjected to the process referred to.[1] It may be stated, in a general way, all circumstances tending to weaken the vascular connection between the uterus and placenta, whether these be constitutional or local, must have an influence on the production of this lesion.

External hemorrhage, or the discharge of blood *per vaginam*, was noted as a symptom in six out of the seven cases in which injury was received. In the remaining case, in which external hemorrhage did not occur previous to labour, the disease was very partial, and confined to the centre of the placenta,—it did not exceed an inch in diameter. The patient remembered distinctly having received a blow upon the abdomen from a man during the sixth month of utero-gestation, after which she felt sick and languid, and was seized with rigors; in the course of two or three days these entirely disappeared, and she carried her child to the full term, when it was born alive, though rather emaciated, and of smaller size than normally. It is only in certain circumstances we can look for the presence of this symptom—only when the seat of laceration of the vessels is situated at the circumference of the placenta, or the extent of rupture so great as nearly to dissever the whole organ from the interior of the uterus. The hemorrhage may occur only once after the reception of the injury, or, as was observed in two cases, it may occur every month during the remainder of utero-gestation. It is curious to remark, that the hemorrhage in these cases occurred at periods corresponding exactly to the ordinary monthly molimina,—to the time at which the patient, had she not been pregnant, would have expected to menstruate. Perhaps the matter extravasated acted as an excitor of reflex action on the interior of the uterus, and that the uterus was thrown into action at those periods at which normally it is in its most irritable state. One of these patients, in whom repeated hemorrhages occurred, was a strong healthy woman æt. forty-five, who was confined of her fifth child, and never had a previous miscarriage. At the fifth month of pregnancy following a blow upon the belly she perceived a red discharge *per vaginam*, somewhat abundant, on which she applied to a medical man, who ordered rest and cooling drinks, which had the

[1] *Vide* Report of Meeting of London Medico-Chirurgical Society (*op. cit.*)

effect of checking it. Soon it became diminished in quantity, and at last disappeared. She saw no more of it for four weeks, when it again appeared at the time at which, in her unimpregnated state, she would have expected the catamenial discharge. It lasted on this occasion five days, and again gradually disappeared. She did not now consult any medical man, not feeling alarmed about it, although she was affected for some time with nausea, loss of appetite, and rigors. Twice more the same phenomenon presented itself at an interval of three weeks and a few days, until on the ninth month she was delivered of a male putrid child. The other case was that of a woman in her third pregnancy, who never had any miscarriage, and who appeared free of any constitutional taint. At the fourth month of utero-gestation, consequent on a hurt she received on the left haunch-bone, she sustained a hemorrhage *per vaginam*. Labour supervened at the end of the seventh month; and on three different occasions, corresponding exactly to her ordinary menstrual molimina, the same discharge returned in no very abundant quantity. In both of these cases, the placentæ presented characteristic specimens of this morbid condition; in both they appeared to bear a normal proportion to the size of the fœtus,—they felt hard and indurated. Overlapping the surface of the chorion in one, and penetrating down between the intra-cotyledonous spaces, in some places even to the amnion, was found a compact, tough, membranous substance, of greatest thickness in the centre, and becoming gradually thinner as we approached the circumference; it presented a yellowish-white colour, and on section was found to consist of layers which could be easily separated the one from the other, thus shewing it to be composed of successive deposits of coagulated blood.

The general symptoms manifested after the receipt of the injury were very similar in all of the cases,—for the most part the patients complained of pain in the abdomen and back, nausea, anorexia, tendency to fainting, dizziness in the head and coldness of the extremities, and rigors. From the presence of this last symptom, some have inferred the lesion to be of inflammatory origin. No man at all conversant with uterine disorders need be told in how many instances shivering is a prominent symptom, although not the slightest trace of inflammation can be detected. The sympathy existing between the uterus and the general female economy is so intense, that the slightest disturbance of the former gives rise to a severe commotion of the latter.

According to Dr Barnes, in one case, flooding occurred

twice without obvious cause, and unaccompanied by pain, at the third month, and again at the seventh, when labour followed. In the second case there was no hemorrhage previous to delivery. In neither is any idea given as to the relative extent of the disease; in both death of the child had occurred some time before delivery, from which it is to be presumed the extravasation must have been considerable in amount.

External injury, then, seems the great exciting cause of this lesion, which, when produced, if at all extensive, must inevitably lead to the death of the child.[1] Indeed, from my own experience—which is perhaps as yet rather too limited—and from what I have heard in conversation with older practitioners, I am inclined to believe—and beg to draw the attention of obstetricians to the point—that it is extravasation of blood into the placenta, arising from rupture of the utero-placental vessels, and leading to the death of the fœtus, and not "inflammation of the womb"—or of the placenta" or "rheumatic stitches of the uterine parietes," or even premature parturition itself, that is most to be dreaded from a woman considerably advanced in pregnancy receiving any bodily injury, especially in the lower parts of the body. The practical precept I should draw from this fact is, the necessity, on being summoned to such a case of careful auscultation, and watching the state of the fœtal heart; if pregnancy should have reached the seventh or eighth month, and the fœtal pulse tends to become slower and more feeble, the obligation the medical man is laid under of inducing premature labour, with the view of saving the life of the fœtus in utero. I am quite convinced many lives might be saved by practice based on such principles. Indeed, I had occasion myself, when a medical student, to witness such an occurrence happening before my eyes. It was the case of the wife of a gamekeeper in a distant part of the country, who, in the eighth month of pregnancy received accidentally a rather forcible blow upon the abdomen. Immediately thereafter she felt squeamish and sick, had rigors, slight pains in the back, not very severe nor regular in their time of accession, and was obliged to lie down in bed. I saw her in the course of an hour after the accident. I found her with a feeble pulse, cold surface, and tendency to syncope. On examination *per vaginam* there was no hemorrhage, the os uteri was occluded, the vagina was cool and moist. I then proceeded to auscultate the abdominal tumour, and found the "placental souffle"

[1] It may be remarked, causes which might operate quite unobserved by the patient herself, are sufficient to produce a sudden contraction of the uterus and consequent severance of the utero-placental vessels.

very limited, but quite distinct over a portion of the uterus, midway between the umbilicus and the right anterior superior spinous process of the ilium. After a good deal of attentive examination I heard the pulsations of the fœtal heart; they were faint and feeble in character, and numbered 100 per minute. The patient told me most distinctly that for several days previous to the accident she was quite conscious of violent intra-abdominal movements, and that since the accident she had felt no motion at all. Not possessing any legal qualification, and being rather timid of inducing premature labour on my own responsibility, without the concurrence of another medical man, I thought the more prudent course of treatment, especially as it was the patient's first pregnancy, was to adopt "la médecine expectante," and use those measures which I considered the best calculated to check the extravasation which I presumed was taking place into the substance of the placenta. Next day, nothing remarkable having occurred in the interval, after the most attentive and searching examination over every point of the uterine tumour, and in all kinds of position of the patient, I failed in detecting the pulses of the fœtal heart, not only at the place where I formerly had heard them, and which was marked with ink, but at nowhere else. A week afterwards, having in the meantime felt no movement in her abdomen, she began to complain of a strange sensation of something rolling in her belly whenever she moved in bed; she was seized with loss of appetite, depression of spirits, and vertigo; the mammæ, formerly large and well developed, became small and flaccid; in short, it became evident death of the fœtus in utero had occurred. At the end of six weeks the uterus threw itself into action, and expelled a dead male child, quite putrid, in all other respects well developed. The placenta presented quite the appearance I anticipated—the disease was very extensive, and principally confined to the central portions of the organ. I remain to this hour convinced that that woman might have been delivered of a living and viable child, had I had the courage to act on the principles I have been attempting to enunciate.

The mode in which this lesion proves fatal need not be a matter of conjecture. By obstructing, and in some cases completely preventing, the circulation of the blood through the placenta, it must lead to one or both of the following results, as regards the fœtus. It must interfere with the due oxygenation of the fœtal blood, thus tending to produce death by asphyxia—and this undoubtedly happens in cases of sudden and extensive extravasation; or by interfering with

the proper assimilative function of the placenta, of abstracting from the maternal blood substances necessary to the maintenance of nutrition and growth in the fœtus, will likely prove fatal by inanition: this will explain the great degree of emaciation presented by the fœtus in some instances of partial extravasation. Indeed it is matter of surprise how very small a portion of healthy placenta seems sufficient for the maintenance of life in, and carrying on of the processes of respiration and nutrition in the fœtus. A case happened in M. Dubois' ward, in Paris, in which a placenta fully two-thirds diseased was expelled from a woman, having been preceded by the birth of a child at the full time, perfectly well developed, and not a whit emaciated. That, and such cases, are doubtless exceptional; and I cannot see how such a lesion, if at all extensive, can exist with no prejudicial effect on the condition of the child. As regards the mother too, this morbid condition is not altogether devoid of danger. If coagulation does not take place, and a portion of the placenta remain adherent (supposing the present doctrines respecting the source of hemorrhage in placenta prævia to be correct), there is a risk of the mother dying from loss of blood. It may be sometimes fortunate for the mother, if coagulation does take place; although, as respects the child, it must always be attended with disastrous consequences. Might not compression exercised over the spot where the placental murmur is heard, be attended with advantage in such cases?

III.—*The pathological anatomy of the so-called fatty degeneration of the placenta.*

Two views in particular have been taken of the pathology of this lesion; the one regarding it as a type of inflammation, the other as a true fatty deposition in, or degeneration of, the tissue proper of the placenta. The former was that maintained by the anatomists of the last century—thus, Ruysch has described, and caused to be delineated a placenta, the whole of whose fœtal surface is studded with numerous "tubercles" of a consistence so great as to deserve the epithet[1] of "scirrhous." He considers it the consequence of inflammatory action; on section, he adds, the tubercles displayed a homogeneous appearance, and presented no trace of organization. Wrisberg has described a case in which he found the uterine surface of a placenta covered with a layer of membrane 2½ lines thick in the intralobular spaces, and from 1½ to 2 lines

[1] From this hardened or hepatized state of the placenta, the distinguished pathologist Rokitansky has been led to regard this lesion as a result of placentitis.—(*Patholog. Anatomy*, vol. i., p. 142—Sydenham Society's edition.)

in thickness where it overlapped the lobes. This membrane was white in colour, and of a hard and compact consistence. Its structure was cellular and nearly tendinous. To inflammation he imputes its origin. It may be added, the placenta was non-adherent to the uterus. Albrechtus writes "he has seen small tumours scattered throughout the substance of the placenta, of a whitish colour, and closely resembling scirrhous glands. And Mauriceau, in his book on Midwifery, gives several cases of placentæ he calls "scirrhous," or "affected with chronic inflammation."

As to the signification to be attached to the term "*scirrhous*," as used by these authors in their description of morbid conditions of the placenta, it may be stated that, primitively and etymologically, nothing is intended to be implied by the term, beyond a certain physical quality, viz. hardness. It was only as pathological science advanced, hardness being found a constant and most striking property of certain morbid growths which were coincident with a cachectic state of the system, and shewed a peculiar tendency to invade all neighbouring tissues, that "scirrhous" came to be applied to and associated in every person's mind with a growth of a *mali moris* or malignant character. Now-a-days, when one speaks of a scirrhous tumour, he usually means cancer. Formerly every tumour, provided it was possessed of a certain degree of hardness, received the same appellation. This opinion seems to derive some support from the experience of the present day, for we know that if there is any organ of the body more than another exempt from malignant disease, it is the placenta. I believe it possible to affirm, without the slightest risk of error, that cancer of the placenta is a lesion never met with, at all events so rare as to be unworthy of consideration. In Dupuytren's Museum at Paris, there is a preparation labelled "cancerous tumour of the placenta." No account is given of any examination this tumour has been submitted to, and to me I confess it seemed nothing else but a considerably hypertrophied placenta deprived of its blood by long maceration in spirits.

Pathologists of more modern times, and some even of the present day, have attempted to defend the inflammatory view of this lesion on the grounds of the microscopic appearances; the oléo-albuminous granules they regard as arising from an exudation of liquor sanguinis from the interior of the bloodvessels; these bodies in short they consider as diagnostic of an inflammatory lesion. I hope to shew immediately this opinion is based upon very limited pathological observation, and that these same granules, in place of being met

with in states of inflammation only, are met with so frequently, it becomes a difficult task to enumerate the morbid conditions in which they do not occur.

That this lesion is not of an inflammatory character may be proved first of all by the general symptoms. Now shivering frequently occurs in the course of this disease, and some have grasped at this as indicative of inflammation. But rigors alone, in the absence of other febrile symptoms, such as pain in the belly, hot skin, bounding pulse, great thirst, &c., cannot be held as proving unconditionally a state of inflammation. In these circumstances they prove merely a derangement of the nervous system. By way of illustration let me instance the case of rigors occurring at the end of the first stage of natural labour. Who has seen anything of obstetric practice, and has not witnessed such a phenomenon; nay, perhaps frequently, and to a degree sufficiently intense? Yet who, in the absence of other febrile symptoms, ever thought shivering in these circumstances symptomatic of a type of metritis, or an indication for the use of the lancet. So in this case of disease of the placenta the patient no doubt shivers, but this is, as I have already stated, dependent on the intense sympathy which exists between the uterine and general system of the woman, and is caused by the irritation of the extravasated matter on the interior of the uterus. She presents, in addition, coldness of the surface of the body in place of heat; a depressed and weak, in place of a bounding pulse; thirst no doubt sometimes; and no pain on pressure over the seat of the placenta. In the face of such symptoms, who would argue the case was one of inflammation, acute or chronic, or be prepared to adopt the usual antiphlogistic treatment? The circumstance which militates most against the inflammatory theory of this lesion, is to be found in the fact, that a placenta so affected is never found preternaturally adherent to the interior of the uterus. It is contrary to all pathologists of the present day know regarding the results of the inflammatory process, to conceive it possible that such layers of coagulable lymph as are sometimes seen deposited between the uterus and placenta, could exist there without giving rise to the same results, as is generally seen when lymph is effused between two solid substances. In the ten cases which happened in my experience, not only was the placenta non-adherent, but it seemed to be unusually less adherent than commonly, for in several of the cases the whole of the contents of the uterus were expelled simultaneously. Another anomaly exists, which is difficult to be accounted for, if the inflammatory theory be accepted; it is this,—why if the most common and almost essential microscopic elements of an inflammation be recognisable, should these cells never attain any higher organization? why, in fact, should pus or abscesses be never met with in such conditions? It cannot be answered,—time is not allowed—for in many of the cases, months positively elapsed between the occurrence of the extravasation, and the birth of the placenta. In the analagous case of the bloodvessels of the pia mater, it has been explained by some pathologists, by saying there is no mucous membrane in their vicinity. But this reasoning cannot be applied to the uterus, for there they are in immediate proximity to mucous membrane in the shape of deciduæ. Another set of arguments in favour of this explanation has been attempted to be drawn from the microscopic examination. Peculiar bodies, argue these pathologists, have been detected by the microscope as composing the essential characters of this disease; these granules are identical in appearance and chemical properties with those seen in ordinary states of inflammation,—hence this lesion must be also inflammatory. This reasoning, however, is inconclusive, in as much as it is based upon a too limited generalization. While we admit, because we cannot deny, that bodies, so far as appearances go, identical with those met in this diseased condition, are to be found in the matter of abscesses, or in the exudation poured into the air-cells of a hepatized lung, we must not forget to notice, that precisely similar corpuscles are to be met with in many other morbid states of the body, which no one now-a-days would consider in the remotest degree inflammatory. They are to be found in great abundance as composing certain forms of cancer, viz., the encephaloid, in certain diseases of the liver and kidneys presenting not the least trace of inflammation; in certain softenings, such as that of muscular fibre in contact with pus, or of bone, or of the brain, nothing is more common than such microscopic appearances. If such be the case,—if such corpuscles are to be found so generally in diseased states of the human system, they must have some other value; their presence must be indicative of something else than any individual lesion, or particular species of morbid action. Any conclusion, therefore, to be deduced from their existence in favour of inflammation becomes equivocal. Now, to my mind I confess, I consider any conclusions to be derived from the presence of such granules as of trivial importance, except as a kind of confirmatory proof of an otherwise tolerably distinct morbid condition. Isolated *per se*, and not contained within other normal structures, it can-

not be held they determine the specificity of any lesion whatever. In the case of pneumonia, it cannot be said, oleo-albuminous granules are characteristic of the disease; for not only are they, but molecules and granules, epithelial scales, and true pus-corpuscles, highly organized cells, found in the matter poured into the air vesicles; the former are merely an early stage in the development of the elements composing the matter exuded. It is of little moment to our present purpose to inquire—besides, it is a disputed question—whether or not the true pus-corpuscle is really formed from these granules; this, however, is certain, the former are more highly organized than the latter. Again, in some soft cancers, they are found in great abundance. What shall we conclude from this fact? that soft cancer is an inflammatory lesion! It is impossible; for, in addition to these bodies, and in as great abundance, are seen cells highly organized, possessing one, two, or three nuclei, and these again often nucleoli. Besides, the general symptoms will not permit it. Shall we not rather conclude that the former are the source, or rather the remains of the latter, for it is in the softest forms that these fat-corpuscles are the most numerous;—they seem to me to be the lowest type of organization, the first in the series of cell growth, the penultimate in their decay. In the case of the placenta, that these granules do result from the softening of an exudation external to the villi is proved by their being loosely adherent to their walls, not incorporated in their true structure; easily acted on by water, which causes them to swim loosely in the field of the microscope; and, being easily dissolved on the addition of sulphuric ether,—a striking difference of action we have seen in those cases in which the oleo-albuminous granules were deposited around the bloodvessels within the villi, and therefore removed from the action of water and sulphuric ether. In this last case, it has been disputed whether the corpuscles are owing to an inflammatory exudation, or to a fatty degeneration of the true tissues of the walls of the bloodvessels. In the analogous case of the disease in the bloodvessels of the *pia mater*, in connection with cerebral softening, Mr Paget[1] maintains the lesion in that case consists of a true morbid condition of the proper tissue of the vessels, while Dr Bennett[2] has endeavoured to prove, it is in consequence of an exudation of *liquor sanguinis*, and is therefore inflammatory. I need hardly add, the same general arguments which have been stated already, apply with equal force against the inflammatory view of this condition. I have not been able, in those two instances which fell under my observation, to satisfy myself that the layer of granules was deposited underneath an external membrane, as Mr Paget has pointed out in the vessels of the brain. The regular and equal distribution of the granules over the exterior of the vessels; the non-coincidence with any general inflammatory symptoms; their occurrence in patients of a certain cachectic constitution, in whom it is reasonable to suppose there existed some modification of the nutritive process; their continuance in a low stage of development, with the fact of fibrine occurring as a constituent of the coats of the vessels,—all such circumstances go in support of the view of fatty degeneration of the coats of the vessels.

[1] Lectures on Surgical Pathology, vol. i., p. 142.
[2] Edin. Med. and Surg. Journal, 1843, vol. lix., p. 344.

That this morbid condition of the placenta is not an instance of fatty deposition,[1] I consider demonstrated already by what has been said in the preface to this paper regarding what is to be understood by the phrase "fatty deposition."

1*st*, The terms imply a certain amount of fat to be a normal constituent of the organ. True fat cannot be detected, even by chemical analysis in almost inappreciable quantity, in the tissue of the placenta; the first condition, then, for fatty deposition is awanting.

2*d*, The terms imply a deposit of true adipose tissue. In the placenta true adipose cells have never been seen under the microscope, either in its normal or abnormal state.

The only other view of the pathology of this lesion which remains is that of fatty degeneration, and this is divisible into two branches, according to the supposed material which undergoes degeneration; some regarding it as the tissue proper of the placenta, others, a material foreign to its normal constitution.

The facts opposed to the former view are the following:—

1*st*, The villi, even in the most exaggerated forms of the disease, present their natural configuration; they never appear broken up nor disintegrated; occasionally, they are more compressed, and present a smaller extent of surface than normally, but this is quite explicable on mechanical grounds.

2*d*, The placenta in these cases feels hard and condensed. If such were the real explanation of the phenomenon, it would be natural to expect the tissue of a softer consistence.

3*d*, The fat globules are situated on the surface of the villi, and do not form part and parcel of their structure. Besides

[1] Dr Quain, in his paper on "Fatty Heart," in the London Medico-Chirurg. Society's Transactions, restricts what is implied by the phrase fatty deposition in the same manner. (January 1851.)

these, there are certain characters presented by the placenta so affected,—such as an atrophied and shrunken state of the organ, with an appearance of anæmia,—which are capable of explanation only on the hypothesis of a foreign matter having been extravasated amid the tissue of the organ, and undergone a process of condensation.

It is to this view of the pathology of this lesion we are therefore driven, as the only one comprehensive enough to include all the phenomena. I have said already amply sufficient to shew the facts on which this hypothesis is grounded. It would be a mere repetition stating these reasons in the present place. It will suffice to say, that this lesion presents an instance of a degeneration of the fibrine of the blood, which Mr Gulliver[1] has found to take place in certain conditions external to the body, as in the softening of fibrinous clots, and in the formation of "adipocere" from the fibrine composing muscles, and which several pathologists have witnessed in cases of pulmonary and cerebral apoplexy. Viewed in this light, it cannot be regarded as a new morbid process, but merely a diseased condition found in a previously unknown locality.

[1] London Medico-Chirurg. Society's Transactions, vol. xxii.—Mr Gulliver considers a low state of the vital powers as favourable to the formation and softening of coagula.

ON

THE PATHOLOGY

OF

DELIRIUM TREMENS;

AND ITS

TREATMENT WITHOUT STIMULANTS OR OPIATES.

BY

ALEXANDER PEDDIE, M.D., F.R.C.P., ETC.

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PREFACE.

At the suggestion of several professional friends, I have been induced to reprint and publish the following paper, which lately appeared in the *Monthly Journal of Medical Science;* but, in order further to illustrate the opinions therein advanced, three additional cases of Delirium Tremens are now detailed: and I have introduced also the result of inquiries made at the Medical Officers of some of our Prison Establishments, in regard to their experience of the effects produced by the sudden withdrawal of wonted stimulants, in the case of civil and criminal prisoners, of known intemperate habits.

The only other additions of any importance which I have now made are references to the opinions given by Dr Craigie, in his very able Dissertation on Delirium Tremens (Methystic Brain Fever, Practice of Physic, vol. ii., p. 50, etc.); and I take this opportunity of expressing regret that I had not seen that paper previous to the publication of my own views, both on account of the similarity of opinions held on some points, and the very full and learned notice given by him of the literature of the disease. It will be seen, however, by any one acquainted with his observations, that we differ materially on many points connected with the phenomena, pathology, and treatment of the disease; and that what he has merely hinted at in regard to the affection, as being attributable partly to the cerebral vessels loaded with imperfectly aerated, spirit-charged, or alcoholized blood (pp. 77 and 81), I have more fully stated, and endeavoured to explain and illustrate, as a peculiar toxicological result.

A. PEDDIE.

Edinburgh, 15, Rutland Street,
August 1854.

ON DELIRIUM TREMENS.

Before attempting to explain the following views of the pathology and treatment of delirium tremens, I think it necessary to state what are considered to be the symptoms of the disease in its genuine and uncomplicated form; for it appears to me that the opinions generally received and acted on in regard to this affection are erroneous, and have resulted from a loose and partial observation of its phenomena, and inattention to the history of individual cases. In consequence of this, the disease is frequently confounded with very dissimilar affections, or its usual features lost sight of under the effects of injurious treatment, and its true nature, therefore, misunderstood.

The most characteristic symptoms of delirium tremens are general muscular tremors—more especially of the hands, and of the tongue when protruded—along with complete sleeplessness, and delirium of a muttering, sight-seeing, bustling, abrupt, anxious, apprehensive kind. The affected has not ability to follow out a train of thought, to explain fully an illusion or perverted sensation, or to perform any act correctly; because he may be one moment rational and the next incoherent; now conscious of his real condition and of surrounding realities, and then again suddenly excited by the most ridiculous fancies—principally of a spectral kind—such as strange visitors in the shape of human beings, devils, cats, rats, snakes, etc.; or by alarming occurrences, such as robberies, fires, pursuit for crimes, and the like. He is easily pleased and satisfied by gentleness and indulgence, and much fretted and agitated by restraint and opposition. The face is generally of a pale dirty colour, and wearing an anxious expression; eyes startled but lustreless, sometimes considerably suf-

fused, and the pupils not contracted unless considerable doses of opium have been administered, or very decided arachnitic symptoms have supervened; skin warm and moist, often perspiring copiously; tongue sometimes loaded but generally pale and moist, occasionally remarkably clean; appetite small, but the patient will often take whatever is presented to him; thirst by no means urgent, and seldom or never any craving for spirituous liquors; urine scanty and high coloured, and in some cases which I have tested, containing a large quantity of albumen, which, however, disappears immediately after the paroxysm is over;[1] alvine evacuations bilious and offensive; and the pulse usually ranges from 90 to 120, generally soft, but of various degrees of fulness and smallness, according to the strength of the patient and the stage of the affection. The precursory symptoms are by no means peculiar or pathognomonic, but common to many febrile affections implicating the sensorium in the way of sleepless and restless nights, with, perhaps, more of a hurried and agitated manner than usual for some days previously. The paroxysm, which is distinguished by the phenomena above described—occurring with remarkable uniformity, independently of age and constitution,[2]—usually runs its course, if uncomplicated and properly treated, on the second or third day, though sometimes earlier, and it seldom extends beyond the fifth day. It then terminates in a profound natural sleep, which may continue for many hours, and from which, if it even lasts for six hours, the patient awakes quite coherent, although weak and languid, but from which state, considering the severity of the symptoms, he is restored with singular rapidity to physical strength and mental soundness. The casualties of the disease are convulsions or coma, which, if not immediately fatal, are apt to leave the unfortunate sufferer a wreck for the remainder of life.

[1] Probably albumen is always present. In the thirtieth volume of the Transactions of the Medico-Chirurgical Society of London, Dr Bence Jones has endeavoured to prove, from three cases of this disease, compared with three of phrenitis, that the phosphates are found in much smaller proportion in the urine of the former than in the latter, and that thus a new diagnostic mark is presented of the nervous and non-inflammatory nature of delirium tremens. His observations, however, are not sufficiently numerous to be conclusive, and there are many probable sources of error. In the only two instances (Cases VII. and IX.), in which I have examined the urine, there were present in the one a great many, and in the other a considerable number of beautiful phosphatic stellæ; and I have no doubt that these will often be found in abundance in the most genuine cases of the affection.

[2] The age at which delirium tremens most commonly occurs, seems to be between 35 and 50; the constitution is the irritable, excitable, and somewhat weakly; and what is interesting, it is very uncommon in the female, although from what cause I cannot explain, for the number affected does not bear any relative proportion to the number of dram-drinkers. I have seen several cases of the delirium ebriosum, but no instance of delirium tremens in the female sex. Rayer observed the disease in 7 women out of 176 cases; Bang in 10 out of 456 cases; and Dr Hoegh-Guldberg of Copenhagen (*Commentatio de Delirio Tremente*), in 1 out of 173 cases. Dr Gibson of Glasgow, however, informs me that out of 57 cases treated in the prison of that city during 10 years, 8 were females.

The paroxysmal phenomena may occur variously modified in the progress of pneumonia, bronchitis, fever, erysipelas, and other diseases affecting the habitual drunkard, or after he has received a personal injury which occasions a severe shock to the system. I need scarcely add, that delirium tremens thus complicated, is frequently fatal under any mode of treatment; and the remarks about to be offered are not meant to apply to such instances. It may also be observed, that the foregoing account of the phenomena of the paroxysm is descriptive of the disease when running its ordinary course without being interfered with or obscured by the action of stimulants, opiates, or other treatment.

There is a form of *mania* which is sometimes mistaken for delirium tremens, but which must not be confounded with it, although characterised by very considerable muscular tremor. It is nothing more nor less than a severe and protracted form of intoxication,—an affection of the brain and membranes, in which there is great vascular excitement, resulting from the direct or immediate action of alcoholic liquors. Even so careful and discriminating a physician as Dr Watson, has noticed two cases[1] as instances of delirium tremens, the first of which partakes more, and the last entirely of the character of the affection which I am now about to describe. It has been styled by Darwin the *Delirium Ebriosum*. It originates from a single fit of intoxication, or at least from a short course of intemperance (in vulgar phrase, "a boose," or "a ramble") engaged in by persons of a peculiar mental constitution and temperament, and which is most commonly induced by some depressing emotion. It is marked by an uncontrollable desire for more drink, which, when gratified, excites to further imperious demands, begetting indecorous conduct, and engendering passions so wild and vicious, that when

[1] Principles and Practice of Physic, vol. i. pp. 394, 395.

the hereditary mental constitution is imperfect, and the previous moral habits loose or depraved, not unfrequently lead to the perpetration of violent and criminal acts. The other symptoms and circumstances characterising the paroxysm, are dry heat of skin, particularly of the scalp; sometimes considerable muscular tremor; flushed countenance; a sullen, determined, or fierce aspect; red, ferrety eyes—as in the cases of Dr Watson already noticed—; dry tongue; strong, quick pulse; and loss of appetite for everything but liquor—and that of the strongest kind—although in some instances, beastly ravenousness for anything or everything eatable which comes in the way, until the affection has attained its height, when loathing, sickness, and free vomiting take place, after which occurrence, recovery begins. This state may be brought on once in a lifetime, from some accidental circumstance leading to an act of intoxication; or it may be induced at particular periods—distant, perhaps, months or years—as in the case of those unfortunate individuals to whom the name of *dipsomaniacs*, or *oinomaniacs*, has been applied. The attack is in general easily overcome by the immediate withdrawal of all stimulants, confinement under the care of one or two firm minded and strong attendants, and the administration of emetics and purgatives. All who have witnessed the various forms of disease affecting the drunkard, will readily distinguish genuine cases of delirium tremens from this or other affections attended with delirium, as its character is so well marked.

The substance of the opinions generally held regarding the essential nature of delirium tremens may be stated to be—that it is a disease of exhaustion or irritation of nervous power, and that it has the habitual abuse of intoxicating liquors for its predisposing cause, and abstinence from, or the abstraction of, an accustomed stimulus for its exciting cause.

The first part of this definition, namely, that delirium tremens is a disease of exhaustion or irritation of nervous power, appears to me to be vague, and not easily explainable, either on the principles of physiology or pathology. Various authors,[1] in the most arbitrary manner, make use of the terms exhaustion and irritation, separately or together, as it seems to suit their purpose. One can understand what the term nervous irritation, or what that of nervous exhaustion

[1] Blake, On Delirium Tremens. Copland, Dict. Pract. Med., p. 497. Watson, Pract. of Physic, vol. i. pp. 400, 401. Carpenter, On the use and abuse of alcoholic liquors in health and disease, pp. 28, 29, 30; and others.

may mean, and also how the latter state may succeed to the former; but that these two conditions—so opposite in their nature, and standing more properly in the relation of cause and effect—should coexist in this disease, is not so easily comprehensible; and consequently I believe that the idea of exhaustion—as interpreted according to the common sense acceptation of the word, namely, weakness, has led to much error in the treatment of delirium tremens. The more that the history and phenomena of genuine cases of this malady are considered, the more numerous do the difficulties surrounding the above explanation become. It seems to be forgotten that the disease is not occasioned by a fit of drunkenness, but that it is the result of the long-continued excessive use of stimulants, and therefore on the ordinary and well understood physiological law that exhaustion succeeds excitement in an almost invariable ratio, this affection ought rather to follow the cessation of an out-and-out debauch, than a course of systematic imbibition. The affection is, I consider, quite specific and peculiar. It is something more than simple "nervous irritation" or "nervous irritability:" it is essentially a form of nervous poisoning, which, in every instance—whatever be the state of the constitution, or however combined or associated with other diseases—is distinguished by a very remarkable uniformity of phenomena. In every instance of delirium tremens, the stimulus or alcoholic principle, a powerful narcotico-acrid agent, in whatever way atomically combined or chemically changed after its introduction into the system, acts slowly on the nervous pulp through the medium of the circulation, poisons its substance and sets up at last what may be termed an alcoholic erythism, or, if I may be allowed the expression, an alcoholism.[1] This in turn, no doubt,

[1] Since writing the above, my attention has been drawn to the work of Dr Huss of Stockholm (see an able analysis in the Brit. and For. Med. Rev., No. XIII., 1851), on what he calls "Alcoholismus chronicus," or "the chronic alcohol disease." Delirium tremens, however, is not recognised under this appellation; and does not appear to be viewed as an alcoholism at all by this author. The term is applied solely to a group of affections of the nervous system occurring in those long addicted to the abuse of spirituous liquors. These are distinguished by tremors and jerkings of the voluntary muscles, and diminished or increased sensibilities of surface to a greater or less extent; they are of gradual development; and terminate very frequently in paralysis, epilepsy, or idiocy, without any notable pathological alterations of structure. Such symptoms and results, variously modified by and combined with organic disease, are by no means uncommon in this country, but they appear to be

produces a certain amount and kind of debility in the cerebral functions, but combined with over-action of the circulation through the membranes of the brain, constituting a decided form of irritation, the tendency of which, if not allayed by judicious treatment, is to inflammatory action, and serious encephalic mischief.[1] Scipio Pinel

much more frequent, and earlier developed in Sweden and the northern parts of Germany, owing, it is supposed, to the very pernicious composition of the alcoholic liquors in general use. "With but few exceptions the symptoms have been caused by the potato brandy, which is served out over the counter of the spirit shops to the lower classes of this metropolis. Spirit from grain is not common, and the distilled spirit freed from the volatile oil (finkelolja) does not suit the palled taste of the habitual spirit drinker. The presence or absence of this oil must be carefully borne in mind in estimating the causes of the disorder."—P. 34, *Dr Huss's work*. It further appears that these potatoes, skins and all, are generally diseased or decayed; that mildewed grain is also used, and various poisonous vegetable products, such as spurred rye, *lolium* (frequently mixed with bad barley), and the seeds of the *raphania raphanistrum*—which latter was by Linnæus himself thought to be the cause of this disease; and this poisonous mixture is likewise favoured by preparation in copper vessels.—*Review*, pp. 56, 57. While Dr Huss acknowledges that all this must greatly increase and confirm the maladies described by him, he regards the alcoholic principle as the chief cause. On the other hand, the reviewer expresses a doubt (p. 59) "whether the *alcoholismus chronicus* be really dependent even chiefly upon alcohol," and considers it probable "that its phenomena result from the habitual use of alcoholic drinks holding various (narcotic and acrid-narcotic) poisons in solution in different amounts, and differing in their nature and action, but all having in common, that they exercise a highly deleterious influence, especially on the *nervous* system." Besides the probability that the various maladies described by Dr Huss are not altogether owing to a condition of *alcoholism*, it must be borne in mind that there is a great want of uniformity in the character and course of the attending phenomena, that there is nothing in fact to point out a peculiar physiological action such as may be attributed solely to alcohol. It appears to me therefore that they must be regarded simply as bad effects from drinking habits on the cerebro-spinal system in particular, through the general impairment of the nutrition of the body; and be placed on the same footing as those diseases of the heart, arteries, liver, kidneys, and other organs with their respective functions, also resulting from intemperance, and with which the so-called alcoholismus chronicus is more or less associated. The application of the term *alcoholism* to such affections as these, appears to me to be inappropriate—that given by Romberg (*Dis. of Nervous System*), namely, *tremor potatorum*, is preferable; and in the following pages, I trust it will be apparent that if there is one disease more than another arising from habitual excess in alcoholic drinks, in which *a peculiar toxicological effect* is manifested, it is delirium tremens.

[1] Watson, Prac. of Phys., pp. 400, 401.

considered delirium tremens to be "a first degree of paralytic cerebritis;"[1] Dr Abercrombie appears to have considered it as "a dangerous form of meningitis;"[2] Dr Bright actually includes it among his cases of "arachnitis;"[3] Dr Hoegh-Guldberg[4] also views it as a febrile affection, indeed a species of arachnitis; and Frank, Speranza, Andreae, and several other writers, have entertained similar opinions.[5] Such opinions, I think, come nearer the truth in regard to the nature of the disease than those generally received; and they obtain strength from its symptoms, the injurious effect of stimulating treatment, and the appearances observed in fatal cases. The post-mortem cerebral changes in so far as regards paleness of tissue and shrunken convolutions, steatomatous and other alterations of the coats of the vessels, and to some extent also thickenings and opacity of the arachnoid, along with a large amount of subarachnoid and intraventricular serous effusion, are most likely to be found in those who have had several attacks of the disease, and who have long been noted dram-drinkers. But even after first attacks the membranes generally present great vascular fulness, the arachnoid is thickened, and under it, throughout the brain, and within the ventricles, there is very considerable serous effusion. Dr Craigie's opinion of the pathology of the affection—which he terms *Meningitis phantasmatophora*, or methystic brain fever (*μεθυω ebrius sum*)[6]—is the following:—

"In the early and incipient stage of methystic brain fever, the symptoms depend on irritation of the brain. That is to say, the meningeal veins are loaded with an unusual quantity of venous blood, and the arteries with imperfectly aerated arterial blood, both charged with spirituous particles; and as this circulates slowly, it irritates the brain, and disorders the cerebral functions, *first* of sensation and perception; *secondly*, of memory; *thirdly*, of fancy; and *fourthly*, of judgment. The cerebral irritation thus induced is the great cause of the sleeplessness and restlessness, as well as of the fantastic delirium and hallucinations. In this stage of the disease, which is the *erethismus cerebri abdominalis* of Töpken, the *delirium erethicum* of Hufeland, and the *encephalopathia* of Leveillé, the symptoms may subside spontaneously, or under the use of ap-

[1] Traité de Pathologie le Cérébrale, par Scipion Pinel, p. 400.
[2] Diseases of the Brain and Spinal Cord, p. 63.
[3] Medical Reports, vol. ii. p. 10.
[4] Commentatio de delirio tremente, quoted in Brit. and For. Med. Review, vol. vi. p. 328.
[5] Referred to by Dr Craigie, Pract. of Physic, vol. ii. p. 66, 67.
[6] Op. cit., p. 53.

propriate remedies, by the poisonous blood being eliminated in the manner of excretions during sleep.

"If, however, the irritative action do not thus subside, if the vessels be not unloaded, and the circulation re-established, it is liable to become fixed in the form of inflammatory congestion, and to give rise to effusion of serum and other morbid products. This seems to be the *encephalitis* of Frank and Hildenbrand, and the Hirnentzündung of Andreae. Even without effusion of serum, the fatal termination may take place; but this result is much more frequent in consequence of effusion, sub-arachnoid, cerebral, and intraventricular. The disorder, therefore, though merely irritative in the early stage, from the unhealthy state of the blood sent to, and retained within the cerebral vessels, becomes at least in the latter stage congestive, and perhaps even inflammatory."[1]

To get rid of the difficulties which the above considerations raise up against the favourite theory regarding the essential nature of delirium tremens, some have spoken of it as occurring in two forms —asthenic or sthenic, congestive or inflammatory; but such distinctions, while presenting a fine hair-splitting diagnostic aspect, really serve a bad purpose, by originating perplexing doubts and difficulties. Like plumbism, mercurialism, ergotism, or narcotism, alcoholism is, manifestly, specific in its nature. Lead, mercury, and other agents, may affect individuals in different degrees from difference of age, constitution, continuance of exposure, etc.; or the effect—like that of the virus of small-pox—though the same in kind, may be modified in one case more than another. When alcoholic liquors have been long abused, the active principle appears to affect the system, by accumulation, like some other poisons. It has been supposed that the gastro-enteritic disorder, from long continued drinking, originates an attack of delirium tremens, through the medium of the solar or coeliac plexus, and affects the brain only in a secondary or sympathetic manner;[2] but although disorder of the stomach, liver, and other organs of disgestion is an ordinary result of drinking habits, yet thousands of individuals are affected in this way, and die in consequence, without ever suffering from delirium tremens. There is something more required to occasion this disease, the first manifestations of which are shown rather in disturbance of the organic functions by transmitted influence *from* the brain and nervous system to the digestive organs. The effect is brought about after the manner of a cumulative poison, the action of which is on the nervous centres. The experiments and observations of Dr

[1] Op. cit., pp. 81, 82.

[2] Göeden of Berlin, as quoted by Dr Craigie, op. cit., pp. 67, 68.

Percy[1] prove that alcohol has a peculiar favour for cerebral matter, fixing at once on it, by a sort of elective affinity; and indeed in fatal cases from direct intoxication, its actual presence in the substance of the brain is demonstrated. Now, in the longer continued abuse of alcoholic liquors, is it probable that the selection of the agent will be different? Its accumulation may be slow, and the change in chemical constitution may be considerable, but it is not the less sure. Every additional drop imbibed brings the grey matter of the brain into that state which assists in the development of the alcoholic erythism; and thereafter, while disturbing the functions of digestion, etc., occasions those relative changes in the sanguiferous system of the encephalon, the tendency of which is, as I have already affirmed, to pass from irritation, or abnormal activity of circulation and functions, to inflammatory action, according to the severity of the attack, and other circumstances. It therefore does appear strange that physicians while describing delirium tremens as a disease of exhausted or irritated nervous power caused by intemperance, should recommend and practise as a remedy the very agent which occasioned it, or another, namely, opium, which, although unable of itself to produce it—as I shall afterwards show,—greatly assists and hastens the affection in those who habitually indulge in intoxicating liquors; and moreover an agent, the physiological action of which is to occasion engorgement of the vessels of the brain—vessels already too highly charged with blood containing a poisonous ingredient. This is truly acting, to a certain extent, in the spirit of the homœopathic dogma—"*similia similibus curantur!*"[2] But of this more hereafter, for I am now brought to the consideration of the second position, which assumes in explanation of this affection, that while the abuse of intoxicating liquors is its prédisposing, the abstraction of, or abstinence from an accustomed stimulus, is its exciting cause.

With the first part of this proposition I quite agree; but the opinion that the privation of a usual stimulus must be regarded as

[1] Experimental inquiry concerning the presence of alcohol in the ventricles of the brain.—*Prize Thesis*, Edin. 1839.

[2] Hahnemann, however, and his disciples, do not recognize alcohol as a curative agent; and, strangely inconsistent with the dogmas of their creed, say, (*Organon*, § CCLXXXVI., p. 329), "it is only the most simple of all excitants, wine and alcohol, that have their heating and intoxicating action *diminished* by dilution"! Opium is homœopathically recommended in delirium tremens, not on account of its sleep-giving power, but because its effects are supposed to resemble the symptoms of that malady!

the exciting cause of the malady, I consider to be entirely erroneous. Analogy, certainly, will not bear out the theory. Mercurial fumes, or the oxides of mercury, when long inhaled or absorbed into the body, as in the case of gilders, quicksilver-miners, and others, in the course of time produce an attack of shaking paralysis—the *tremblement mercurial* of the French pathologists; but will it be averred that the workmen long exposed are more likely to be affected with tremors, if removed from this poisonous atmosphere and occupation, than if they continued at their work? The reverse is well known to be the fact, not only in the case of such artizans, but of those also who are beginning to suffer in a somewhat similar way from lead poisoning. In both affections, when the symptoms are recent, a cure can only be effected by removal from the injurious occupation; otherwise the symptoms deepen with hourly increasing rapidity, until tremors are succeeded by sleeplessness, delirium, and, ultimately, coma.

But, then, even granting it possible that the privation of a wonted stimulus may be the exciting cause of delirium tremens, is it a fact that it is so?

The supporters of the theory now under discussion, with the exception of Dr Blake,[1] do not make a positive assertion on this point. They speak of the disease as "commonly resulting from the abstraction of the accustomed stimuli after a habitual or continued indulgence in it, or after a protracted fit of ebriety;"[2] or as, "chiefly when sobriety has followed a protracted debauch."[3] Again, "This disease most frequently occurs in habitual drunkards, and especially when after repeated fits of intoxication, they suddenly lessen, or leave off their ordinary stimulus for a time."[4] "Delirium tremens occurs more frequently when the accustomed stimulus is withheld."[5] "The disorder frequently does not show itself until the accustomed stimulus has been withdrawn for a certain period."[6] "Very frequently, from some cause or other, this habitual stimulus has been taken away."[7] The stimulus "has been, in general, suddenly withdrawn before the disease distinctly shows itself."[8] From these quotations it is apparent that some instances of the disease are admitted

[1] Op. cit., p. 23. [2] Copland, Dict. Pract. Med., p. 496, sect. 9.
[3] Ibid. sect. 10. [4] Armstrong, Practical Illustrations of Fever, 1819, p. 493.
[5] Carpenter, sect. 27. [6] Taylor, Med. Jurisprudence, p. 613.
[7] Watson, Practice of Physic, vol. i. p. 393.
[8] Alison, Path. and Pract. of Med., p. 734.

to occur without *any* privation of accustomed stimulants; and Dr Watson honestly says, "Sometimes it comes on in men who are perpetually fuddled, even although they have not intermitted their usual allowance of drink." It happened thus in several of the cases which I shall give at length in the present communication; and I could mention many other instances, in which there was no diminution in the quantity of liquor consumed—and some even in which there was a decided increase—up to the moment of seizure.

Since, then, intoxicating liquors may be, or rather often are, the directly exciting, as well as the predisposing cause, of delirium tremens, the second position in the theory—stated with all the precision of a law, and which, as such, ought to be invariable—is untenable; and as the idea expressed in the first part of the theory, that, namely, of exhaustion of nervous power, has been formed on the supposed correctness of the second proposition, the whole structure must be abandoned as without foundation. To me it is apparent, that habitual excess in the use of stimulants is alike the exciting and the predisposing cause of delirium tremens; and that if a suspension or diminution of habitual supplies be at any time attended by symptoms of the disease, these are not to be regarded as resulting from change in the quantity consumed, but as occurring in spite of such change, and because the peculiar constitutional effect has already been induced, and the premonitory stage of the affection already begun. I feel persuaded, that every practitioner who has seen much of this disease must, on an impartial review and consideration of his cases, confirm this remark. For my own part, I can affirm, that in a very considerable number of instances the patients were drinking freely up to the period when the disease was developed, there being no interval, and no diminution of quantity; and where there really was some diminution from the amount of previous supplies, it was on account of the system having already been brought into the condition of alcoholism, and a less quantity now produced a greater or equal effect, compared with that of the larger quantity taken formerly. There are, in some instances, no doubt, an entire cessation from the use of stimulants, or very nearly so, at the time when the symptoms of delirium tremens are in the course of development, but this is because no more can be taken by such individuals:—they are already saturated, as it were, with the alcoholic poison. From overlooking these circumstances, I believe all the statements in regard to the supposed effects of diminution, suspension, or abstraction of an accustomed stimulus

have originated. The error is a popular one, and has arisen from imperfect inquiry into the history of individual cases, and incorrect observation regarding the circumstances connected with the supposed reduction or abstraction. When called to see a case of delirium tremens, on inquiry as to the habits of the patient, we are frequently informed by his friends, that for a long time large quantities of spirits, or wine, or malt, or of all of these—and perhaps, in addition, morphia or opium—had been systematically consumed, but that for some time—a few weeks perhaps—much less had been taken, and within the last few days little or none; and then the inference is drawn for us, that the unfortunate patient has actually brought on the attack by meritorious efforts to free himself from a habit of which he had begun to be ashamed. Now all this is very plausible, but not in accordance with the strict facts of the case, as the individual himself, if put on his word of honour, will probably confess. The statement ought to be, that he was formerly in the habit of consuming large quantities of his favourite stimulant, until he found that a much less dose began to affect the system; that then he reduced the amount still further, but experienced an equal, if not greater, constitutional effect therefrom; and thus, from day to day, reduction was forced on him by his own sensations of gastric irritation, nervous excitement, and muscular debility,—these feelings having been, in fact, neither more nor less than the premonitory symptoms of the attack of delirium tremens, and just what might have been expected if—as I have ventured to assert—the alcoholic principle is to be viewed as a cumulative poison.

The habitual and excessive use of intoxicating liquor, however, does not affect all individuals alike. Some drinkers are early cut off by diseases of the liver, heart, or other organs, to which they may have a hereditary or constitutional liability; others in fevers or inflammations, which they have no stamina to contend against; others by apoplexy or paralysis, from the direct effects of a debauch; and some from hereditary predisposition, or otherwise, are early doomed to spend the remaining years of a miserable existence in mania, idiocy, or in a general paralytic condition of the system.[1] Some few drinkers, again, by reason of extraordinary constitutional vigour, escape all these ills, and live on to old age; but the greater number who are not early removed from society by the diseases enumerated, suffer more

[1] This comprehends the group of affections described by Huss under the name of the Chronic Alcohol Disease, and which has already been referred to.

or less from the attacks of delirium tremens. There appears to be a certain peculiarity of constitution which predisposes the individual to this malady—provided drink has been systematically indulged in,—that, namely, in which there is a highly sanguine temperament, and a nervous, irritable disposition. And I consider also, that the readiness with which the disease occurs, and the mode in which it is developed, are well explained by other individual peculiarities and circumstances. While one is attacked suddenly, without any diminution of the quantity, or change in the kind of stimulant, another is more slowly and gradually affected, and it may be, after very considerable reduction in the supply of liquor. In these respects, however, the effects of the stimulant are simply analogous to those of various other potent medicinal agents. For example, some constitutions are easily affected with mercury, others with difficulty. Salivation may all at once be displayed in one individual, who has taken the drug for a long time in very full doses, while in another it is produced by very gradual degrees, although with an equal amount of the mineral. But in both instances, after the constitutional effect is once produced, it may be kept up and increased to an excessive and serious extent by very small quantities of the mercury; and in the latter circumstances it would be very absurd to aver, that salivation was owing entirely to the more recent and smaller doses of the drug; or, still more absurd, if the mercury was altogether withdrawn, to say that the increase or continuance of salivation depended upon the abstraction. In like manner, when the nervous tissue of the brain has become charged with the alcoholic poison beyond a certain point, the effect it produces is kept up, and even increased, notwithstanding very considerable reduction in the amount consumed; and we are thus enabled to explain why diminution is almost universally supposed to be the cause of the malady, when, in fact, the indisposition to take more is itself one of the precursory symptoms of alcoholism. In the delirium ebriosum, there is urgent desire for drink during the violent stage of the affection, until a paroxysm of sickness occurs, which induces exhaustion, and then sleep; but in the delirium tremens, there is seldom any desire for it, even at the beginning of the attack, and certainly none when the affection is developed. Illustrations of the tendency to accumulation might be drawn from the effect of other medicinal agents, each acting in its own peculiar way, and on particular organs or functions. This, however, appears

unnecessary, for I think it has been clearly shown, that the alcoholic principle—imbibed systematically—passing through the channel of the blood, in whatever way atomically combined or chemically changed, has its influence concentrated on the nervous pulp of the brain, accomplishes its work on the perceptive, sensory, and motor powers,—in one case quickly, in another, if not soon, at last suddenly; or, by disturbing the varied functions of the economy, it induces such a condition of the system, that a smaller quantity taken will ultimately produce a more intense and lasting effect.

These views of the subject, to my apprehension, help to explain why delirium tremens is so readily brought on in dram-drinkers, when subjected to external injuries, or when seized with any kind of inflammation or fever. The sudden shock to the system in the one case, and the altered balance of the circulation and disorder of nutrition in the other, brings, I conceive, the individual at once into the condition of susceptibility to this disease, which would not otherwise perhaps have been so early accomplished. The effect is somewhat similar in the case of those who possess the gouty diathesis, for an injury of a limb is extremely apt to precipitate an attack of gout, which, in the ordinary course of events, would probably not have taken place for a considerable period of time. There is, apparently, in the habitual drinker of a nervous temperament, a tendency to delirium tremens, as there is in the *bon vivant* of a certain temperament to gout, and as there is in the epileptical or hysterical subject, to epilepsy and hysteria, although in each instance there is great dissimilarity as regards condition, cause, and effect; and any sudden excitement, shock, or severe malady which powerfully affects and disturbs the vascular and nervous systems in individuals so predisposed, may greatly aid in bringing on a characteristic attack or paroxysm. It is in this irritable state of the habitual drunkard's constitution, although he may not be on the verge of delirium tremens, that alcohol, by its presence in the blood—in whatever way combined—and its interference with the nutrition of the brain and nervous system, will superinduce on the receipt of an injury—say a gunshot wound, a severe burn, or a fracture—a febrile attack, attended by delirium presenting somewhat of the appearance of that disease, but which in reality has more of a typhoid character. This affection has been named by Dupuytren "*Delirium nervosum s. traumaticum*;"[1] and although some writers have considered it identical with delirium tremens, it only similates it, being a symptom of the sympathetic fever which occurs under the circumstances above noticed. But whether delirium of this character, with some degree of tremor, takes place under these circumstances, or whether an attack of pure delirium tremens immediately supervenes on the receipt of the local injury or disease from that state of the constitution, and the previous habits of the individual, already explained, it is quite erroneous to suppose, in either case, that the affection originates from the suspension of the stimulants to which he had been previously accustomed, however plausible the theory may be which thus accounts for its production.

[1] "Annuaire Médico-Chirurgical des Hôpitaux."

The idea, that bad consequences result from a sudden abstraction of stimulants, having got possession of the minds of many able writers on this malady, all their views of its nature have been perverted, and they have misled the profession into a dangerous system of treatment. Dr Blake, for example, who has been much quoted as an authority on delirium tremens, says: "It is purely idiopathic, arising *invariably* from the same cause, namely, the sudden cessation from, or a material diminution of, intemperate habits;"[1] and he goes so far as to assert that at "almost any time he *could have* brought on an attack of delirium tremens in the habitual drinker, by simply taking him into hospital for three or four days, and keeping him on spoon diet."[2] He does not appear, however, to have tried this experiment. It is an assumption from a theory supposed to be true, and has appeared plausible from the fact already admitted and explained, that the disease sometimes occurs in those taken into hospital on account of sudden or severe shocks to the nervous system from injuries and other maladies, but who have had the alcoholic erythism strongly developed, and who are, in fact, already on the verge of an attack of delirium tremens. The gourmand would feel equally weak and miserable, and his general tone be for a time depressed by the abstraction of good living; but however strongly the gouty diathesis was in such a case, this deprivation of good things would not occasion an attack of gout, although an injury of any kind, nay, a scratch, might. So I hesitate not to say, that the dram-drinker, in whom the delirium tremens diathesis is not yet fully established, and who is not already under the precursory symptoms of the disease, could not be subjected to a paroxysm by such treatment. From the sudden change on his circulation, he would doubtless experience

[1] Op. cit., p. 23.

[2] Op. cit., p. 18.

much mental disquietude and physical discomfort, and be made "shaky," according to common phraseology, for a time; but this would soon pass off, without the occurrence of the usual pathognomonic signs of delirium tremens, more especially without those spectral illusions or phantasms which are common to poisonings, with several other agents of the narcotico-acrid class.

The opinion of Dr Craigie on this point is so decided, that I cannot resist quoting it. He says:—

"Without positively denying that the disease may come on in this manner, I can only say that I never witnessed an instance of this mode of development; and, after perusing all the published cases extant, I cannot perceive that any of them, excepting the one recorded by Dr Armstrong, in the 9th vol. of the 'Edinburgh Medical and Surgical Journal' (p. 146), afford satisfactory evidence that the disease is induced in consequence of the sudden abstraction of the use of spirituous liquors; and even that case, I think, may be explained without having recourse to the supposition now mentioned. I have, on the contrary, never observed that the sudden and complete abstraction of these liquors aggravated the symptoms of the disease. I find further, that neither Berndt, Toepkin, Hufeland, Andreae, Göeden, Sieburgundi, nor any other foreign physician by whom the disease has been observed, admit that it is produced in this manner; and in all the cases recorded by them, the symptoms were developed after a continuance, more or less protracted, of stimulation by spirituous liquors."[1]

In order to obtain some additional evidence on this disputed point, I submitted some queries to Drs Simson and Gibson, the medical officers of the large prison establishments of this city and of Glasgow, and to Mr Page and Dr Scott, surgeons to the county gaols of Carlisle and Dumfries; and the following information has been kindly furnished by them, as to the effect of the sudden withdrawal of all stimulants from civil and criminal prisoners known or presumed to be of intemperate habits, and the immediate substitution of prison fare, which is well known not to be of the most generous description.

As regards the prison of Carlisle, it appears that, although the annual number of commitments during the last fifteen years has been about 600; and that, although three-fourths of these are considered to have been, in one way or another, the consequences of drunkenness, Mr Page states emphatically he has never yet seen any ill "result from the sudden abstraction of stimulants from

[1] Op. cit., 57.

habitual drunkards, who had been drinking to excess up to the time of being placed on prison fare." Mr Page had also, during nine years' experience in connection with the Carlisle County Pauper Lunatic Asylum, observed the same impunity with which all stimulants could be at once withdrawn. (*Letters, 9th and 21st June* 1854.)

Of the gaol of Dumfries, it is stated by Dr Scott (*Letters, 12th and 21st June* 1854) that, during the last fifteen years, the number of civil and criminal prisoners have amounted to 5539; that of this number he supposes about two-thirds were committed for crimes resulting from intemperate habits; that he believes a very large number to have been habitual drunkards; and that, although all of these, of course, were deprived of their usual libations, and at once put on prison allowance, only five cases of delirium tremens are found on the register of disease, and that all of these patients but one were admitted to the prison with the disease on them; and that in regard to that one, although entered as under delirium tremens on the day after admission, there is every probability for believing that *she* had had the disease on her when admitted, although not reported to be ill. Dr Scott also notices, as an important fact, that during the time the railways were being constructed in the county of Dumfries, a very large number of navvies were committed to prison, who had led a very dissipated life for many months, and although deprived of liquor from the moment of apprehension, not a single case of delirium tremens occurred.

Then, as regards the prison of Glasgow, in which the annual commitments amount to upwards of 4000, the experience of the year 1850 is adduced by Dr Gibson (*Letter of 16th June* 1854), as affording an approximation to the facts wished to be elicited. A calculation made in that year showed that, while 4122 were imprisoned, the number of assaults, with few exceptions, committed under the influence of liquor, and "the drunk and disorderly," amounted to 1519; and of this number only three cases of delirium tremens occurred—a very small proportion indeed, especially when it is considered that the debtors, who are almost all habitual drunkards, and drinking up to the moment of incarceration, are not included in this list. Many hundreds more, therefore, may be considered to have belonged to the drunken population of the gaol. The average of the last ten years, however, is greater (5·7), there having been fifty-seven cases altogether during that period, but, after

all, this is a very small proportion to the number of dissipated and drunken characters gathered together there, and at once broken off from intemperate habits. Dr Gibson, however, states that he does not altogether enter into my views as to the proximate cause of delirium tremens, although he admits that "it does not so frequently occur as the advocates of the theory, which attributes it to the total withdrawal of accustomed stimuli, such as Blake and others are inclined to suppose;" and he mentions, in proof of his objection, that he had never seen it occur in less than twenty-four or beyond seventy-two hours after apprehension, which necessarily put a stop to dram-drinking. As I have already explained, however, and as the cases given at the conclusion of this paper will show, there is always, whether the individual is drinking much or little, more or less of a premonitory stage present in this affection, distinguished by digestive derangement, nervous irritability, restlessness, and sleeplessness, before much tremor is displayed, or any illusions manifested; and it is easy to suppose that these might not be brought immediately under the notice of the medical officer of a large criminal establishment, such as the Glasgow prison. But even granting that no incipient symptoms of the disease were observed, and that this proportion of the habitual drunkards were not quite on the verge of being affected with it, it is quite in accordance with the views already advanced to suppose that, when there was a certain amount of alcoholization existing, the disease might be hurried on more speedily than otherwise would have been the case in individuals of a nervous and excitable temperament, by the agitation or shock of apprehension, and the deprivation of liberty. But, further, I should suppose it a very just, nay moderate calculation, to assume that out of a population of 2000 confirmed drunkards belonging to any class of society, although enjoying unrestrained liberty and uninterrupted opportunities for indulgence to excess, at least from three to six instances of delirium tremens would annually occur.

But, in fine, on this point, the evidence communicated by Dr Simson, the medical officer to the prison board of this city (*Letter, 4th July* 1854), is sufficiently satisfactory; for while the number of civil and criminal prisoners, committed during the last year, was 5864 (which may be assumed as a sample of the previous fourteen years, over which Dr Simson's experience extends), only four cases of delirium tremens occurred within the last eighteen months. The average number of cases during former years, Dr S. states as from

2 to 3 per annum. Dr S. considers that, at least one-half of the whole prisoners may be assumed as dissipated characters, and that at the very lowest computation, 500 must have been regular, systematic drunkards, from whom all drink was suddenly abstracted; and he goes on to state as his decided opinion, that "the sudden taking away of spirits, etc., does not produce delirium tremens. In *every* case, the prisoner had symptoms of the disease on him when admitted—that is, they were all restless, irritable, etc.; and I have, no doubt, but that in many instances the crimes committed were the effects of this disease. I do not remember a single case of delirium tremens occurring when the prisoner was quite well when received into prison. There is not the least doubt that a peculiarity of constitution predisposes to delirium tremens," etc.

Here then, it has been shown, that hundreds of individuals among the public at large, and of the criminals committed to our gaols, leave off or are suddenly deprived of the stimulants to which they had been previously addicted, without being seized with delirium tremens, or anything approaching to it.[1] On the other hand, also, it is unquestionable that numerous instances of the disease do occur in which there has been no suspension either voluntarily or by compulsion of the amount of liquor consumed, nay, even an increased excess in drinking up to the very moment of seizure.[2] The assumption, consequently, that this disease is produced invariably, or chiefly, or even occasionally, by the diminution or abstraction of an accustomed stimulus, is not supported by facts. Any cases, therefore, noticed as occurring under these circumstances, are simply of an exceptional character, but which in my apprehension, fall quite short of proof from the considerations already so fully explained.

Some even, borne away by the theory that delirium tremens is a disease simply of "exhausted nervous power," and refining on the idea, have gone so far as to aver that it may occur independently of the use of intoxicating liquors altogether. Thus it has been alleged to have been produced by the use and disuse of opium and of tobacco;

[1] Dr Scott has mentioned to me the case of a debtor who, although up to the hour of his incarceration in the Dumfries gaol, was in the practice of taking on an average, one bottle of spirits, and upwards of three ounces of laudanum daily, yet had every drop withdrawn without experiencing any bad symptom. This individual, too, it is interesting to know, had been twice previously the subject of delirium tremens.

[2] Cases II. III. VII. and X. are instances of this.

to have been resulted from protracted mental application, from excessive depletions or evacuations, from rheumatism, from exposure to extreme cold, from hunger, etc., when no liquor of any kind had been indulged in. In all such instances there must have been some mistake or misapprehension. I can suppose that the continued use of inordinate quantities of laudanum might occasion delirium tremens, as has been reported, from the amount of alcohol necessarily consumed, which of itself would be sufficient to occasion it, and more especially when combined with opium, which, as shall afterwards be shown, has a great influence in hastening its development; but that opium alone ever produced the disease I do not believe. I have never seen it; and when it has been supposed the cause, there must undoubtedly have existed some misapprehension of the history of the case, or some concealment as to the previous habits of the individual, for nothing is more common than for an opium eater to indulge also in some stimulating beverage. Opium, when habitually taken by itself, may, in the course of time, break down mental and physical energies, giving the aspect of premature old age, if not occasioning actual imbecility or paralysis, but it will not produce delirium tremens; and when left off suddenly, the poor victim of the enslaving habit will for a time feel very wretched and feeble, but will not manifest the pathognomonic symptoms of delirium tremens, and will have the best chance of regaining to some extent his constitutional vigour. In regard to any influence which the disuse of tobacco may have in occasioning this malady, I would say that it is quite out of the question, and that any attack occurring in the case of the recent smoker, must have been owing to the conjoined habit of drinking. As an illustration of the erroneous notions prevailing in regard to the matter, and to the disease generally, I will give one of the last published instances of delirium tremens—a good example of a mistaken cause, and a misunderstood effect—wrong theory, and wrong practice. It is entitled "*Delirium Tremens produced by Abstinence from Tobacco.*"[1] The italics are introduced to draw attention to some points of importance.

"Delirium tremens, and its twin sister, traumatic delirium, are now so well understood to be dependent on *asthenic irritability of the nervous system*, that but one opinion prevails as to the principles which should regulate their treatment. *Sudden disuse of accustomed stimulants* is always to be deprecated, and

[1] Medical Times and Gazette, No. 163, Aug. 13, 1853.

in the event of a patient of known intemperate habits coming under surgical treatment, especially on account of an accident, care should always be taken that he is not deprived of his wonted allowance of alcohol. There is, however, another very potent drug in but too common use among the lower orders, the probable effects of suddenly relinquishing which, have, we suspect, been too little considered, and respecting which, the notes of a case lately under the care of Mr Curling, appear to offer a valuable hint to the practical surgeon. A withered old woman, *a gin drinker*, and a habitual smoker, was admitted on account of *a severe burn. Stimulants were from the first freely allowed her, and opiates administered*, but in spite of them she continued extremely restless, wandering at times, and quite unable to sleep. Her manner and aspect indeed *much resembled those of delirium tremens.* At this juncture, *several days after admission*, Mr C. ordered that she should be permitted to smoke. The salutary influence of the permission was at once apparent, the woman became quiet and tranquil, and on the next night slept fairly. All tendency to delirium disappeared, and she afterwards progressed steadily to recovery."

Now this was nothing but a mild case of delirium tremens, from habitual gin-drinking, precipitated by the severe burn, and aided by the stimulants and opiates so freely given; and in consequence of these combined circumstances—not in spite of them—the restlessness, etc., continued. The absence of the tobacco had nothing to do with this state of matters, but the disease originated from its ordinary cause, and was running its ordinary course of a few days. Convalescence was in all probability begun when the tobacco was allowed, but if not, no doubt its effect would be good, for it would act, not as a stimulant or a narcotic, but as a sedative—soothing and depressing the cerebral excitement—and sleep would follow as a natural consequence. It would not be surprising if tobacco alone, given in other cases, proved beneficial.

Then as regards the other causes, independently of alcoholic liquors, said to produce delirium tremens, the kind of delirium differs in each case, or partakes more of the characters of insanity; and there is also a corresponding diversity in the nature of the wakefulness, the muscular tremors and other symptoms, all of which circumstances, if space permitted, could be explained on very different grounds. But the mental phenomena of true delirium tremens, distinguished by a quick, eager, busy, apprehensive, spectral character, viewed in conjunction with the peculiar tremors, and sleeplessness, and with the uniform course and character of the other general symptoms, are surely sufficiently diagnostic. The term delirium tremens is, no doubt, pathologically incorrect, for incoherence and tremor may coexist in very dissimilar

states of mind and body, and originate from a diversity of causes; but it has been so long assumed by the profession, and known by the public as applicable to a disease originating solely from continued and excessive indulgence in alcoholic stimuli, that a more general signification cannot be recognised without leading to confusion and error. The cause and the course of this interesting disease are so very different from that of the affections above noticed, that no affinity in nature or pathology can be admitted. The functions of the brain in it are, I conceive, interfered with in consequence of the vitiated nutrition of its substance, and the irritation of the membranes. A peculiar erythism and excitement, as has been already asserted, is thus set up by the continued introduction and presence of the alcoholic poison; and every drop of intoxicating fluid now supplied to the circulation increases the poisonous action. If the supplies are still increased beyond this point—the furthest limit of endurance—the unfortunate individual will, in all probability, be seized with fatal convulsions or coma; or be cut off, or shattered for life, by the establishment of decided inflammatory action from protracted excitation of the brain and its membranes.

Now, if I have succeeded in showing that the alcoholic principle tends, by long-continued and excessive use, to occasion delirium tremens as the result of a specific poisonous action on cerebral matter; and that this happens on the principle of accumulation (as is the case with many other agents, such as mercury, lead, iodine, opium, Indian hemp, strychnia, etc., each acting in its own peculiar way), it should follow, that even a small quantity administered in the treatment of that disease, must necessarily increase the mischief instead of curing it. On theory, therefore, the rule of practice appears to be sufficiently evident; but whether or not the above reasonings and statements are considered sound and satisfactory, no inconsiderable amount of experience may be claimed in recommending an entirely non-stimulant and non-narcotic plan of treatment. I am aware that in advocating the disuse of stimulants and opiates I may be considered guilty of a medical heresy. The practice objected to has, there is reason to believe, been for long almost universally followed in this country, to a greater or less extent. It has arisen, partly, from blind adhesion to the popular error I have already attempted to expose, which assumes that delirium tremens originates from, and is aggravated by a diminution, suspension, or abstraction of an accustomed stimulus, and therefore to be treated successfully only

"by a hair of the dog that bit;"[1] and it has arisen partly also from acquiescence in those modern pathological notions which attribute so much to the disordered organisation, and diminished nervous power of the solids, and so little to vascular disturbance, to chemical change, and to poisonous action of the fluids of the body. It is pleasant to observe the spirit of inquiry now drifting towards the much neglected claims of a humoral pathology, and bent on investigating the nature and extent of blood-poisonings; and I despair not of seeing, ere long, still greater advances made in this direction.

As regards the treatment of delirium tremens on the views which I have endeavoured to unfold, the experience of upwards of fifteen years may be pleaded; and during the five previous years I also had ample opportunities of witnessing the practice of others, and of personally testing the merits of the mode of treatment ordinarily pursued. In the earlier period of practice the observations were made almost entirely in connection with hospital and dispensary attendance, affording a great many examples of the disease both in its pure and in its complicated forms, as occurring among tavern-keepers, brewers, butchers, and the lowest order of dram-drinkers generally; latterly the instances have been mostly among a better class of society, yet the disease presenting the same features, and originating from the same degrading cause. The frequent sudden fatalities which I have witnessed from arachnitis, convulsions, and coma, when stimulants and opiates were freely administered; and the length of time ere recovery took place, even in the most favourable instances of the malady, when these agents were given more sparingly and cautiously, long since convinced me that their tendency is highly dangerous. I do not say that I would never give a stimulant in delirium tremens. It may possibly happen, although I have never met with such a case, that in the advanced stage of the affection the pulse may begin to falter, the heart lose its usual rythm, the surface of the body to become of a leaden hue, the tremors to disappear, and subsultus tendinum occur, and delirium of a muttering character only continue, then certainly the flagging powers of life would require to be sustained by some diffusible stimulus. Here there would be no alternative. Then, again, I would not hesitate to give an allowance of his usual stimulant to a habitual drunkard

[1] The common practice has been, and, I have reason to know in many quarters still is, to give from one tablespoonful to a wine-glassful of spirits every two or three hours, either alone, or combined with opium.

when affected with a wound or ulcer, so as to obtain a healthy action therein; or to administer stimuli of one kind or another freely in ordinary fever, or in the typhoid state of traumatic delirium, so that his circulation may be enabled to keep up the functions of organic life until food be made use of. This would only be using legitimate means to maintain his ordinary condition of body; but it is quite another thing to prescribe alcohol when the individual is already manifestly in a state of alcoholic poisoning.

From all that I have seen and read, I believe that the combination of stimulants with opiates is a most hazardous practice in the treatment of delirium tremens; for while the former increases the determination of blood to the head, the latter is apt to occasion engorgement there, and thus, doubtless, they are the joint cause of many sudden deaths, and of many incurable palsies of body and mind—indeed of the great proportion of those casualties which take place, and for which the disease, and not the treatment, is blamed.

Opium given alone in delirium tremens is, I am aware, almost universally considered by the profession to be quite an indispensable agent—the *sine qua non*—for securing what is called the critical sleep; and hence it is prescribed in smaller or larger doses in as routine a manner as sulphur is for the itch, or colchicum for gout. Notwithstanding this high estimation of its value, however, I hesitate not to say that it is a very doubtful remedy even in the most promising cases of the disease, and a most dangerous one in others. It is well known that a moderate dose of opium in delirium tremens, so far as regards its action on the brain and nervous system, is in the first instance exciting and preventive of sleep. I have frequently seen such doses as in other affections would have been considered very large, in this greatly increase the agitation and excitement after each successive administration; and although sleep was secured at times, it was but short and disturbed, and followed by delirium as violent as before. Besides, the most unmanageable cases of delirium tremens which are met with, are those affecting opium or morphia eaters, who appear to be extremely liable to this disease if they indulge in spirituous liquors. From the use of opium or morphia alone, as already stated, true delirium tremens never occurs; but with the unfortunate slaves of this debasing habit, a very short course of intemperance is sufficient to develop it.[1] I have also remarked in

[1] See Case No. V.

several of these instances, that if, during the attack the usual dose of the narcotic is taken under the impression that it would soothe distress and procure sleep, more especially if that dose be morphia—which is apparently much more stimulating in this affection than opium—the paroxysm is greatly aggravated.[1] It is evident then, that if opium is to be used at all in delirium tremens, it must be given in a large dose (in from two to three or more grains, and repeated at intervals of a few hours); and it is thus generally given, the object being to overstep the stage of excitement, and force on the desired sleep. Now the acknowledged effect of a large opiate on the encephalon is to occasion engorgement of the vessels, more especially of the veins, and consequently, the larger the dose the greater will be the amount of sanguineous compression of the brain. What then must be the probable result in a disease in which there is already, if not an approach to arachnitis, at least a very excited action of the meninges, and a preternatural loading of the vessels generally? The cerebral functions are oppressed, and at length overwhelmed, and sub-arachnoid effusion is the result. The symptoms attending this untoward event are characteristic. Sleep is obtained, but it goes on deepening, and, as it becomes more profound, the pulse becomes smaller and less frequent, the surface of the body covered with a cold sweat, the face pale, the pupils contracted, the breathing slow and soft (although sometimes stertorous). An epileptic fit may now occur and terminate the scene, or the powers of life gradually become more and more depressed, and the victim perishes as if in a profound and gentle sleep. Now this progress and catastrophe, although viewed as evidence of an unmanageable, a malignant form of the disease, in a bad subject, is nothing more than the common course and result of injudicious management. Even Graves, who prescribed opium in delirium tremens in the manner I will afterwards notice, warns emphatically against its premature and incautious use.

"Opium," he says, "if given in the beginning, will increase the congestion and bring on sub-arachnoid effusion. I treated a case of delirium tremens in this way too boldly, and the man died with sub-arachnoid effusion; it was a lesson to me, and I advise you to profit by my experience."[2]

I am convinced that it is in this way very many of the sudden deaths we hear of in delirium tremens occur. I saw it frequently in early practice, and have seen it occasionally since in the practice of others; and feel persuaded that any practitioner who has been

[1] See also Case No. V.

[2] Clinical Lectures, vol. i. p. 530.

accustomed to treat this affection with large doses of opium, will be able, on reflection, to explain his want of success, and the occurrence of casualties. In fact, when recovery takes place after a long sleep forced on by a large opiate, it is simply from the wonderful conservative power of nature resisting the evil influence of the agent, just as some will recover from a severe apoplexy or a palsy. The practice is one of the utmost hazard. If death were the certain alternative in delirium tremens should sleep not be early obtained—for it it is said that "the patient must sleep or die,"—there might be some reason in attempting to force on the sleep by opiates. This, however, is certainly not the case, and consequently such interference is not only uncalled for but most improper, when there is danger to be apprehended from the practice. Sleep occurs as the natural, the favourable crisis, or rather termination of the disease; and it is not to be viewed as a part of the affection, or in the same light as we are accustomed to regard a critical sweat or other discharge. It is the result and the proof of an improved condition of the brain and nervous system,—a salutary relaxation succeeding a state of dangerous tension. It will take place in the mild but genuine forms of the affection at the proper period, which, as I have already remarked, is on the second or third day, when the paroxysm has run its course, when the peculiar erythism, the "nervous irritability," is brought to an end, and a condition of "exhausted nervous power" now truly produced. That sleep may likewise ensue in severe examples of the disease, although no opiate of any kind is given, the cases at the close of the present paper will prove; and while convinced that the plan of treatment now to be recommended will be found the most efficacious, I have no hesitation in saying that in a larger proportion of instances, sleep would take place spontaneously at an earlier period, and the subsequent condition of the patient be much more sound and safe, by doing nothing at all, than by the use of opiates. I have seen very decided cases of the disease recover well when a mere placebo was given with a view to keep up the appearance to friends of something being done, and prevent them from using as remedies things which would be hurtful. Dr Ware of Boston, in an excellent memoir on delirium tremens,[1] strongly advocates from experience the do-nothing plan of treatment. Among other things, he says of opium:—

[1] Quoted in the British and Foreign Medical Review, vol. xxiii. p. 603.

"In the cases which I have formerly treated with opium, and which have at last terminated well, a salutary sleep has not taken place till the close of the third day, let the quantity of opium be what it would. I have, indeed, seen sleep induced by opium at an earlier period, but it was premature, it passed into a state of coma, and the patient died. I am satisfied, therefore, that in cases of delirium tremens, the patient, so far as the paroxysm alone is concerned, should be left to the resources of his own system, particularly that no attempt should be made to force sleep by any of the remedies which are usually supposed to have that tendency, more particularly that this should not be attempted by the use of opium."

Dr Cahill[1] also cites several cases of the genuine disease, in which he found opiates decidedly injurious, and treatment without them salutary.

The treatment recommended by Dr Graves,[2] to which I have already alluded, is advocated on the ground that opium is highly dangerous in the early part of the paroxysm. His rule of practice is to begin with tartar emetic alone, with the view of combating vascular excitement, then to add a little opium, and gradually to increase the quantity, keeping its action carefully guarded and controlled by the antimony, until at length when engorgement of the cerebral vessels is no longer to be apprehended, to use opium alone. If opium is to be given at all in delirium tremens, this is certainly the safest mode of prescription. For some time I tried it, but from previous experience of the beneficial effects of antimony in this disease,[3] I soon became convinced that it was from that agent solely, especially its effects in the first stage, that ultimate benefit was derived; that the relative quantity of opium employed at first is too small[4] to counteract the power of the antimony, or to produce any notable effect whatever; that in ordinary cases, ere the time arrives for increasing much the amount of the opium, the

[1] Dublin Medical Journal. Observations on the Treatment of Delirium Tremens without Opium. Vol. xv. p. 397.

[2] Clinical Lectures, vol. i. p. 530.

[3] This experience of the effects of tartrate of antimony I had before I was aware that Dr Graves had recommended it with opium, or that Stoll, Göeden, Klapp, and others, had advised it in emetic doses.

[4] Dr Graves' formula for first use is:

℞ Antimon Tart. gr. iv.
Tinct. Opii. 5i.
Aquæ 3viij.

Signa. A tablespoonful to be taken every second hour. There is thus in each dose only five drops of laudanum to ¼th grain of antimony.

affection has run, or nearly so, its natural course, and the period for the salutary sleep commencing is at hand; and that when a greatly increased dose is given before this much wished-for change has arrived, there is a proportional increase of excitement and consequent delay of its occurrence.

From these considerations, I resumed the use of the antimony alone; and, during the last ten years, have treated upwards of eighty cases of the genuine disease, many of them very severe ones, with uniform success,—not only in regard to the speediness of the immediate recovery, but the comparatively thorough restoration to a healthy condition of body and mind;—as much so, at least, as could possibly be expected in individuals, many of whom had been, and were likely soon again to become, habitual drinkers. The dose which I have been accustomed to give has ranged from one quarter to one-half of a grain, in simple solution, every two hours, sometimes at shorter intervals, according to the degree of excitement and irritability. The action of the antimony appears to be chiefly sedative. Its direct influence is in reducing the vascular excitement of the brain, soothing the nervous system, and diminishing muscular power; and its more indirect action is exerted on the functions of the skin, kidneys, and intestinal canal. In two or three instances only have I found it necessary to suspend its employment, in consequence of diarrhœa and hemorrhagic discharge from the bowels; and in these cases digitalis and ipecacuan were substituted with marked benefit; and I do not recollect of ever seeing it produce continued vomiting, although occasionally I have found the first or second dose eject from the stomach a quantity of bile. It is for the sake of its emetic effect that, in Germany and America, it has been prescribed in large oft repeated doses, even from four to seven grains every hour, and that, too, according to report, with benefit.[1] But although there is, doubtless, extraordinary tolerance of this agent in delirium tremens, I do not think that the use of such, or any other very heroic means, are warranted. Bleedings, large opiates, or large doses of tartar emetic, are all, although certainly not equally, unsafe, and therefore to be deprecated. An antimonial course of treatment in moderation, and with the design I have indicated, gently diminishes excited action, induces weariness of muscle, general nervous exhaustion, and mental

[1] Quoted in the work of Höegh Guldberg, already referred to. See also British and Foreign Med. Rev., vol. vi. p. 330. Also Copland's Dict. of Pract. Med., p. 501.

languor. It thus removes all hindrances to the occurrence of the salutary sleep. It prepares the way for it, not by forcing, but by favouring it; and when the individual, exhausted, seeks his couch, and finds repose, that goes on, not as a drugged sleep, but as a purely natural and profound repose, from which he awakes with restored reason and muscular control.

Although I have recommended the tartrate of antimony as a chief remedy in delirium tremens, there are several other means essential to its successful treatment. In the department of medicinal agents, however, I have only further to suggest, that, should the bowels not be moved by the antimony, the compound powder of jalap (ʒi) will generally be found speedy and efficacious. The other means of cure belong strictly to regimen and diet; and the first of these in importance is bodily freedom. Nothing is more hurtful in delirium tremens than restraint, particularly that of the strait-waistcoat. I have seen instances, and heard of many more, where the cerebral excitement was so increased by the never-ceasing maddening struggles for liberty, that fatal convulsions at last afforded release. All the control required is the presence of one or two judicious attendants, who will humour the patient in his whims and fancies; who will speak and act regarding them so as to assure him of safety, and to relieve him of apprehension, which is the most characteristic feature of the delirium; and who will mildly but firmly interpose, if he attempts anything which may accidentally prove injurious to himself or others. Of course injury inflicted wrathfully or vindictively is not to be anticipated, for rage, violence, or outrage, do not occur in this remarkable disease, but only in that affection which I have already briefly noticed, and with which it is sometimes confounded, namely, the madness of drink. Hence the frequent accounts met with in the public prints, of homicidal, suicidal, and other violent acts, said to be perpetrated during fits of delirium tremens, originate in an entire misapprehension of the nature of the two diseases. The apartment, however, in which the delirium tremens patient is confined should be well secured, for he may rush out at the door, or jump over the window, in the fright and frenzy of imagined danger. The larger, too, the room is, so much the better, that he may have ample space to advance and retreat, according as he wishes to scrutinize or avoid a suspicious or distressing object of his fancy; to arrange and re-arrange articles of furniture; or to carry on, after a fashion, the duties of some bustling occupation.

All this expenditure of muscular effort, without any restraint, aids greatly the antimony in producing a safe kind and amount of physical and mental exhaustion, from which the patient, languid and worn out, at last lies down voluntarily, and falls into the much-desired sleep. It is thus, too, that "the walking drill," according to Dr Blake's experience in the West Indies,[1] was found efficacious in warding off attacks of delirium tremens in the case of drunken soldiers; not, however, as supposed, from the exercise proving a new stimulus in place of the rum, to which they had no access, but from its wearing-out effect, while the proper nutrition of the body was maintained. No one would ever think of ordering continued and monotonous hard work, and muscular fatigue, for an affection of "exhausted nervous power."

During the entire paroxysm of the attack, it is of some consequence to afford the patient abundance of light; not, however, as supposed by Dr Blake,[2] on account of its stimulant or excitant effect, but for its aid in correcting false optical impressions. The excited brain is very apt to receive erroneous impressions from the appearance of surrounding objects, if there is an uncertain light. Hence the exaggeration of many of those agitating and terrifying illusions and phantasms which more distinct vision would prevent, or quickly dispel. During the daytime, therefore, there should be no half-closed shutters, nor half-drawn blinds or curtains, but advantage taken of the clearest light available; and during the evening or night, the more distinct the artificial light is so much the better. Perhaps perfect darkness may serve the purpose equally well; but this can be available only in the well-padded chamber of a lunatic asylum; and, besides, in private practice, the other parts of the plan of treatment here recommended, which requires the presence of an attendant to regulate the doses of antimony, or other sedative, and to administer, from time to time, suitable nourishment, could not be carried on without the admission of light. This leads me to remark, in conclusion, that, during the administration of the tartar emetic, I give, at intervals of a few hours, a moderate quantity of good beef-tea, mutton broth, or chicken soup, and sometimes *café au lait*, with the white of an egg switched up with it. Thus, while the vascular action in the brain is being subdued, and the nervous system liberated from the presence of the alcoholic poison, the functions

[1] Op. cit., p. 19. [2] Op. cit., p. 60.

of organic life are sustained, and a better ultimate recovery is secured.

I shall now conclude this paper by appending reports of nine cases, in order to illustrate the views advocated in regard to the nature and treatment of delirium tremens, and these I shall give in detail, so that it may be acknowledged that the true disease has been understood and described. These cases, too, it must be borne in mind, are not complicated, or the symptoms masked by the effects of stimulants and opiates—unless in the instances of the unauthorized supplies hereafter to be noticed. They may probably, therefore, on first consideration, not appear to be remarkably severe instances of delirium tremens. They are indeed, however, most characteristic examples of the affection,—five of them at least being considerably above average severity; and I have no hesitation in stating my conviction that all of them would have assumed a more severe complexion and taken a more serious course, had the ordinary remedies been employed; nay, that it is the stimulo-narcotic plan of treatment alone which makes this disease in almost any case appear a formidable one. The following cases also are not selected; for, with three exceptions, they all occurred consecutively at a period —some years since—when I purposed bringing my views before the profession; and the others have occurred in succession also, and were noted with a view to publication; and, but for extending this communication to an undue length, I could have given the details of many more examples of the disease and its treatment.[1] Enough, however, I trust, has been brought forward to show, that delirium tremens is a form of alcholic poisoning—or an alcoholism; that its exciting, as well as its predisposing cause is the habitual abuse of intoxicating liquors—and not the sudden abstraction or diminution of accustomed stimulants; that these produce a specific form of irritation of the brain and membranes, the tendency of which is to arachnoid inflammation; that this takes place most readily in those who have a highly sanguine temperament, and a nervous irritable disposition; that the chief phenomena attending this disease are invariably uniform in their character, and distinguish it from every other

[1] As the individuals, whose cases are noted in Nos. I., II., III., IV., and V. were affected repeatedly with delirium tremens before or since the occasions described, amounting in all to twenty-three times; and as they were all these times successfully treated on the plan now recommended, the cases now brought forward may be considered as thirty-two in number, instead of nine.

affection; that the occurrence of the salutary sleep is the normal termination of the paroxysm, indicating diminished activity of the cerebral circulation and functions, and the commencement of convalescence; that the cordial and opiate treatment is generally pernicious, and frequently dangerous; and that the main indications of cure, are, to reduce the cerebral excitement by a moderate but decided and steady course of antimony, or other agent capable of exerting a somewhat similar influence, and thus favour—not force—the wished for sleep; to soothe the feelings and dissipate the fears of the affected by kind and judicious superintendence, and the permission of light and liberty; and to support the physical strength by a moderate allowance of animal nourishment.

CASES.

Case I.—An innkeeper, aged 43.[1] A habitual drinker, but seldom or never intoxicated, has been attended by me in eight different attacks of delirium tremens—more or less severe—within six years; and on all the occasions treated successfully without stimulants or opiates. For some time previous to the present attack he is said to have been drinking less than in former years, in consequence of ill health, and still less within the last fortnight, although every day, early and late, imbibing a little with his customers. On my *first* visit I found him very dull; without appetite; sleepless; complaining of slight cough and pain in the chest; tongue slightly furred; bowels constipated; pulse 90. As yet no visions, and no decided tremors. Calomel gr. iv. Pulv. Jalapæ, comp. 3i., ordered.

2nd day.—Bowels slightly opened; cough troublesome, but no bronchitic signs. In other respects same as on previous day. On account of the catarrhal symptoms ordered a mixture every four hours, each dose containing, Sol. Mur. Morphiæ ♏ 5, and Liq. Acet. Ammon. 3ij.

3rd day, noon.—Paroxysm of delirium tremens evidently begun. Had passed a restless night. There is considerable tremor and agitation of manner. Pulse 94, full, but soft. Discovered that he had been getting a little wine and spirits from time to time during the last three days. Forbade everything of the kind, and withdrew also the mixture ordered yesterday.

4th day, 10 A.M.—Pulse 106, soft and small; hands and tongue very tremulous; face pale; perspiring copiously. Could not stand still for a moment, but darted from one window to another, as he anxiously expected the police to come for thieves, who, he said, were tied up in the next room. They had been stealing his property for the last six days, and he had just been writing down a list of the articles amissing. This inventory he showed to me, but it was utterly unintelligible. He knew me at once; and answered correctly questions when this could be done in four or five words. It appeared that he had been in bed, but was sleepless, tremulous, and agitated, until 3 A.M., when he was seized with what his wife described as a fit. She then gave him a wine-glassful of brandy, and ever since he has been up and much excited,—running about the house after imaginary rats and thieves, and once escaped to the street in his night-shirt publishing his wrongs. A trustworthy attendant was now placed over him with instructions to see that he had as much freedom and light as possible. All stimulants prohibited, and the following mixture ordered: —℞ Tart. Ant. gr. iv. Vini Ipecac. 3iij. Aquæ 3vij., 3v.—a table spoonful to be given every two hours. A cup of tea, café au lait, or beef-tea, to be given at a few hours' intervals, if cared for.

8 P.M.—Pulse 96. Bowels twice moved since the morning; perspiring copiously; and much calmer, although still talking in a rambling manner concerning all sorts of difficulties and troubles. A cupful of beef-tea twice taken. The antimony has been given regularly, and is to be continued until there should be an appearance of depression, and a wish to go to bed.

5th day, 11 A.M.—He had his last dose of tartar emetic at ten o'clock last night, and soon thereafter appeared exhausted, and was prevailed on to go to bed, when he slept profoundly from 11 P.M. to 5 A.M. He had then some tea and bread, and had been sleeping again until now. Is dull, but quite rational; pulse 70, soft and regular; skin moist; bowels once purged. To be kept very quiet, and to have nourishment as formerly directed.

6th day.—Had slept the most of previous day, and all the last night; is now quite convalescent, although weak.

Remarks.—A good example of an ordinary case of delirium tremens; reduction of accustomed stimulants from inability to take more; aggravation of all the symptoms from (unauthorised) administration of stimulants, and probability that the disease would have taken a more severe form had these been continued; early and decided improvement under treatment with antimony, animal nourishment, and careful watching; the patient a living proof of the safety of a non-stimulant and non-opiate treatment from good recovery under so many attacks of the disease—the more frequent the recurrence of delirium tremens, the greater being generally supposed the danger.

Case II.—A spirit-dealer, aged 48. Long an habitual drinker. His average daily amount for some time had been four gills of whisky and one bottle of beer, taken from early in the morning until late at night; and there had been no diminution in the quantity previous to the present seizure. Had

[1] For obvious reasons the names—even the initials—of the individuals, whose cases are detailed are not given, or the dates of attack mentioned.

slept very little for a week, and none at all on the last two nights; and for some days was very tremulous, and quite unable to transact business.

1st day's visit, 3 P.M.—Was very distressed and agitated during the last night,—walking constantly up and down through the house, terrified with visions; had his last glass of whisky at 11 this forenoon. Pulse 104, small; skin cool and clammy; great muscular tremor; tongue foul; eyes yellow and lustreless; mind constantly occupied with false and horrific impressions of all kinds, although in no very definite form; but can answer a question put directly to him. *Instructions*—Plenty of light; complete liberty to promenade through the house, the doors and windows being secured; and two intelligent men to attend and humour all his fancies. To have a wine-glassful of the following mixture every two hours:—℞ Tart. Ant. gr. iv., Infusi Quassiæ et Aquæ ā ā ℥ss., whether it sickened or not, and only to be discontinued if he should go to sleep. Beef-tea and weak coffee with milk to be given occasionally. 8 P.M.—Took one glass of the mixture at 3.30 P.M., which caused vomiting of a quantity of bilious matter; one at 5 o'clock, which was followed soon after by a loose alvine evacuation; and one at 7 o'clock. He is at present pale and perspiring; very tremulous and restless—in constant apprehension of rats and strange men; quite sensible when spoken to; pulse 110. To have the mixture only every third hour. Beef-tea, etc.

2d day, 10.30 A.M.—Pulse 106, very small; perspiring freely; face very pale; urine scanty and high-coloured; great tremulousness. He can put out his tongue, or rise up, or sit down when desired, but that is nearly the amount of his intelligence. He is in constant motion, not rapid or boisterous, but chiefly busy in arranging bed-clothes, carpets, small articles of furniture, and sweeping imaginary crumbs from off the table. Had never been in bed, and had taken only three doses of the mixture since I saw him last. Took a glass from me, supposing it to be pale brandy:—no sense of taste. The mixture to be continued regularly. Was seen by my friend Dr Cappie at 3 P.M., and again at 9 P.M., who found him much the same as when last reported. Had been purged several times. Antimony, etc. continued.

3d day, 2 P.M.—In bed, sound asleep; pulse 84, of good character; a good deal of subsultus tendinum; skin very moist; paleness of countenance gone. It was stated that he had appeared very much exhausted last night about 12 o'clock; was then got to bed, fell asleep almost immediately, and did not awake until 7 this morning. When awake he was not quite sensible, but took some bread, coffee and milk, and fell asleep again. Continued so for other two hours, and was then perfectly coherent, but not inclined to speak. He had some more breakfast and an egg, and went to sleep again. An hour ago he was awake for a few minutes, and took some beef-tea. The antimony had been given once this morning:—to be discontinued. Nourishment only to be offered when he awakes.

4th day.—Found him quite well; mind perfectly clear, and had been able to read a little.[1]

[1] This patient has since had another attack—not quite so severe, originating, without any diminution of habitual supplies, and successfully treated in the same way as on the former occasion.

Remarks.—An ordinary case of the disease, rather more severe than Case I.; no suspension of wonted libations up to the period of seizure; excellent illustration of the *modus operandi* of the tartar emetic; and also of the benefit derived from the other means recommended for sustaining the organic functions, and bringing about natural sleep.

Case III.—An engineer, aged 30. Had been twice formerly under my care in delirium tremens, and recovered well without the use of stimulants or opiates. Has been drinking largely and constantly for some months past, and exhibiting at times excessive irritability and violence of temper—even to the extent of threatening the lives of his wife and children. In apprehension of this disposition he was some time since treated for delirium tremens in the Morningside Asylum; but on what plan I do not know, save that he was confined in a dark chamber. On the present occasion, at my *first visit*, I found that he had been drinking up to the moment of his attack, which had commenced decidedly two days before. His pulse was 110, soft, and of tolerable strength; hands very tremulous; aspect extremely haggard; skin moist; tongue clean. He had been quite sleepless for two nights; but not violent in his manner or conduct. He was laughing and talking incoherently,—looking constantly under his pillow, and carrying on a conversation with imaginary beings underneath, in this way,—"aye, oh yes, yes, certainly, just so," etc. On requesting to know what the devils were wanting, he replied, "a glass of whisky." Prescribed—℞ Tart. Ant. gr. vj. Aquæ ℥ij. Solve. A wine-glassful every two hours; and desired that he should be closely watched, and kindly treated. To have some weak beef-tea occasionally.

2d day.—Had a few minutes sleep this morning, but his general aspect is in all respects worse. Pulse 116. He is very restless and agitated, wishful to get out; thinks his workshop is on fire; that the police want to get hold of him, and has many such like fancies. I discovered that a bottle of table-beer had been given to him this morning. A wine-glassful and a half of the antimonial solution to be given every two hours.

3d day, 11.30 A.M.—He had walked about all yesterday in a state of great excitement; got the antimony very regularly, and lay down for the first time about 10 P.M., when he fell into a sleep. This continued until 6 A.M., when he awoke quite collected, and has since continued so. Pulse 80; hands very tremulous; has taken a good breakfast, and is in all respects apparently convalescent.

4th day.—Quite well.[1]

[1] Since the above occurred, this patient, on account of domestic calamities, was removed to the Royal Infirmary, under another attack of delirium tremens, where he died in an epileptic seizure. I understand that the plan of treatment practised in that instance was the one usually followed—namely, restraint in a strait-waistcoat, stimulants, and opiates.

Remarks.—A well-marked case of delirium tremens occurring in an individual in whom the *delirium ebriosum* might rather have been expected; but the long-continued course of intemperance gave to this attack the usual characters of cerebral alcoholism. The case also shows the liability to this disease without diminution of the wonted stimuli, and the tendency to aggravation even from a slight stimulus, such as table-beer; it also illustrates the singularly sedative effect of antimony.

Case IV.—A gentlemann, aged 40, of highly sanguine temperament and nervous, but kind disposition. Has had two previous attacks of delirium tremens under my care, both very severe. Was a total abstainer for some time following his last illness, having been informed that he would not probably survive another attack, or if so, that in all likelihood he would become insane; but had been gradually led back to drinking habits through company. His digestion having soon become so impaired that he could not take substantial nourishment, he drank systematically to overcome the distressing sensations of sinking. Four days prior to my first visit he was unfit for business; had disturbed wakeful nights; was very tremulous and nervous—being aware that delirium tremens was approaching, and fearing that he might be deprived of reason; and he had lost entirely the appetite—even for drink. Some brandy, however, had been given to him on the previous day, and his bowels were well cleared out by laxative medicine.

1*st day, afternoon.*—Found him very much agitated, and talking quickly and incessantly. He was hearing sounds which reproached him as a bad man. Thanked me for visiting him, saying that he was undeserving of notice, having been such a rogue, and so cruel to his wife and children. Pulse 100, soft and full; skin warm and moist; countenance pale and anxious; tongue and hands very tremulous. ℞ Tart. Antim. gr. iv., Aquæ font. et Aquæ Cinnam. ā ā ʒiij. Sig. One tablespoonful every two hours, or oftener should there be more excitement.

2*d day*, 10.30. A.M.—Had several times last night a few minutes' sleep; pulse 104; tongue white. He is at present more excited and restless; looks on himself as a lost man; is constantly hearing strange sounds; and is every little while eagerly examining the corners of the room. An experienced male attendant now placed over him with usual instructions. Antim. continued.

8 P.M.—Much the same. Mind more agitated with regret, and the apprehension of some impending calamity.

3*d day*, 10 A.M.—Has spent a sleepless and agitated night. His aspect is very anxious and apprehensive. Considers himself "between the deep sea and the devil; in fact, too bad for the devil himself." Pulse 108; tremor great; tongue cleaner; bowels confined; sweating considerably at times; urine scanty and high coloured. Tartar emetic continued; and to have Calomel gr. iv. Pulv. Jalapæ Comp. ʒj.

7 P.M.—Pulse 110, smaller than formerly; bowels have been twice opened. He is at times moving rapidly about as if searching for something; now whispering as if aware of some secret; and then again standing gazing at the floor bowing and scraping, and answering questions as if before a high tribunal. Already 12 grains of the tartar emetic have been given. To have now half-grain doses every two hours, or oftener if he becomes more excited. Also to have some beef-tea occasionally.

4*th day*, 11 A.M.—Had passed a violent night; fancied robbers were in the house, etc., and occasionally he shouted so loud as to be heard in the street. Had a large basinful of beef-tea at one time, and some coffee and milk at another, during the night. Between seven o'clock last night and seven this morning he had taken 6 grains of antimony. Shortly after the last dose he fell asleep, and has slept until now—four hours—awakening quite sensible. His look is now free from apprehension, but haggard and as if worn out. Voice and manner wonderfully firm. Pulse 96, and of good character. To have beef-tea, and no more antimony.

8 P.M.—Is again much worse. He is lying on the sofa sullen and dejected, with a very maniacal aspect, and declaring that he was the worst of men and doomed to die. It appeared that he had gradually been getting worse since the forenoon. His keeper and friends supposing him beyond risk, had allowed several acquaintances to visit him; and it was my firm conviction at the time, that a stimulant had been administered by some one. The half-grain doses of tartar emetic were ordered to be renewed.

5*th day*, 11 A.M.—Had wandered from room to room all the past night. Thinks that the newspapers contain a great deal about him, and that various enemies are plotting his destruction, etc. Pulse 98, firm; tremor inconsiderable; perspiring freely; bowels opened twice during the night. Antimony to be continued.

10 P.M.—Was very calm about noon; sat for some time in the parlour with his wife, reading the newspapers, and kissed the children. He then again became much excited. I found him standing in an attitude and with an expression of reverential awe, arms extended, body slightly bent forward, and eyes turned upwards. His language was as if answering questions put by his Maker, such as, "Yes, Almighty God."—"Dr Peddie cured me of my fever, Almighty God."—"M.D., Almighty God," and so on. Having sat down for an instant I was implored with an expression of deep alarm and concern to get up, otherwise I would be killed on the spot. Pulse 118, not so firm as in the morning; skin moist; tongue pretty clean; bowels twice moved. Had taken tea and some soup. The antimony to be continued.

6*th day*, 10 A.M.—Had passed a very restless night, having had altogether only about half an hour of sleep, from which he has newly awoke. Lying in bed with his clothes on, pretty calm. He said, "Don't speak to me, as you little know what a bad man I am. The sooner I am out of the world the better, for I am to be publicly whipped through the streets to-morrow morning," etc. Pulse 92; not much tremor; tongue a little furred. Bowels repeatedly purged. On account of this looseness I withdrew the tartar emetic and substituted—℞ Vini Ipecac. Tinct. Digitalis, ā ā ʒss., 20 drops to be given every three hours.

9 P.M.—Had been much much calmer since 2 P.M. The last mixture to be continued.

7th day, 10.30 A.M.—Had slept soundly from ten last night until nine this morning. Is still gloomy and desponding. Digitalis, etc., to be continued at intervals of eight hours, and as much nourishment as he will take.

8th day.—Passed a good night, and is in all respects much better.

From this period he gradually improved. Sleep became more and more refreshing, and the mind stronger. In a week he was able to resume all his ordinary occupations; and has continued ever since—after a considerable lapse of time—temperate and well.

Remarks.—This case is an example of true delirium tremens at the outset, modified somewhat in the relapse, with symptoms of a maniacal character occurring in an individual apparently so predisposed, but not of the nature of delirium ebriosum. This maniacal relapse was in all probability occasioned by some improper tampering. It shows also the benefit derivable from ipecacuan and digitalis, when the tartar emetic begins to purge too much.

CASE V.—A journalist, aged 41. Had for many years been in the habit of Morphia eating. The ordinary quantity consumed was 9 grains of the solid Muriate per day, or ℥i. in the week. Had been under my care (over a period of several years) during seven previous attacks of delirium tremens, most of them severe, and on all occasions treated without stimulants *or opiates*. After each attack he abstained for a few days or weeks from morphia, but the necessity of fulfilling some literary engagement drew him again into the vice. His whole appearance was that of the confirmed opium eater, yet there never had been any tendency to delirium tremens so long as stimulants were abstained from. The occasions in which he indulged in intoxicating liquors to any extent, only occurred at intervals of many months. Begun by the excitement of the social board on some festive occasion, they continued for from two to three weeks, but never so as at any time to produce decided intoxication. The quantity generally taken amounted to three wine-glassfuls of spirits, and one pint of porter daily; and this short course of drinking invariably led to an attack of delirium tremens. While indulging in liquor, he had always found it necessary to diminish a little the habitual dose of morphia, on account of the nervous irritability and tremor which he soon began to experience.

1st Visit, 5.30 P.M.—Was made aware that the course of cause and effect has been the same on the present as on former occasions; and that for the last four days, feeling himself under all sorts of horrors and fancies, and unable to sleep, he had left off the morphia entirely. Last night, however, he had taken three grains, having been induced again to try if it would produce a composing effect, but instead of this, it made him, as he himself expressed it, "ten times worse." He is at present walking up and down in a most wretched condition. States that strange visitors had been all day talking to him in whispers; that his breath as it went in and out took the form of whispers accusing him of misconduct, which, he says, is little needed, as his conscience is sufficiently reproving. Feels as if he had two heads—the one conjuring up fancies, and

the other thinking and judging correctly. Asps are also crawling on his breast, and he cannot shake them off, etc. Pulse is 100, soft, and of very good strength; pupils contracted; tongue clean; no appetite. Tremors, not only of his hands and tongue, but of his head and whole body have much increased to-day; and his voice also is bleating and unsteady. Says he has slept none for some nights, and is much afraid of the one approaching. He also states he had vomited himself freely with some antimonial solution which he had beside him since last illness; and likewise that he had been well purged. Desirous of ascertaining whether any other depressing or sedative agent would answer as well as the antimony formerly employed, the following was prescribed:—℞ Tinct. Aconiti gtt. x., Aquæ ℥iv.,—a tablespoonful to be given every two hours, and some beef-tea occasionally.

2d day, 10 A.M.—Has spent a very restless night; had some short, but very disturbed sleeps. Asps and other reptiles are crawling in great multitudes about the bed. Appearance of countenance most wretched; skin slightly clammy, and of a dirty colour; tongue a little white; pulse 110, weaker than yesterday; urine scanty, and high-coloured; no appetite. Had taken during the course of the night one-half of the mixture (Aconite 5 ♏), but being unable to visit him frequently, so as to watch the effects of the medicine, and fearing it might prove too depressing for the circulation, I withdrew it, and ordered instead 10 drops of Ipecacuan wine, to be given every two hours, and ℥i. of the Pulv. Jalapæ Comp. at noon. To have also beef-tea.

4 P.M.—Much more excited and delirious; says there is a court sitting in judgment on the five senses, etc. Tremors are excessive, and he is sweating profusely. He confesses to me that his attendant, thinking him very weak, had at noon given him a wine-glassful of spirits. And on close questioning, also admits that he has some morphia in his possession, but will not say whether he has taken any, and will not give it up. To have 20 drops of the Vin. Ipecac. every two hours.

10 P.M.—Is rather better. Pulse 108. Says that he has had a great many strangers visiting him, pressing him very much to go out with them. Pupils contracted.

3d day, 10 A.M.—Has passed a most agitated night; walking from room to room incessantly; sometimes waving a white handkerchief from the window, under some notion of making peace with God. Has taken a little breakfast. He had gone to bed a little before my visit, and is lying with his eyelids half-shut, and squinting when they are open; is working with his hands in the air, as if arranging things, or seizing objects between the finger and thumb, or pointing a way. There are great general tremors, and considerable twitchings of the eyebrows. Pulse 110, and very weak. To have plenty of good beef-tea; also the Vin. Ipecac. to be continued.

4 P.M.—Pulse 114. Is sitting up in bed, very pale, and talking more humorously. A lady and gentleman, whose portraits are hung on the wall, have been speaking to him from out of their frames, and annoying him much, but he had discovered that by a particular wink of his eye, he could make the one jump into the frame of the other, and thus stop their discourse. His attendant delivered to me a small packet of morphia which was found in his

possession, a search having been made for it by my instructions; and he did not deny that he had been helping himself to a little from time to time. Ipecac. to be continued; also beef-tea, etc.

4th day, 10.30 A.M.—Had spent a very turbulent night. Is lying at present with a poker in his hand, with which he had been warding off supposed intruders. Now, however, he is asleep, and has continued so since 9 A.M., but is very distressed, judging from his moans, and the movements of the muscles of the forehead and eyebrows.

2 P.M.—Has just awoke after sleeping five hours. Pulse 84; hands very tremulous, and voice bleating; tongue cleaner. Mind confused, but no raving. Inclined to be quiet. To have beef-tea or a mutton chop, if he can take it. Discontinue medicine.

5th day, 10 A.M.—Has passed an excellent night. Pulse 80, soft, and of good character. Feels his head quite cool and clear; and talks intelligently on some favourite subjects of study.

6th day.—Greatly improved. Intends to walk out a short distance.

7th day.—Feels better to-day than he has done for months.[1]

Remarks.—The above case is an illustration of the opinion that morphia alone (or opium), will not produce delirium tremens, but that the combination with stimulants will very readily induce an attack—even a very short course of drinking. Here also it is apparent, that the morphia latterly produced a stimulating and injurious effect, so much so, that during the premonitory stage of the delirium tremens, the patient voluntarily diminished the dose; and that subsequently, its stealthy use protracted and aggravated the attack. I am of opinion that the ipecacuan did good (although, perhaps, not so much as the tartar emetic would have done); and that its effects would have been more apparent had spirits not been taken on the second day of the paroxysm, and morphia repeatedly.

Case VI.—A clerk, æt. 41. For a number of years a hard drinker, and for some time past indulging to a great extent, but said to have been very moderate during the last six or eight days, in consequence of general indisposition. Appears to have twice had delirium tremens. On the last occasion is said to have been very violently and dangerously ill for three weeks, and to have been treated with spirits and opiates.

1st day.—Pulse 93; tongue foul and tremulous, also considerable tremor of hands, and agitation of manner; countenance anxious; bowels much disordered; sleepless. To have ℞ Mass. Pil. Hydrarg. gr. iv., Ext. Colocynth. gr.

[1] This patient died sometime since apparently from an apoplectic effusion, without having been under any form of treatment. Upwards of two years had elapsed since the attack above described. The practice of morphia eating, however, was soon resumed, and was persevered in to the last.

vi. Misce ut fiant, pil. ij.,—for immediate use; and afterwards 30 drops of the Vini Tart. Ant. to be taken every three hours.

2d day, 6 P.M.—Was unable to see him earlier. Pulse 93, full, but soft; bowels have been freely purged. Slept none since last visit; has been very restless all day; and being himself apprehensive—from previous experience—of the approach of delirium tremens, has ordered the windows to be nailed down, and his razors removed. Antimonial wine to be continued. 9 P.M. Is now seeing objects in bed, and becoming more restless. Pulse 100, of good character. Tongue cleaner. Now to have the antimony in larger doses. ℞ Tart. Ant. gr. iv., Aquæ ℥ij.,—a wine-glassful to be given every two hours. Arrow-root and coffee with milk allowed. To have liberty to walk about the house; to have all his opinions and diseased impressions humoured as much as possible; and to have the advantage of clear light.

3d day, 11 A.M.—Says that he has had eight hours of excellent sleep, and feels quite well and comfortable. In reality, however, he has spent a very restless night; up and down through the house several times with a lighted paper, looking for thieves. Has been much quieter since the last dose of the antimony. Pulse 106; more tremor; considerable warmth and moisture of skin. Antimony to be continued, and beef-tea to be given occasionally.

4th day, 10 A.M.—Pulse 110, weak, and slightly intermitting; muscular tremor great; perspiring copiously; pupils large; face pale; urine scanty and high coloured; bowels open; no sleep; and very much excited with all sorts of fancies, although he can answer any question distinctly. 3 P.M.—In most respects much as he was in the morning. Pulse 120; skin clammy. He has been travelling all day along with his wife, and something or other has constantly been going wrong. Now, they are both (his wife to please him) sitting in bed with their knees drawn up to prevent water covering them, as it has got into a boat in which they are crossing a river, etc. To continue the antimony, etc.

5th day, noon.—Pulse 100, and steadier. Tremor not quite so great. Has taken some breakfast with relish, and more beef-tea. Has been in bed several times over night, but has only had one hour's sleep. At present is doing penance by walking on the floor barefoot. To continue the antimony, etc. 4 P.M. Is much calmer; pulse down to 96. In order that fatigue and consequent sleep might be produced, advised a half-hour's walk out of doors in charge of a friend. 7 P.M. Sent for hurriedly. He is much more excited—more than he has been for the last 24 hours; pulse 115; said that he saw his wife disposed of at a lottery a few minutes ago to another gentleman, which he considered most disgraceful, etc. He had been out walking for a very short time only; and although I suspected that liquor had been given to him by some one, I could not ascertain that this was a fact. Antimony now to be given in half-grain doses every two hours.

6th day, 11 A.M.—Has passed a very agitated night. A short time since made an attempt to get over a window, and knocked down a large flower box into the street in the attempt. Is now writing dispatches to the Duke of Wellington, as he thinks himself in a besieged fortress. The writing is mere scratches of the pen, no letters being formed. Pulse supposed to be about 120,

but the muscular tremor and tendinous jerkings are so great as to prevent its being correctly counted. Half-grain doses of antimony to be given every hour, and beef-tea liberally. 11 P.M. Shortly after last visit the excitement had begun to subside, and with each successive dose of the tartar emetic he was observed to become calmer and more rational. At 3 P.M. he was taken out by a hired keeper for a short walk, but the latter having shortly before had some drink, took his charge into a spirit shop to get more. Here the patient, although offered liquor, declined; but the attendant having drank freely, and being unable to take care of himself, the former had actually to help him home. Worn out by this exertion, he went immediately to bed—half-past 4 P.M.—and has slept soundly until now. He is quite composed and rational. Antimony discontinued.

7th day, 11 A.M.—In all respects quite convalescent. Had eight hours sleep over night.

8th day.—Left cured.

Remarks.—The above is an instance of a somewhat severe attack of delirium tremens, in which recovery took place under the use of tartar emetic, etc., at a much earlier period than on the previous occasions when the treatment was stimulo-narcotic. It is also an additional illustration of the fact that the accustomed stimulus is frequently diminished by the patient himself when the disease is forming and advancing, simply from a sense of inability to stand the same amount as formerly. It likewise illustrates what I have frequently observed, namely, a severe outbreak of excitement shortly before recovery commenced, but which must be unhesitatingly met with the antimony, perhaps in increased doses.

Case VII.—The last-mentioned patient having fallen back into habits of intemperance after the lapse of a year, was again seized with delirium tremens. On my *first visit at* 5.30 P.M., I found that he had been carefully treated and watched by an intelligent student of medicine who lodged in the same house, and who not only had observed the success of the practice pursued on the previous occasion (Case VI.), but had during the interval, by the timely use of the same means, cut short two threatened attacks of the disease. It appeared that he had been drinking very freely up to the premonitory period of this attack, three or four days ago, since which time he has had an aversion to liquor, has tasted none, slept none, and laboured—especially during the last two days—under considerable excitement of the usual kind. There had already been given to him, in one-fourth and half-grain doses, viij. grains of the tartrate of antimony. His pulse I found to be 120, weak and slightly irregular; skin clammy; hands tremulous; face pale, notwithstanding a considerable eruption of acne; pupils dilated; and the tongue furred and presenting prints of the teeth along its edges. His chief occupation was examining anxiously the corners of the room, presses,

etc., arranging articles, partly real, partly imaginary, and taking memoranda, which, however, were quite unintelligible. Advised a continuance of the antimony.

2d day, 1 P.M.—Pulse 120; considerable heat of head and skin, but with general moisture. Answers any direct question rationally, but immediately thereafter talks incoherently. Is greatly occupied looking after dogs and cats, who, he says, have got into the house. The urine is scanty and high coloured, coagulates readily with heat and nitric acid; and a drop evaporated spontaneously presents numberless beautiful phosphatic stellæ. Antimony to be continued; and beef-tea to be given occasionally.

10 P.M.—Pulse 110. Is not quite so agitated and restless, but still loquacious and fanciful. Has hitherto had the antimony at the rate of 4 grains in 12 hours:—to be continued at the same rate, and good beef-tea to be given from time to time.

3d day.—Did not see him to-day, but in the evening received the following report from my medical friend in attendance:—"After you left last night he talked for a little, but quieted down about 11 o'clock, went to bed shortly afterwards, and slept soundly until 5 o'clock this morning. On awakening he took some of the antimonial solution, drank freely of barley water, and fell asleep almost immediately. He awoke again at 8; and has had short naps during the course of the day. He complains a good deal of headache, and his eyes are dull and heavy, and water a good deal. There is a copious flow of urine, which is much lighter in colour. Pulse 88. No excitement or delusion, and he is inclined to sleep. I have been giving the antimony, but not so frequently of late."

4th day, 11.45 A.M.—Is quite convalescent but weak; urine still somewhat high coloured, but is unchanged by heat, although rendered slightly turbid with nitric acid; and on evaporation no phosphates are discoverable, but a large amorphous deposit of the urate of ammonia.

Two days subsequently I found him up and going about quite well, and the urine perfectly normal.

Remarks.—The above case is a well-marked instance of delirium tremens in which stimulants were freely taken up to the occurrence of its premonitory stage, when they were suspended in consequence of the constitutional effect of alcoholic accumulation having been established. The easy course which the disease afterwards ran, compared with that of the previous attack, was undoubtedly attributable to the prompt and decided use of the tartrate of antimony. The case is also interesting from the circumstance that during the paroxysms of the affection, the urine was found to be highly albuminous, and loaded with phosphates, a condition which is probably invariable in this disease.

Case VIII.—A wine and spirit merchant, æt. 42, long addicted to drinking

habits, not consuming much at one time, but imbibing upwards of a pint of spirits daily. He was attended by me, about a year before, in an attack of delirium tremens of considerable severity, but recovered well under the antimonial plan of treatment. There did not appear to have been any diminution in the amount of his supplies on the present occasion, but rather the reverse, up to the moment of my *first visit*, which was *at* 4 P.M. I found him in his shop, to which he had escaped from home, although apparently in the second day of the paroxysm, extremely excited, and busily engaged among boxes, bottles, and barrels, searching for lost articles. The delirium was of a very rambling and confused character, and his hands and tongue excessively tremulous. Pulse 110, and small; skin very dry; pupils large; face pale and having an anxious expression. Prescribed ℞ Tart. Ant. gr. vj. Supertart. Potassæ 3i, Aquæ fbij—a wine-glassful to be taken every three hours, or oftener, if there be more excitement. To be taken home and closely watched, etc.

2d day, 11 A.M.—Pulse 114. Tremors greater and more general, as if he was paralytic. Urine high-coloured and scanty. Has been wandering through the house all night in an extremely excited state, and, so far as was intelligible, under the apprehension of visits from burglars. Antimony to be continued every two hours, and 3viij. of strong beef-tea every six hours.

3d day, 11 A.M.—Pulse 80, and of good strength; tongue clean; urine like strong beer in colour, but more plentiful. Manner calm; mind collected; and altogether apparently quite convalescent. It was stated, that yesterday, about 4 P.M., he fell asleep, awoke about 6 P.M., took some beef-tea, immediately fell asleep again, and did not awake until 10 P.M. He then had more nourishment, and was again soon fast asleep, in which he continued until 6 o'clock this morning, when he awoke and took a hearty breakfast of porridge and milk. His reason was only now, however, found to be quite restored; for when awake at the former times, he still talked incoherently. Early this morning he had the last of the 6 grains of antimony ordered on my first visit. Quiet, and nourishing diet prescribed.

4th day.—Found him in all respects perfectly well—better than he had been for many months past, both physically and mentally, and now reading in bed and conversing intelligently.

Remarks.—A characteristic example of delirium tremens of average severity; occurring without any abstraction of the usual stimulants, even on the second day of the paroxysm; and yielding satisfactorily in almost 24 hours to the influence of antimony, aided by good nourishment and careful watching.

Case IX.—Formerly a butler, now a first-class lodging-house keeper, æt. 53. Florid complexion. Has drunk spirits for many years in a systematic way, beginning early in the morning; but confesses to much greater indulgence during several weeks past, and more especially two days before he came under treatment, when, as expressed by himself, he had been "much the worse of it" (intoxicated); and it appeared also that on this occasion he had partaken

largely of salmon, stewed rhubarb, and sundry other articles, which had disordered his stomach. At 5.30 next morning, he was seized with severe pain in the bowels, and soon thereafter with vomiting and purging. In the course of that and the following day, he had taken repeated doses of tincture of rhubarb, laudanum, and brandy, but without benefit, and I was sent for in the afternoon. I saw him at 7 *evening*. He was then much pained in the bowels and purged; tongue exceedingly white; thirst considerable; urine not passed for many hours; pulse 90, rather full and vibrating; and extremities cold. Fearing an attack of cholera, I ordered a large sinapism to be applied to the epigastrium, heat to the feet, 3vj. of castor oil to be given immediately, and an opium pill, 1 grain, after the first movement of the bowels. Three other opium pills of the same strength were prescribed, one to be given at intervals of from two to four hours, according as the diarrhœa should be more or less urgent.

2d day, 11 A.M.—Has had 4 grains of opium during the night, which has checked the diarrhœa; but he has slept none, and complains much of pain in the bowels, about which he is nervously anxious, fearing that some dangerous malady is in progress. His manner is considerably excited, and he is very restless. Pulse 100; pupils contracted. Another sinapism ordered to the epigastrium. I now perceived that an attack of delirium tremens was approaching, and regretted much that I had prescribed opium so freely.

6 P.M.—Pulse 116; tongue white; perspiring copiously; pupils still contracted; no return of diarrhœa, and does not complain of abdominal pain; hands slightly tremulous; considerable agitation of manner; voice weak; speech rapid and stuttering; and since the forenoon he has at times been talking incoherently, fancying that he saw strangers in the room, etc. To have ipecacuan wine ℥ xx every two hours, and some tea, or weak coffee and milk and bread for diet.

10 P.M.—Pulse 120. Symptoms of a paroxysm of delirium tremens confirmed. Is now out of bed, dressed, cheerful and active, as if there had been no previous ailment. Says that he is quite well, but that a number of "queer customers" have been visiting him, etc. To be strictly and judiciously watched, and the ipecacuan to be continued.

3d day, 11 A.M.—Pulse 103, feeble; pupils quite natural, but eyes dull and expressionless. Urine dark and turbid. Has not been in bed at all during the night, but busy arranging furniture and hunting rats, particularly two old fellows, which, he avers, are hiding among the bed-clothes. Continue the ipecacuan and give beef-tea occasionally.

5 P.M.—Pulse 116, rather firmer than in the morning. Was permitted to walk out for an hour in the afternoon, well attended. He is now much more excited, but not quite so tremulous. Is perspiring copiously, and very earnestly catching imaginary objects in the air, which, however, are no sooner secured than they invariably slip through his fingers, and are broken and lost, thus occasioning him great distress. Ipecac. etc. to be continued. A specimen of urine passed in the morning became turbid with heat, and on evaporation presented a considerable abundance of phosphates.

10 P.M.—Head bathed in perspiration; hands cold; pupils natural; pulse 112, and small. Urine high-coloured. Bowels not moved since early yesterday

morning. Is at present standing close to a wardrobe, where he says he has locked up in a small crevice "a female thief of the long-nebbed kind." To continue the ipecacuan, and to have a laxative dose of the Pulv. Rhei. Comp. to-morrow morning.

4th day, 9.50 A.M.—Perspiring much. Pulse 116, very weak, yet he is extremely active. Has not been in bed during the night, but going about very excited, and troubled with phantasms. Thinks he has been sentenced to be flogged on the Castle Hill in the forenoon, to the extent of fifty lashes; and is now shouting into the corner of a press "how many are on my side?" on the supposition apparently that a petition is to be got up to prevent the punishment from being carried into execution. Has had beef-tea at one time, and *café au lait* at another time this morning. The urine examined after this visit was clear, but high coloured, became slightly turbid with heat, and was found to contain numerous phosphatic stellar and penniform crystals.

5 P.M.—Pulse 120, weak; more tremulousness of hands than formerly, and some tendinous jerkings; and is more excited also than he has yet been. Every article of furniture in the room, even two very heavy wardrobes which might have been supposed quite beyond his strength, have been moved out of their places again and again; and he is now in the midst of his confusion, haggard-like, and bathed in perspiration, searching for seven children, who, he says, were sent from Newcastle. Ipecacuan to be continued, and nourishment as formerly.

11 P.M.—Has been sleeping quietly and calmly for the last hour, quite worn out apparently; by his exertions among the furniture, searching for thieves, dogs, and children; stopping up water pipes which had burst; replacing bell-ropes which had been pulled down, etc. During the afternoon he has had his shirt changed three times on account of the excessive perspiration. Had some minced collops in the afternoon, and some tea and bread in the evening. He has now had, within the last forty-eight hours, ℥i. ℥vj. of the ipecacuan wine. Ordered to be kept very quiet, so as to prolong the sleep if possible; and when he awakes, to have some ipecacuan and some strong beef-tea.

5th day, 11 A.M.—Last night had slept three hours, and then three hours again this morning; but between these periods he was as excited as formerly, and is now toiling as hard as ever among the furniture, which is piled up in the middle of the floor. He is covered with perspiration; looks very anxious and alarmed, and is very cross, contradicting whatever is said by his attendants. Pulse 120, and small; pupils somewhat dilated; urine still high coloured, but not so scanty, and only very slightly turbid with heat. To discontinue the ipecacuan, and to have instead, R. Tart. Antim. gr. iv., Inf. Quassiæ ℥iv., a tablespoonful every two hours; nourishment also as formerly.

11 P.M.—Worn out by exertion; he had voluntarily gone to bed about 4 P.M., when he fell at once fast asleep, and has not awoke since. He is now breathing calmly, and his pulse is 96, soft, and not so weak as in the forenoon. Has only had one grain of the antimony, which is now to be discontinued.

6th day, 10.30 A.M.—Has slept well all night, awakening for a short time about midnight, when he got some nourishment, and then slept again until lately. Pulse 74. Mind quite clear and composed, and hands steady. Feels the whole body stiff and aching from the fatigue which he has undergone.

7th day, 11 A.M.—Continues improving. Feels more clearness of head and general lightness of the system than he has experienced for a long time past. Is to be up in the afternoon.

8th day.—Was able to walk out, quite well, and in every respect capable of resuming his ordinary occupations.

Remarks.—The above is a very severe instance of *delirium tremens*—the paroxysm lasting about seventy-seven hours. It was a first attack, and resulted from habitual drinking and no diminution of supplies; but on the contrary, excessive indulgence up to the period when seized with the choleraic symptoms—which probably precipitated or hastened on the disease, aided no doubt by the doses of brandy, laudanum, and tincture of rhubarb, taken before my first visit, and by the four grains of opium afterwards unfortunately prescribed to check the diarrhœa. In regard to the phenomena presented by this case, and the effect of remedial means, I would remark 1st, That the contracted state of the pupil only lasted while the effect of the opium continued. 2d, That the tendency to perspiration usually attending this affection was much increased apparently by the ipecacuanha, but without proving injurious. 3d, That the urine was found during the paroxysms, to be albuminous, and to present phosphatic crystals. 4th, That the speedy improvement following the change to the antimonial treatment suggests the probability that the attack would have been shorter, had that agent been employed earlier, but which was withheld from the supposed risk of bringing back the diarrhœa. 5th, That very considerable weakness of pulse may exist along with astonishing capability for muscular effort. 6th, That muscular exertion may be permitted with safety, nay, even with benefit, as a means of inducing natural sleep; and 7th, That this mode of treating the disease does not lead to any subsequent debility, but on the contrary, holds out the best expectation of recovery, with a sound condition of the mental and physical powers.

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VERTIGO

A PAPER READ TO

THE NORTH LONDON MEDICAL SOCIETY,

APRIL 12, 1854.

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

UNIVERSITY MEDICAL SCHOLAR.

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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 δίνη 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 ἴλιγξ 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 acidulæ 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 sese 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. 400.

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: Et. 2, p. 38.
† Fait à l'Académie 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. 432.

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 lesioni 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 pédoncule."† 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. 498.
‖ 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: enceph: &c., 1826, p. 23.
† The Nervous System (by Bell.) Ed. 1844, p. 194.
‡ Thèse cit, and Longet's Traité.
§ De vi motoria, &c., p. 82.

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.
† Uber 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 *demon* 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 Brutorum. 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 rétina. 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. 269.
† 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 paraît 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. 90.

† 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.) Matteuci's Leçons 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 "symptômes 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 Médicine. 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 Bonetî Mercurius compitalitius, art. Vertigo, for numerous instances of these remarkable prescriptions.

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AN ACCOUNT OF THE

ARRANGEMENT OF THE MUSCULAR SUBSTANCE

IN

THE URINARY

AND CERTAIN OF THE

GENERATIVE ORGANS

OF THE

HUMAN BODY.

BY

GEORGE VINER ELLIS,

PROFESSOR OF ANATOMY IN UNIVERSITY COLLEGE, LONDON.

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Some careful dissections of the muscular substance connected with the urinary and generative apparatus of the human body have made known to me many facts with which anatomists seem to be unacquainted; and the desire to add some of these to our common stock of knowledge, so that inaccuracy in our anatomical descriptions may be removed, has influenced me in making the present communication to the Royal Medical and Chirurgical Society. I do not purpose entering on the consideration of the nature of the muscular fibre, and the characters by which this may be

recognised where its existence is doubtful. The microscope will not, therefore, be needful to decide on the correctness of what I advance. With a scalpel and forceps, and a good eye, or, at most, with the aid of a watchmaker's eye-glass of an inch or an inch and a half focus, the following statements may be verified.

Of the Muscular Substance of the Urinary Bladder.

In the bladder there are three more or less perfect strata of involuntary muscular fibres; viz., an external or longitudinal, middle or circular, and internal longitudinal or submucous. These three are united by a mutual interchange of fibres, so that neither can be detached from the rest without cutting through these intercommunicating parts. In each layer, as in all muscular tissue,[1] the fibres are collected into bundles, and from these, offsets pass to adjacent bundles, producing thus a plexiform condition of the muscular web, or a network with meshes of varying size. (Fig. 1 and 2 *a*.) And as fleshy fasciculi connect the different strata, the fibres on the surface in the external layer become deep in the middle one, and the opposite; a like change in depth is observable between the submucous and the middle fibres. In consequence of this blending of the several layers, contraction of one will call into action the others, and thus the whole fleshy wall will take part in producing diminution of the viscus and expulsion of the urine.

The *outer layer* consists of fibres directed longitudinally from the apex to the base of the viscus, and is best marked below, in front of the neck. Above, the fibres end in tendons which are inserted, for the most part, into the fibrous peritoneal covering of the top of the bladder; but some are prolonged on the urachus, and end as the rest. Below they are differently arranged before and behind; in front some are attached to the back of the pubes by the

[1] The arrangement, structure, ending, &c., of the involuntary muscular fibre are elsewhere described by me.

anterior ligaments of the bladder, whilst others are continued over the sides and upper surface of the prostate as far as its apex, and end by tendon in its sheath; behind, the fibres terminate chiefly by blending with those of the deeper layer and the prostate, though in the female they reach also the fascia investing the lower part of the vagina.

The bundle of fibres connected with the back of the pubes is about half an inch wide, and has been described by other anatomists. Fleshy fibres radiate from it; some reaching the prostate and the neck of the bladder, where they cross those of the opposite side, and the remainder diverging to the base of that viscus. From the position of this fasciculus it is most advantageously placed for raising with its fellow the prostate and the neck of the bladder; and its action being, doubtless, to elevate those parts, the term *levator prostatæ* might with propriety be applied to it.

The *middle layer* of circular fibres (fig. 1 and 2 *b*) is thickest towards the cervix vesicæ, where it forms the ring called sphincter, and communicates largely with the submucous and external strata. Where the urethra begins it is continuous without any line of demarcation with the fleshy fibres of the prostate (fig. 1 *e*) in the male, and with a corresponding band of circular fibres in the female. The so-called sphincter is only part of the general muscular layer.

The *inner* or *submucous* stratum, alike in both sexes, is much thinner, and does not form so complete a layer as the others. In the lower third or half of the bladder, the fibres are longitudinal in direction, and of tolerably uniform thickness (fig. 1 and 2 *c*); but higher up they are thin, and become oblique and scattered on the mucous membrane. Behind, below the openings of the ureters, this stratum receives an accession of fleshy fibres from the muscular coat of those tubes (fig. 2 *r*). At the neck of the bladder this stratum is continued into the submucous coating around the urethra; and as the fibres pass from one part to another they form the projection called "uvula vesicæ." The existence of this layer may be shown by dissecting it

from below upwards, after the manner indicated in fig. 1 and 2; the bladder having been previously hardened in a strong solution of salt, and then distended with cotton wool or tow, introduced through a small opening in the top.

The ending of the ureters may be traced by taking away the two outer vesical strata and part of the prostate, as in fig. 2. These canals pierce the outer and middle strata of the fleshy wall of the bladder, and the fibres of their muscular coat are disposed in both sexes as in fig. 2; viz., the internal, the most numerous, are directed transversely, and unite with the corresponding fibres of the opposite ureter; whilst the remainder join the submucous muscular layer of the bladder, and are directed obliquely downwards over the "triangular space" to the submucous stratum of the urethra.[1]

Muscular Substance of the Prostate.

The prostate is essentially a muscular body, consisting of circular or orbicular involuntary fibres, with one large central hole for the passage of the urethra; and another smaller, oblique opening, directed upwards below the former, for the transmission of the common ejaculatory seminal ducts to the central urinary canal. The few longitudinal fibres on the upper surface of the prostate, which are derived from the external layer of the bladder, can scarcely be said to form part of that body.

Its circular fibres[2] are directly continuous behind, without any separation, with the circular fibres of the bladder; and in front a thin stratum, about one thirtieth of an inch

[1] The ending of the ureters has been differently described by the late Sir Charles Bell, in vol. iii of the Transactions of this Society. A fleshy bundle, which he called muscle of the ureter, is described as descending from the uretral opening towards the neck of the bladder, where it joins the like part of the other side; and the two conjoined are said to be inserted by a tendon into the middle lobe of the prostate.

[2] The thickness, dimensions, and topographical relations of the prostate will be found described in the common text-books.

thick, is prolonged forwards from it around the membranous part of the urethra[1] (fig. 1 *e*), so as to separate this tube from the surrounding voluntary constrictor muscle. These facts seem to show the inappropriateness of the older view respecting the distinctness and glandular nature of the prostate, and indicate its being a portion of a muscular layer, which surrounds the intra-pelvic part of the urethra, and is continuous without interruption with the circular fibres of the bladder. Within, and quite distinct from the circular fibres, lies the tube of the urethra, incased by its submucous layer of longitudinal fibres (fig. 1 *d*). Towards the lower and outer aspects, the fibres are less firmly applied together, especially where the vessels enter; and they appear to be superadded to those which join the coat of the bladder.

As only so small a portion of the prostate is glandular, the propriety of calling that body a gland is rendered doubtful; for the small secreting glands contained in it are but appendages of the mucous membrane, which project amongst the muscular fibres in the same way as the other glands of the urethra extend into the surrounding submucous tissues. The glands are situate towards the base of the prostate, and more are below than above the urethral tube, especially in the part called middle lobe; their largest ducts enter the urethra opposite the opening in the circular fibres for the common ejaculatory seminal ducts.

In the female the urethra corresponds with the intra-pelvic part (prostatic and membranous) of that of the male, and is surrounded in all its length by circular involuntary muscular fibres, which are external to the submucous, and are continued, as in the male, into the middle stratum of the bladder. Thus, the posterior third of the urethra of the

[1] Professor Müller has described a layer of voluntary circular fibres around the membranous part of the urethra; but he considers it to form part of the constrictor urethræ muscle outside, and he does not show its connection with the prostate and bladder. See the treatise of J. Müller, 'Ueber die Organischen Nerven der erectilen Männlichen Geschlechts Organe des Menschen,' &c., Berlin, 1836.

male differs chiefly from the tube of the female in having the surrounding involuntary circular muscular fibre extremely developed at one spot in connection with special functions, and in being provided at the same spot with large secreting glands, for the purpose of increasing the quantity of the seminal fluid.

From the above-given anatomical facts, we may conclude that the prostate is less of a glandular than a muscular body, and is only a largely developed portion of the circular muscular layer that invests all the urethra behind the bulb or the spongy portion. The existence, too, in the female of a thin muscular stratum in the corresponding position gives support to the view of its muscular office. As the prostatic enlargement includes only part of the muscular stratum on the urethra, I would propose the name *orbicularis vel sphincter urethræ*[1] for both the prostate and the prolongation around the membranous portion of the urethra; whilst I would confine the old term prostate (without the word gland) to the thickened and more powerful part near the neck of the bladder. This orbicularis may be considered as only an advanced portion of the circular layer of the bladder, though it must have the power of acting independently of the vesical fibres, as, for instance, in the propulsion of the seminal fluid. Its chief office will probably be, to hurry on the semen, and deliver this into the grasp of the voluntary muscular fibres of the constrictor urethræ, which are external to it along the membranous part of the urethral tube.

Submucous Fibres of the Urethra.

A submucous stratum of longitudinal muscular fibres surrounds the urethra throughout its whole length, and is continued behind into the submucous layer of the bladder. It is strongest around the first third of the urethra (that

[1] Professor Kölliker applies the term *sphincter prostatæ* to some of the more internal circular fibres. He says the other fibres radiate from the centre to the circumference. ('Mikroskopische Anatomie.')

next the bladder), especially so in the prostate (fig. 1 and 2), and becomes gradually thinner as it proceeds towards the end of the penis; much fibrous is intermingled with the muscular tissue. At the fore part of the urethra its fibres end in tendons in the usual way, many of these blending with the submucous fibrous tissue. At the hinder third, that part embraced by the circular fibres of the orbicularis urethræ, many longitudinal fibres become oblique in direction, and are applied to those of the orbicularis. And at the neck of the bladder, other fibres join the circular stratum of that viscus, as is represented in fig. 1 and 2. In the prostatic part of the urethra, the central median crest or ridge (crista seu verumontanum) is formed by a bundle of this layer, whose longitudinal fibres separate to enclose the opening of the vesicula prostatica (sinus pocularis); and in the same spot the submucous stratum is joined by muscular fibres that accompany the ejaculatory ducts. No circular fibres have been recognised by me in the submucous layer of the urethra; where such external fibres appear after the removal of the longitudinal, they belong to other structures, viz., to the orbicularis muscle in the posterior third, and to the septum corporis spongiosi (to be afterwards referred to) in the anterior two thirds of the urethra.[1]

In the urethra of the female, the submucous fibres are like those in the male, and have a similar position and arrangement, blending behind with the circular fibres of the urethra and bladder; but as there are not any seminal ducts in this sex, the accessory bundle in the male is wanting. About a quarter of an inch from the anterior opening of the tube, the longitudinal fibres are collected into bundles, between which are openings of the submucous glands arranged in lines.

[1] I have not met with the circular fibres in this layer, which Professor Kölliker describes (Mikroskopische Anatomie). Still less could I verify the statements of Mr. Hancock respecting it. (Lettsomian Lectures, 'Lancet,' 1852.) Those who are acquainted with the descriptions of the writers referred to, will perceive that I have not borrowed my account from them.

Muscular Covering of the Vesiculæ and Vasa Deferentia.

A muscular layer covers and partly surrounds the vesiculæ seminales and the ending of the vasa deferentia. It will be brought into view, when the bladder is placed upside down, by removing carefully from the seminal vesicles their sheath of the recto-vesical fascia. It consists of one layer of longitudinal and another of transverse fibres; and the whole might be named, from its office, *compressor vesiculæ et ductûs seminis*.

The transverse fibres are the more superficial in the inverted position of the part; some stretch over the vesiculæ, and are inserted at each side into the investing fascia; but others, and these are the most numerous, reach only as far as the outer border of the vasa deferentia: by this arrangement both the parts of the seminal apparatus may be compressed. This stratum is thickest near the prostate, and joins in front the circular fibres of that body.

The longitudinal fibres are beneath the others, and form a less extensive plane. For the distance of half an inch behind the prostate, they give rise to a continuous fleshy layer over the seminal apparatus; and from this, fibres are continued forwards and backwards. The anterior offset surrounds the common ejaculatory ducts, and joins the submucous layer of the urethra in the prostate; and the posterior is continued along the vasa deferentia for a short distance, as well as along each vesicula, where it is strongest at the outer and inner margins.[1]

This muscular layer will compress and shorten the vesiculæ seminales, and the lower dilated ends of the vasa

[1] Professor Kölliker gives the following description of this structure: "Externally the vesiculæ are surrounded in part only by a membranous, and, in part, by an evidently muscular sheath, as at the hinder surface. This enters between the different windings of the canal uniting them together, and at the lower end passes as a broad muscular band from the one vesicula to the other." Mikroskopische Anatomie: zweiter band, p. 405.

deferentia. If the bladder is distended during the contraction, giving, in this condition, support to the seminal organs, the muscle will be enabled to act much more efficiently in the expulsion of the semen.

Sheaths around the Spongy Structure of the Penis.

The sheath investing the spongy material of the corpora cavernosa is commonly described as being composed of fibrous tissues, like those in tendons, without any special arrangement, except that they are said to be mostly longitudinal. My dissections demonstrate two layers of fibres in it, with a constant arrangement, one being superficial and having longitudinal fibres, the other deep, with transverse fibres, as in the wall of the alimentary tube. Moreover, the fibres have the same arrangement as in involuntary muscle, that is to say, they have a net-like disposition with small meshes.

In the outer stratum, where the fibres are directed longitudinally in bundles $\frac{1}{50}$th to $\frac{1}{25}$th of an inch wide, meshes about $\frac{1}{4}$th of an inch in length are left between the offsets that pass from bundle to bundle. (See fig. 3.) Towards the front of the penis the bundles and the meshes are smaller; and in the crus the bundles are whiter, apparently from a greater mixture of fibrous tissue.

In the inner stratum, which is rather thinner than the other, the fibres are disposed circularly or transversely, though with the plexiform arrangement before described; and they will be best seen by opening the sheath and removing the spongy substance, as in fig. 4. Passing circularly around the cavity, the fibres blend in the middle line with those of the opposite corpus cavernosum, and construct in this way the imperfect septum (sept. pectiniforme) along the centre of the penis. (fig. 4 *m*.)

Only circular fibres enter into the composition of the septum between the cavernous bodies, as before described; and the fissures in it, corresponding with meshes in the other

parts, are here greatly enlarged to permit the free communication of the vascular or erectile tissue at opposite sides.

Where the cavernous bodies terminate in points anteriorly, small bands are continued into the glans penis.

The sheath of the corpus spongiosum urethræ resembles that of the cavernous bodies in incasing the vascular substance, and in possessing like them a central partition; but it has only one layer of fibres. It is formed of thin circular or transverse fibres, as is the deeper layer of the corpora cavernosa, but near the bulb some of them are oblique. Towards the front of the penis the place of longitudinal fibres is supplied by the "fascia penis."

The septum is connected with the surrounding sheath, and reaches vertically, as in the cavernous bodies, from the upper to the under aspect of the corpus spongiosum. As it crosses the included space it incases the urethral tube with the submucous layer, a piece being continued on each side. Its length below the tube of the urethra, and the way in which it surrounds this canal, are seen in fig. 1 *f*, *g*. This partition exists throughout the whole extent of the corpus spongiosum, though towards the front it is very imperfect, resembling in this respect the representative part in the corpora cavernosa. In a favorable body, I have traced it from the bulb to the glans, but its connection with the outer sheath is very slight in front; commonly it will not be traceable as a distinct partition much more than two inches from the bulb. It is composed of vertical fibres with minute clefts, as is the septum of the corpora cavernosa. All that part of the glans penis which is reflected back on the cavernous bodies above the tube of the urethra, wants the median partition.[1]

The sheaths surrounding the vascular or erectile structures of the penis are, therefore, alike in their texture; but one is composed of two strata with fibres taking different directions,

[1] Mr. H. Thompson, in a 'Treatise on Stricture of the Urethra,' calls in question the fact of the septal process being continuous with the sheath; but I think he will allow the existence of this junction if he seeks it in the way represented in the drawing.

whilst the other has only a circular layer. Across the space included by the sheath in both the cavernous and spongy bodies is placed an imperfect partition, which is formed of circular fibres.

EXPLANATION OF THE FIGURES.

All the figures have been taken by the skilful hand of Mr. Ford from my own dissections.

Fig. 1.—For this figure the outer and middle strata of the muscular coat of the bladder, and the muscular layer around the prostatic and membranous parts of the urethra, were cut through in the preparation, so as to show the submucous stratum of the bladder and urethra. The corpus spongiosum was cut into, and the contained spongy material removed for a short distance to lay bare the septum. The same letters are used to indicate like parts in fig. 1 and 2.

a. External or longitudinal muscular layer of the bladder.

b. Middle or circular.

c. Internal or submucous. Many of its fibres are shown entering the circular fibres of the middle stratum.

d. Longitudinal submucous fibres of the urethra as they pass through the prostatic and membranous parts of that tube, internal to and separate from the circular layer *e*. At the neck of the bladder some are represented cut, as in the dissection, and others blend with the circular fibres.

e points to the circular fibres around the hinder third of the urethra, which I have named orbicularis urethræ: their continuity, behind, with the circular fibres of the bladder appears in the drawing.

f. The septum of the corpus spongiosum: the way in which this encloses, and shuts out the urethra with its longitudinal submucous layer from the spongy tissue, is referred to by *f*. Some apertures have been made in the septum to show the space on the other side: the piece of whalebone, introduced into the left half of the corpus spongiosum, appears through the apertures.

h. Corpus spongiosum urethræ.

k. Corpus cavernosum penis.

l. Ureter of the right side.

Fig. 2.—In the dissection here represented, after the separation of the vesiculæ seminales and vasa deferentia, which have been drawn forwards, the two outer strata of the muscular coat were taken away over the triangular space at the base of the bladder, in order that the expansion derived from the ends of the ureters might be seen joining the submucous layer. The lower half of the prostate was cut away in greater part, and the submucous layer of the urethra laid bare.

a, *b*, *c*. The different muscular strata of the bladder as in fig. 1.

d. The layer around the urethra in the prostate: the central ridge corresponds with the prominence in the floor of the urethra. Some of the fibres join the circular of the bladder, while others enter the deepest or the submucous layer of that viscus.

n. Vesicula seminalis and the vas deferens joining to form the common ejaculatory duct.

p. Membranous part of the urethra.

l. End of the ureter: some fibres from it, *r*, are seen joining those around the urethra, while others enter the submucous muscular layer of the bladder.

Fig. 3.—The longitudinal fibres and the meshes of the outer stratum of the corpus cavernosum are here indicated. The part selected for the drawing was not far from the crus penis.

Fig. 4.—The piece for the drawing fig. 3 was cut open, and the spongy tissue removed to make the preparation for fig. 4. It illustrates the circular arrangement of the inner layer of fibres of the corpus cavernosum; *m* points to the formation of the septum pectiniforme by those fibres.

Fig 2

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Fig 3

Fig 4

C.E.Root del. & lith.

W.West Imp.

ON THE

PHYSIOLOGICAL ACTION OF ATROPINE

IN

DILATING THE PUPIL.

BY

GEORGE HARLEY, M.D.,

TEACHER OF PRACTICAL PHYSIOLOGY AND HISTOLOGY, IN UNIVERSITY COLLEGE, LONDON.

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ON

THE PHYSIOLOGICAL ACTION OF ATROPINE

IN

IN DILATING THE PUPIL.

During the perusal of a paper of great merit (in the July number of the *Edinburgh Medical Journal*), on some of the cases treated at the Eye Infirmary,[1] I came upon a very ingenious theory with regard to the action of atropine and belladonna in dilating the pupil. After a lucid description, throughout which the physiological effect of these substances upon the iris, is ascribed, not to their producing paralysis of the third pair of nerves, which are supposed to supply the circular fibres, but to the excitation of the filaments of the sympathetic supplying the radiating fibres of the iris, the author proceeds to express a wish that the question "might be brought to the test of direct experiment by some one accustomed to such inquiries."

I happened, during last winter, to have made, at the suggestion of Professor Garrod, some experiments exactly analogous to those recommended in the same paragraph by Mr Bell, and considering that the question might be fraught with a certain amount of interest, especially as it would seem to have excited some discussion at a recent meeting of the Medico-Chirurgical Society, I beg to lay before your readers the mode of proceeding, and the results obtained from the experiments.

In the early part of last year Professor Sharpey made an experiment, in order to ascertain if atropine, when directly applied to the cervical sympathetic would cause dilatation of the pupil, and in the beginning of this year, Professor Sharpey and I repeated the experiment upon a cat, in the following manner:—

Experiment I.—The left cervical sympathetic nerve was carefully dissected out from the neighbouring tissues, for nearly two

[1] Report of Cases occurring at the Edinburgh Eye Infirmary. By Robert Hamilton, M.D., F.R.C.S.E., and Benjamin Bell, Esq., F.R.C.S.E.

inches in extent, and afterwards divided; the pupil of the eye, on the same side, immediately, or at least in a few seconds, became contracted, and permanently remained so. The upper end of the cut nerve was next suspended in a strong solution of atropine, and, notwithstanding that it was retained in the liquid during at least twenty-five minutes, no dilatation of the pupil occurred either then or throughout the day. This experiment I have again repeated with an exactly similar result, and I believe the same thing occurred in Professor Sharpey's first experiment. Atropine, thus certainly appears to have no direct stimulating effect upon the sympathetic nerve in the neck; for had it stimulated as galvanism does, the application of it to the nervous substance of the cervical sympathetic would have been followed by dilatation of the pupil.

Experiment II.—On another occasion, while performing an analogous experiment upon a cat, a drop or two of the atropine solution accidentally fell upon the exposed muscles of the neck, and in a short time after absorption had taken place, the pupils of both eyes became dilated, although in different degrees. The dilatation of the pupil in the sound eye occurred to such an extent, that scarcely more than the border of the iris remained visible, while the pupil of the eye on the side where the sympathetic was divided, became dilated *only to about one-half*, and remained in that condition during several hours. As the result of the first experiment negatives the idea of atropine exciting the nerve filaments supplying the radiating fibres through which the dilatation of the pupil is produced, we are forced to the conclusion, that in the present case, the semi-dilatation must originate, or rather depend upon, paralysis of the nerve supplying the circular fibres which govern the contraction of the pupil. We shall see in the sequel, in how far the phenomena observed in another experiment tend to confirm this view.

Experiment III.—The conjunctiva of the right eye of a dog was moistened with *a single drop* of the solution of atropine,—in half an hour the adjacent pupil became fully dilated, while the iris of the opposite eye was not observed to be in the least degree affected.

Experiment IV.—On another occasion, *several drops* of the same solution were at different times brought in contact with the conjunctiva of one of the eyes; and not only did the adjacent pupil, but also that of the other eye, become fully dilated.

The results of these three last experiments induce me to coincide with the theory advanced by Mr Benjamin Bell. According to his view, the atropine must have, in all three instances, passed into the circulation before making its presence known by its characteristic action upon the pupil. That in the local application in Experiment III., the quantity of the alkaloid employed, although extremely minute, was nevertheless able to reach the periphery of the nerves of the iris so

speedily, as to be still sufficiently concentrated to produce dilatation of the pupil; but that, by the time it arrived at the heart, and became diffused through the general mass of the blood, it was too much weakened by dilution to be capable of acting either on the root, in the course, or at the periphery of the nerve supplying the opposite eye with sufficient power to cause dilatation of the pupil. On the other hand, in Experiments II. and IV., the quantity of atropine (in the one case absorbed by the capillaries of the muscles of the neck, in the other by those of the conjunctiva and adjoining tissues) was sufficiently great, that, notwithstanding its being first diffused through the general circulation, it arrived at the nerves of both irises, in so concentrated a state, as still to be able to cause dilatation of the pupils. Whether in the latter two examples, the narcotic acted on the periphery, or on the roots of the nerves, it is impossible to say; but, certainly, in the case where the quantity of atropine employed was so minute as to have just sufficient strength to dilate one pupil, and that the contiguous one, we are constrained to admit the possibility of the atropine having directly paralysed the periphery of the nerve. The origins of the nerves supplying the opposite eyes, are so close together, that we cannot suppose, with any degree of feasibility, the atropine to have been transported by the general circulation, to the root of the nerve on which it acted;—had it been so, we must have had dilatation of the pupils of both eyes; for, it is natural to suppose, that a similar quantity of poisoned blood would simultaneously arrive at the roots of both nerves. In Experiment II., where the narcotic directly entered the general circulation, as well as in Experiment IV., where such an excess of atropine was absorbed by the conjunctiva, that not only the contiguous, but also the distant pupil, became dilated,—the supposition of the narcotic having acted upon the origins of the nerves, appears to be not altogether unwarranted.

Experiment V.—Into the left eye of a cat, whose pupil on that side had become permanently contracted in consequence of excision of about an inch of the left cervical sympathetic, a drop of the solution of atropine was allowed to fall. In a short time the adjacent pupil became *half dilated*, and, although more of the solution was afterwards added, complete dilatation could not be induced.

This agrees with "the case narrated by Dr Gairdner, in which contraction of the pupil was associated with an aneurism at the root of the neck, * * * and where repeated doses of belladonna, given internally, dilated both pupils. But, it was observed throughout the experiment, that the affected pupil continued smaller than the other," just as we have also seen in Experiment II.

Experiment VI.—The third nerve of a cat was divided at the point of exit from the sphenoidal fissure; the pupil on the same side immediately became dilated, and remained so. The addition of a

couple of drops of atropine solution was not observed to increase the dilatation of the pupil, as we expected, from the observation of Dr John Struthers, who noticed that in the human subject, belladonna acts upon pupils already dilated from some diseased condition of the third nerve. Perhaps in those cases where the narcotic is observed to increase the size of the already dilated pupil, some small twigs of the nerve remain incompletely paralysed. As no effect was observed to follow the application of atropine in the experiment just cited, I divided the cervical sympathetic on the same side of the neck. The iris gradually contracted, but not to the same marked extent as in the cases where the sympathetic was alone divided; in fact, the pupil remained permanently in a state of half dilatation and half contraction. The circular as well as the radiating fibres of the iris being paralysed by the section of their respective nerves, the contractile property of the muscular fibrillæ was brought into abeyance, and there could be neither on the one nor the other side an excess of action so as to produce either a condition of dilatation or contraction of the pupils. This, indeed, is an exactly similar condition to that which supervenes when, after section of the sympathetic, a solution of atropine is dropt into the eye. We have seen, both in the experiments performed by Professor Sharpey and by myself, that atropine can *not* cause dilatation of the pupil by stimulating the cervical sympathetic; and, I think we are justified in the present state of our knowledge, in continuing to attribute the influence of atropine upon the pupil, to its possessing the power of paralysing the third pair of nerves.

In a similar manner I would account for the effect of opium and other substances possessing the power of inducing contraction of the pupil, not to their stimulating the third pair, but to their paralysing the sympathetic which governs the dilatation of the pupil.

In conclusion, the foregoing experiments, I think, tend to prove:—

1. That atropine does not possess the power of dilating the pupil by directly stimulating the sympathetic nerve.

2. That to act upon the pupil it must, as Mr B. Bell says, first be absorbed.

3. That it can act, not only on the periphery, but also on the roots of the nerves.

4. The probable action of atropine or belladonna in dilating the pupil, depends on its paralysing the ciliary branches of the third pair of nerves, and not on its stimulating the filaments of the sympathetic, which supply the radiating fibres of the iris.

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FÆCAL FERMENTATION

AS A

Cause of Disease:

TOGETHER WITH

THE GENERAL RULES OF TREATMENT TO BE OBSERVED.

BY

C. H. F. ROUTH, M.D.,

PHYSICIAN TO THE ST. PANCRAS DISPENSARY; ASSISTANT PHYSICIAN TO THE SAMARITAN HOSPITAL; ETC.

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FÆCAL FERMENTATION,

etc.

PART I.

In the following paper, my object will be to direct renewed attention to a subject which had of late years been too much neglected, until Dr. Snow first recalled public attention to it. I allude to the fact that fæcal matters develope, under certain circumstances, a contagious poison, which becomes a powerful exciting cause of disease. I do not deny that those diseases to which I shall presently allude may be generated by the decomposition of other vegetable and animal matters, nor do I in any way assert that the fæcal poison is the *only one* which is in general operation; but I wish peculiarly to direct attention to this poison as a frequent cause in originating and continuing these diseases, and in afterwards rendering them fatally destructive. I also hope thereby to call the attention of our sanitary corps of officers to this source of disease, so that it may be tested by those facts, with which they will be sure to meet in the performance of their duties; and that both in regard to prophylaxis and treatment a broader and more general principle of action may be adopted. And because, in the same measures by which these fæcal poisons are withstood and counteracted, other vegetable and animal poisons are equally opposed and destroyed, so we may hope that the occurrence of many diseases, especially those of the epidemic and endemic varieties, may be prevented, or at least the *type* of such diseases may be rendered so much more benignant, that their treatment will become, under Providence, comparatively easy and successful. It is, therefore, with peculiar pleasure that I find the subject of malaria as a cause of disease in this country has been selected by the Council of this Society for the Fothergillian medal of 1858,

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and I also trust that among the number of chemical students in the nation there may be found one or more able and courageous enough to grapple with the subject, and to overcome the natural repugnance to make investigations in such a department of science. If so, we may hope that the specific and chemical nature of some of these poisons may be found out.

To go over the whole subject, even in a cursory manner, in one paper, is to me at least impossible. I can only, therefore to-night, consider some points connected with the formation of fæcal matters, their subsequent decomposition in the production of particular poisons, the general laws under which these, in common with other poisons, act, and, finally, their effects when partaken of in substance by the lower animals, or by man, when this can be done. The effects of their ingestion or absorption in solution in water, and of the emanations therefrom in the production of disease, and the rules of prophylaxis and treatment, must form the subject of a second and a third paper. I must, therefore, claim your indulgence for the necessary imperfection of these remarks, and ask you to look upon this paper as only introductory to others, which, I trust, may prove of more interest, and in which I hope to develope the subject more fully and more practically.

One other point, and I have done with preliminary remarks. You must not expect much originality in a communication of this nature. The merit of the paper must be based on the number of facts collected from all sources. Nay more, the collection of evidence of this nature, because it is not mine, is more worthy of your credence. Otherwise, I might be open to the charge of moulding my facts to my theory; not that I believe you would think me capable of doing so willingly, but, even with the most mathematical construction of mind, it is often difficult to escape all bias in our convictions. The facts, moreover, may be all easily examined by each of you for yourselves, as, wherever I can do so, I will give you chapter and verse for all I state. My honest wish is to get at truth, not to pervert it.

CHEMICAL ANALYSES OF FÆCES.

It is a remarkable fact that the analyses of excrement, and especially the human variety, as made by different observers, not only do not agree, but throw after all but a very little light on many interesting points in its composition. The best and the most recent analysis that I am acquainted with is that of Dr. Marcet (*Philosophical Transactions*, vol. cxliv, p. 265). From his inquiries, it would appear that fæces consist of—excretine, a peculiar animal matter; margaric acid; colouring matter like that of blood; a light granular substance, which he regards as a combination of phosphate of potash and pure organic matter; an olive-coloured acid; excretoleic acid. Lastly, there is no evidence of butyric acid.

Excretine contains both sulphur and nitrogen. It is not found in carnivora. Their fæces yield a substance like it, but not identical with it. Moreover, these contain butyric acid in their excrements.

Among herbivora, horses, sheep, dogs fed on bread, wild boars, elephants, deer, and monkeys, neither butyric acid, excretine, nor cholesterine, are found. The crocodile, however, forms an exception in regard to the cholesterine.

From an analysis by Fleitmann, the salts of fæces are very numerous:—

Chloride of sodium	·58
Chloride of potassium	.07
Potash	12·44
Hydrate of potash (or equivalent weight of carbonate of potash)	10·05
Soda	0·75
Lime	21·36
Magnesia	10·67
Sesquioxide of iron	2·09
Phosphoric acid	30·98
Sulphuric acid	1·13
Silicic acid	1.44
Carbonic acid	1·05
Sand	7·39
	100·00

An ultimate analysis by Playfair gave the following results:—

Carbon	45·24
Hydrogen	6·88
Nitrogen (average)	4·
Oxygen	30·3
Ashes	13·58
	100·00

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As a general rule, human excrement is richer in nitrogen than that of animals, in which the proportion of 2 per cent. only obtains; and even among men it varies with the quality of food, being among those who live on vegetables about the same as among herbivora, and greater among those whose food is essentially meat. About 7 per cent. at least of fæces consists of the undigested residue of food, such as pieces of muscle, lignine, chlorophyll, wax, etc. There is, also (varying with accidental circumstances), more or less of the secretions of the liver, pancreas, etc. Many of the changes, so brought about are chemical and mechanical changes occurring in the gut itself from the admixture of varied elements, left residuary during the transit through the intestines. Their occurrence is also best explained on this supposition. The production of fæces, viewed as such, is then simply one of limited oxidation or combustion. The fæces contain the unbruised substances of food, such as chlorophyll, wax, etc., which have suffered no change in the organism—the carbon, hydrogen, and nitrogen being small in quantity as compared with that in food. In fact, these indigestible matters may be compared to the smoke and soot produced when food is imperfectly burned in fireplace. (Playfair's *Liebig's Agricultural Chemistry*, fourth edition, p. 176.)

Still it cannot be denied that, in part at least, though to a very small degree in health, a portion of the fæcal matters are directly secreted from the mucous membrane of the intestines. This secretion, however, is at best only vicarious. Precisely as in amenorrhœa, hæmatemesis may supersede menstruation, or in jaundice the kidneys may eliminate the bile, so in disease or under extraordinary circumstances, a fæcal secretion may occur or be greatly exaggerated. Thus—

1st. Diarrhœa is often persistent, and, at the termination of some diseases, such as phthisis, will come on extensively, and yet after death there is but little fæcal matter found in the intestines. This matter must, then, have been secreted while life persisted.

2nd. In many cases, although the food taken is not greater than usual, the diarrhœal matters voided are unusually abundant.

3rd. Liebig has shown that if fibrine, albumen, or caseine, or gelatine, be treated with solid hydrate of potash, and the heat kept up until all the nitrogen is given off as ammonia, and the hydrogen begins to be given off, and the

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residue be saturated with dilute sulphuric acid, and distilled, then a substance of fæcal colour, and of a horrible fæcal odour, which contains acetic and butyric acids, remains behind, which is, in fact, fæcal matter artificially prepared. "This odour varies with the nitrogenous substance employed, and thus we may have all the degrees of fæcal odour." (Carpenter.)

4th. Fæces differ in many respects in their odour from that evolved by animal substances during fermentation or putrefaction, this very odour disappearing on exposure, and the fæces themselves fermenting after such exposure. (Carpenter.)

5th. Besides the secretion of fluids, Magendie and Girardin have made an observation, which is confirmed by Frerichs, and which, at all events, proves the secretion of gas from the blood into the intestine; for if a loop of intestine in dogs, after being perfectly emptied of its contents, be tied at both ends, it is always found, after some time, filled with air. (*Lehmann's Chemistry*, Day's Translation, vol. ii, p. 132.) Copious gaseous secretions is often observed in fevers, when it gives rise to tympanitis.

The above view gives a ready explanation to many peculiarities observed in the production of fæcal matter during disease, and also to the great advantage which often follows copious purgation, which may be regarded as removing injurious matters from the blood precisely as other eliminants or evacuants; these matters having been probably often, at least in part, previously absorbed with the aliments in their progress through the intestinal canal.

FERMENTATIVE CHANGES.

The retention of fæcal matters in the body give rise to different diseases. Those in which fæcal matters, from their quantity, act, as it were, mechanically, and thus prove injurious, do not properly belong to the subject of this paper. It is to those which may be produced by the development of animal poisons formed in their substance through chemical action, either while in the body or after its expulsion out of it, that I wish especially to call attention.

These changes are essentially due to fermentation, in the full meaning of the term, including those of both the words putrefaction and eremacausis; that is, changes effected by exposure to water or atmospheric air at certain temperatures—eremacausis being the effect of a low combustion taking place in atmospheric air, without production

of odour, and putrefaction the same, with offensive odour. The changes which occur in fæces in the body in health, and out of the body under ordinary circumstances, are thus rather those of eremacausis; while those which occur in disease in the body, and under extraordinary circumstances out of it, are more properly those of putrefaction.

It is an admitted fact in the present day that many of the healthy changes in the body are the products of fermentation. All those different matters which constitute the food of animals are fermentescible substances. And all those varied substances which are capable of putrefaction, while in this state, have the property of acting as ferments. Thus, putrescent flesh, blood, bile, urine, the mucous membranes of the stomach, intestines, bladder, etc., the skin, etc., may act as ferments. To each of these particular ferments a peculiar kind of fermentation belongs, which may not be produced by another; and each results in the formation of a particular product. All that is necessary in this process is exposure to oxygen, which is usually effected through the agency of the atmosphere or of water. Indeed, many substances require the presence of the latter before they can absorb oxygen. This fermentation will continue till the molecular dynamisation set up has been again quieted by the production of a new substance, and, as a consequence, a state of equilibrium: and this equilibrium will persist so long as no new disturbing ferment is introduced, to set up another kind of fermentation. The fermenting principle will be reproduced just as yeast is from wort, whenever, first, there is a substance present with which the ferment was formed, and, secondly, whenever there is a compound capable of being decomposed by contact with the exciting ferment.

Now, there are some circumstances which, when in operation, favour putrefactive fermentation. These it may be as well to review, as a knowledge of them has a practical bearing to the subject under contemplation, more especially in regard to any prophylactic measures to be adopted. "It was long since shown by Barckmann and Hildenbrandt that oxygen is most conducive to putrefaction; but its power is greatly increased by the presence of nitrogen, which, however, appears to exert no other action than that of separating the molecules of oxygen. An analogous fact is presented by hypophosphoric acid, which is formed very readily when its base is in contact with a mixture of oxygen and azote, but not at all when submitted

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to pure oxygen. Hildenbrandt found that meat putrefied completely in eleven days when exposed to this gas in a state of purity. Unmixed azote tends to retard rather than to favour putrefaction, hence this process is slow in privies. Hydrogen, carbonic acid, and nitrous acid are unfavourable, whereas electricity accelerates the process. Muscles subjected to electricity are, after a time, deprived of their salts, the oxides going to the negative pole, the acids to the positive. In the ordinary action upon animal matters, electricity most probably alters the composition of the proximate principles; thus, in milk, it developes acetic acid. Mathieu observes that portions of meat placed upon zinc plates remained fresh for a long time; the meat, being electro-negative, repelled the oxygen. Aqueous vapour is highly favourable. Gay Lussac found that he could retard putrefaction for a considérable period in a bell glass containing at the bottom of it chloride of calcium. Devergie attributes to the solvent power of water its agency in promoting putrefaction. A stream delays the change, probably by removing the putrid particles first formed, which, if remaining with the particles not decomposed, would hasten the process; and, at the same time, guarding the substance from the contact of air. Chlorine retards, by forming a white pearly compound almost imputrescible. Deutoxide of nitrogen has a similar influence by absorbing oxygen and sulphurous acid, by transforming the matter into a substance highly oxygenated" (*British and Foreign Medico-Chirurgical Review*, p. 396-7, vol. ii). "Stagnant water accelerates those stages of putrefaction entitled green, brown, and liquid. Putrefaction is less rapid in the water of privies. This is due, according to Devergie, to the presence of a large quantity of ammonia, which retards the liquid putrefaction, but favours the production of adipocere. He arranges the different media with reference to their power of favouring putrefaction in the following order: air, manure, the *water* of privies, stagnant water, running water, earth." (*Ibid.*, p. 400.) Smoke or carbon, in suspension in air, and creasote, have the same effect in arresting or preventing putrefaction. Animal matters in nitrogen, or chloroform vapour, do not putrefy, but will keep for an indefinite time, as shown by Dr. B. W. Richardson. Dr. Richardson also found that the presence of phosphorus in water, because it also absorbs oxygen, is also in great measure a preservative against putrefaction. Sea water favours decomposition sooner than fresh water; particularly, I believe,

when not in a concentrated state, *i.e.*, if diluted with much water, and of specific gravity 1010·15 instead of 1027, as is evidenced by the rapid putrefaction of sewage matters when admixed with sea water. Indeed, the foul odours of drains at low water in summer, especially if the pipes conveying sewage are not carried out far at sea, are proverbial.

I have said that changes of a fermenting character occur in the body normally. "Fermentation is, however, in amount small in the small intestines as compared with what it is in the large, and the reason is obvious; because the fermentation begun in the small intestines occurs with much greater rapidity in the latter, and is unimpeded by the presence of free acid." (Lehmann, *op. cit.*, ii, 130.) Marcet found that the constant reaction of fæces was alkaline. (*Op. cit.*) The experiments of Magendie, etc., confirm the statements made above. Magendie and Chevreuil found the following proportions in the intestines of executed persons:—

In the Small Intestine.

	Carbonic acid.	Nitrogen.	Hydrogen.
No. 1.	24·39	20·08	55·53
No. 2.	40·	8·85	51·15
No. 3.	25·	66·6	8·4

Chevillot found 2 to 3 per cent. of oxygen in the air discharged from the small intestines by aged persons. The hydrogen in these cases is generated by the amylaceous matters undergoing the butyric acid fermentation.

In the Large Intestine.

	Carbonic acid.	Nitrogen.	Carburetted hydrogen.	Hydrogen.
Magendie and Chevreuil	43·5 to 70.	18·40 to 51·03.	5·47 to 11·6.	
Chevillot (aged persons, 2 or 3 per ct. oxygen)	23·1 to 93.	95·2 to 90	..28·	
Marchand, No. 1.	36·5	..29·	..22·	..13·5
Marchand, No. 2. (1 sulphuretted hydrogen.)	44·4	..14·	..15·	..15·8

Occasionally the amount of fermentation going on is excessive, as evidenced by the presence of copious frothy stools, and by the generation of unusual products. The changes here induced are thus explained by my esteemed

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friend Dr. Ayres, in a private letter on this subject:—"The decomposition of fæcal matters would much resemble that of the food taken, and would differ according to the nature of that food. On the supposition that the animal had been fed on flesh, then a limited supply of oxygen must be afforded. The nitrogen would combine with hydrogen, forming ammonia. Part of the carbon, phosphorus, and sulphur, would also combine with the hydrogen, forming respectively carburetted hydrogen, phosphuretted hydrogen, and sulphuretted hydrogen, and another part of the carbon with oxygen, constituting carbonic acid. All these products, except the phosphuretted hydrogen, were found in the colon by Marchand."

But occasionally, also, the fermentation is one of *putrefaction*. Thus, says Lehmann, "substances stagnating in the different parts of the colon undergo complete putrefaction, and their products, gaseous as well as solid, are precisely the same as those we observe out of the body. In the examination of such masses Frerichs found substances precisely similar to those Bopp obtained from putrefying protein bodies." If it were otherwise, fæcal matter would be an exception to all other animal matters. Apart from the specific substances it contains, there are the usual proteinaceous matters and *débris* of food, which, under circumstances of heat and moisture, will putrefy.

But, further, we may glean something on the nature of such changes by reference to manures, and some of the changes which take place in them. My friend Dr. Ayres, in a letter to me on the subject, writes:—"When night soil is mixed with water, and distilled over by a very gentle heat, ammonia and sulphuretted hydrogen come over. If the distillated substance be now freed from sulphuretted hydrogen by distillation with carbonate of potash, and from ammonia by a second distillation with dilute sulphuric acid, a fœtid liquid is obtained, which gives a rose tint with nitric acid, and a beautiful lemon yellow when the acid liquor is supersaturated with ammonia; and if the original liquid be kept in a stoppered bottle, decomposition again occurs, and fresh ammonia is again generated."

The gases evolved in the atmosphere which surrounds common sewers may be likewise looked upon as produced by the putrefaction of fæcal matters. Thénard speaks of these as of two kinds: 1st. Atmospheric air, with hydrosulphate of ammonia in it. Thus, the air of one of the common sewers of Paris gave for composition, viz., oxygen,

13·79; nitrogen, 81·21; carbonic acid, 2·01; sulphuretted hydrogen, 2·99; besides a small quantity of ammonia. 2nd. The other gas is more rarely met with, and consists of —oxygen, 2; carbonic acid, 4; nitrogen, 94; sometimes mixed with ammonia.

These different products are evidence of a different kind of fermentation, or at least a different stage and progress of it. But the particular changes beside those dependent on the escape of ammonia in the *solid matters*, and in the production of fœtid liquid described by Dr. Ayres, I believe, have never been accurately investigated: the animal proximate and ultimate substances formed are not known. On the changes which *excretine*—the principle of excrement—undergoes, Marcet himself is silent. Such an investigation (because we have here offensive matters, if I might use the term, *in essence*) would be most important in its results, and would doubtless greatly reward the patience of any inquirer who would not allow his zeal for science to give way before the disgusting details and the offensive odours which would surround him. Dr. Ayres has stated to me he hopes some day to attempt the task. I hope he will be spared to unveil a few of the mysteries of this branch of chemistry.

CHANGES OF FÆCES IN DISEASE.

There can be no doubt that fæcal matters undergo very remarkable changes in disease, even *when yet in the body*. Independently of those which are visible to the naked eye and the microscope, as when blood, pus, pieces of fibrin, albumen, or other foreign matters, are detected in them, there are those of reaction and smell—sometimes intensely acid, sometimes neutral. But the smell is to me the most characteristic. If its occasional absence altogether be proof that the ordinary changes in the fæces have not taken place, the offensive odour is sometimes far more so—a smell so foul that even the ordinary odour of fæces, bad as it is, becomes in many cases a perfume compared to it. How frequently is this foul odour in them observed in infantile diseases, especially the *typhoid* varieties of exanthemata and remittent fevers, so characteristic as even to be noticed by the undaunted attendants of a sick room, and to be commented upon as a foul corruption, "smelling like a corpse". Indeed, the term *cadaveric* is the best epithet which I can find to express the character of this odour. Speaking of the smell in teething cases, Dr. Osborne, in a

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paper on the Examination of the Fæces in Disease (*Dublin Journal*, xlv, 109), remarks:—"Sometimes it acquires a pungency almost intolerable, as is observed in cases even of slight diarrhœa, when, after the more solid contents of the colon and rectum have passed, they are succeeded by dark coloured fluid motions, consisting of fresh arrivals from the cæcum, of such overwhelming potency that even the most experienced and apathetic physicians are compelled to a speedy flight," etc. Dr. Osborne compares this odour to that of an ulcerated bony surface. Be this as it may, this peculiarity of odour is also noticed in many of our worst malarious fevers. In dysentery, this odour, according to Schönlein, is very characteristic. "In yellow fever," says La Roche, "in the first stages, and especially when obtained by purgatives, the matter discharged from the bowels has an offensive odour, emitting, indeed, in some cases, an intolerable and dreadful fœtor, alike repulsive to the sick and attendants." (La Roche on *Yellow Fever*, i, 263.) This cadaveric odour I look upon as evidence of the poisonous nature of such stools, as I shall show presently; and it is interesting to notice, in connexion with this advanced state of putrefaction, that the worst kinds of typhus are usually preceded by the most obstinate constipation. So also, "in the very large majority of cases of yellow fever," says La Roche, "constipation of the bowels constitutes a predominant feature in the early stage, proving in some instances as obstinate as in any known disease, and resisting at times the action of powerful cathartic medicines. This condition of the bowels is found to prevail more generally in the severer and more concentrated forms of disease." (*Op. cit.*, i, 260.)

INFECTION THROUGH FÆCAL MATTERS.

There are three conceivable ways in which disease may be generated by the absorption or ingestion of fæcal matters:—I. When in their natural, concentrated, undiluted state; II. When taken after dilution or suspension in water; III. When the emanations arising therefrom are inspired or otherwise absorbed in the system. Under these three heads I propose considering the subject. In doing so, however, it is right to premise, by the following considerations, which must be admitted *in primis*, otherwise the conclusions may prove unintelligible.

Infection, to be propagated, requires three concurrent

conditions:—1. The infecting source; 2. A transmitting medium; 3. A fit recipient.

1. *The source of infection* may be the person affected, fomites, his excretions, or, in fact, any personal part of him so contaminated. Such source is always more concentrated and potent at the *outset* of an epidemic, and the reverse towards the termination of it.

2. *The transmitting medium* may be *(a)* liquid, or *(b)* gaseous. *(a)* In the former case, as in the example of inoculation, it may be the blood; it may be water holding any poison in suspension or solution; and, in fact, any other fluid which does not destroy the poison, applied externally or taken internally with food or drink. *(b)* In the latter case, it may be a gaseous or volatile substance mixed or suspended in the atmosphere. Now, of these two varieties of transmitting agents, the *gaseous* and the *liquid*, the *barometrical changes which influence the development of the one may be the very reverse of those which influence the spread of the other.* Thus, the gaseous will be more energetic in hot and moist weather, and the liquid in moderately cold or temperate and dry weather; because, in hot and moist weather, the air is lighter, and the ammonia more volatile, which gas, with Robiquet, I believe to be a great adjuvant in the suspension of poisonous emanations in the air, especially when damp. Hence it is that in hot and moist weather many contagious diseases are rapidly conveyed and extended, whereas cold and dry weather will prove beneficial in its effect, the air being heavier, and the ammonia not only less volatile, but also less in quantity, because animal matters decompose less rapidly in cold weather. We are all aware how excessively fœtid our London drains smell just before rain sets in, so much so as to be the ordinary and popular prognostic of rain in this town. According to Robiquet, as I have said, the odour of gaseous (which may also be contagious) matter is due to the volatility of the ammonia, which, as it were, raises it to the nose. Ammonia is, moreover, always evolved in the putrefaction of organic matters. It is also very generally produced in cases of disease, and is always emitted in contagious diseases. (See Liebig's *Agricultural Chemistry*, by Playfair, fourth edition.) In cold and dry weather, since the air is heavier, and the ammonia therefore not so volatile, all poisonous matter will be more likely retained in solution or suspension in the flowing or stagnant waters; unless, indeed, the cold be so extreme as to destroy all fermentation. In this case, the poison may,

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therefore, be more readily taken in the water, or other liquid ingesta.

3. *A fit recipient* is necessary: and here, even more than in the preceding conditions, there has been much error admitted. In judging of the effects of poisons, we have not fully considered the effects of *race* in man, *species* in animals, *concentration* of the poison if taken at the commencement or at the end of an epidemic, and the effect of *acclimatisation*, besides *previous diseases*, *occupation*, and *state of health* of the recipient. These are a few amongst many causes, the neglect of which has led to the most heterogeneous confusion and contradictory statements. Thus, a *negro* is less susceptible to fever than a *white* man. All *animals* may not be susceptible of taking certain diseases, or at least of catching them in the same way, as men do. Thus Beclard inoculated dogs with plague matter, and made them eat the matter from plague buboes and pathological specimens of plague; and they did not catch the plague. Similar experiments performed on criminals (*British and Foreign Review*, v, 561) communicated the disorder. Syphilis is another disease which cannot be in any way transferred to the lower animals. The symptoms may vary: thus, the dread of water in hydrophobia is a symptom invariably present in man, but not in rabid dogs. Again, small-pox may become cow-pox in a cow. The same man, *ceteris paribus*, as regards health, may, after exposure, get cholera at the commencement of an epidemic from its virulence, when, at the end of the same or a similar epidemic, from the milder character of the disease, he may get only diarrhœa. Then, in regard to the experiments on animals at the beginning of an epidemic, from the same cause, cholera may be induced by making them swallow the diseased egesta; and yet, at a later period of the epidemic, only diarrhœa will result. A person who has lived in an infected district for some time is less likely to suffer from an epidemic than a new comer; and so on. All these circumstances are most important to notice during contagious epidemics, and yet, I fear, are too often neglected even by our best authorities; and therefore I have thought it necessary to review them briefly, because they bear so manifestly on my subject.

I. EFFECTS OF ABSORPTION OF FÆCAL MATTERS IN THEIR CONCENTRATED AND UNDILUTED STATE.

Effect of Diseased Faces on the Body. Healthy faeces, I believe, are not poisonous; and to some animals, owing to retained nutritive matters in them, they may prove nutritious, as in the case of those of the horse, partaken of by birds. I have seen the experiment voluntarily performed by dogs. The pointers and other *chiens de chasse* are notoriously voracious, and I have frequently seen them devour, apparently with great avidity, the fæcal matters in their way, and have never observed any peculiar effects produced therefrom; and other additional examples from barbarous nations, and hysterical and demented persons, might be adduced to prove the same fact. But, more than this, the annals of ancient medicine record their exhibition in the treatment of diseases. Fonseca (*De Excrementis Humanis*), quoting from Dioscorides (lib. ii, ch. 72) and Galen, enumerates a series of diseases, such as anginas, dysenteries, sore throats, phlegmonous inflammations, colic, many skin-diseases, etc., in which the faeces of man or animals were found curative in their effects. He has the modesty, however, to add that these remedies are better borne by unlettered rustics than by polished townsfolk. The record of such a practice, though eminently disgusting in the present day, is historically curious, and confirmatory of the non-poisonous properties of faeces in health if accidentally partaken of. This must be my excuse for alluding to the fact. On the other hand, many persons retain fæcal matters in their bowels for long periods with apparent impunity. The faeces in these cases are unchanged; no poisonous fermentation has occurred.

But it is not always so. Those who are acquainted with the works of Hamilton on purgatives and Burn on constipation are aware how far, in the opinion of these writers, the retention of fæcal matters in the bowels is capable of producing disease. In the hands of the former writer, typhus, scarlatina, marasmus, many nervous and chronic diseases, seemed sometimes to be produced, at least much aggravated, by accumulations in the bowels, and were greatly relieved or cured by aperients. According to Burn, it seems that the general health may be completely undermined by habitual constipation. There is scarcely a variety of indigestion, an uterine affection, a dysentery, or diarrhœa, not capable of being originated by

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this cause. Without, however, asserting the universality or even generality of this cause, as productive of these diseases, it may be admitted that all these diseases may occasionally be so generated and kept up. In many cases, the effects produced are analogous to those usually observed to follow the absorption in the system of putrid animal matters, such as flesh, and pus in a putrefactive state. When these last are placed on wounds, vomiting, low continued fever, rigors, and death, often result. The danger of dissection wounds is proverbial. "The poison of bad sausages", says Graham, "belongs to this class of noxious substances. Several hundred cases are known in which death has occurred from the use of this kind of food. In Würtemberg especially these cases are very frequent, for there the sausages are prepared from very various materials. Blood, liver, bacon, brains, milk, meal, and bread, are mixed together with salt and spices; the mixture is then put into bladders or intestines, and, after being boiled, is smoked. When these sausages are well prepared, they may be preserved for months, and furnish a nourishing savoury food; but when the spices and salt are deficient, and particularly when they are smoked too late, or not sufficiently, they undergo a peculiar kind of putrefaction, which begins at the centre of the sausage. Without any appreciable escape of gas taking place, they become paler in colour, and more soft and greasy in those parts which have undergone putrefaction; and they are found to contain free lactic acid or lactate of ammonia,—products which are universally formed during the putrefaction of animal and vegetable matters." This change of colour I have before remarked is observed in fæcal matter kept in a moist state and exposed to the air, and I presume it is indicative of an analogous change in it. Very lingering and remarkable symptoms precede death when this is the result of poisoning by putrefied sausages. There is a gradual wasting of muscular fibre, and of all the constituents of the body similarly composed. Other substances employed as aliments, as cheese, pork, bacon, mussels, salmon, in a state of partial putrefaction, occasionally produce poisonous effects. These vary, it is true, in degree and kind; but the effects are not the less certain. It is, therefore, in strict accordance with analogy to conclude that putrefying faeces may generate disease in the same manner, the poison being absorbed by the same intestinal membrane.

Moreover, that faeces are capable of inducing a putre-

factive fermentation in surrounding tissues, is evidenced by the character of the pus in abscesses about the large intestines. We all know how excessively offensive the pus in abscesses about the rectum and colon generally is—in fact, wherever fæcal matters are to be found in its vicinity; and this without necessarily, I believe, a direct communication. Here we have the ferment acting at a distance, perhaps through endosmosis, upon the pus, and exciting its decomposition, which is often acid in reaction, instead of alkaline as in healthy pus. Patients under these affections frequently suffer from typhoid symptoms and low fever. Their very expression denotes serious blood-disease. These symptoms, it is true, may be referred, secondarily, to the absorption of putrid pus; but they were no doubt due primarily to the changes effected in the pus. The habitual foul breath and tongue, the generally offensive, almost fæcal odour of some persons (particularly uncleanly females), who are habitually addicted to constipation, are evidences of absorption of fæcal matter under ordinary circumstances, which, like a contaminating drain odour (only originating in the patient himself), must prove injurious to the organism. The anomalous nervous symptoms originating in hysterical females and boys may often be referred to fæcal impregnation, so speedily are they relieved or entirely removed by a brisk purge. In these cases, we may suppose that the poisonous substance in the colon sets up a partial fermentation in the blood, which may in adverse circumstances excite fever, and even death.

Synocha or ephemeral fever is, I think, as frequently the result of such a change in grown up persons as remittent fever is in infants, yielding frequently to good purging, the motions having the abominable putrid odour before referred to, very different from that of healthy fæcal matter. These and many other allied diseases may be referred to this cause. The effect on the system is, however, certainly less violent than that produced by fæcal fermentation in water or in the atmosphere. If I might venture to call it so, I would describe it as poisoning only in the first degree.

Cholera. The experiments made with cholera excreta on man are necessarily few, and are almost exclusively confined to a few instances where love of science has overcome the natural disgust to such a course. Five such cases are recorded by Dr. Richardson, in his Review on the Water Supply in London (*Journal of Public Health*, vol. i,

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p. 130). A sixth case was the result of accident. On Sept. 5th, 1853, four physicians tasted the intestinal fluid of a cholera patient. One of these (who, however, had been labouring under diarrhœa previously) took cholera on the 8th, and died in the evening; another was taken ill with choleraic symptoms on the 9th, but these symptoms passed into typhoid fever. An assistant in the Cholera Hospital at Berlin tasted choleraic fluid. Diarrhœa soon supervened, which passed into a smart attack of cholera. The accidental case is one related by Messrs. Pearce and Marston, in which a dispenser drank a quantity of rice-water evacuation by mistake, without any result. Dr. Richardson gives also the case of M. Scipion Pinel, who inoculated himself with cholera evacuation, and two hours afterwards suffered from sickness and vertigo, which disappeared after free perspiration; and that of M. Foy, who tasted vomited matters and breathed the air expired by a choleraic patient, but who, although unwell for four days, did not get cholera. In neither of these latter cases, however, were the fæcal matters tasted; and thus they do not bear upon this part of my subject. The other cases afford only negative and inconclusive evidence.

The lower animals have also been experimented upon with the dejections of patients affected with Asiatic cholera, but so far as I know, those of Thiersch of Munich are the only experiments made with putrid choleraic stools. I quote from Dr. Snow's excellent work on cholera. "Dr. Thiersch is of opinion that the choleraic evacuations are not capable at first of generating the disease, but that a decomposition takes places in them, and that in from six to nine days they become in a state to induce cholera. He founds this opinion on experiments which he performed by giving small quantities of the cholera evacuations to white mice." (*On Cholera*, p. 112.) From some experiments detailed at length by Mr. John Marshall, of London, in the *British and Foreign Medico-Chirurgical Review* for April 1853, we may, I think, conclude that choleraic disease, yet unaccompanied with the algide symptoms of Asiatic cholera, may be communicated to animals, by the ingestion of the cholera stools of man. In a paper by Dr. Lauder Lindsay (*Edinb. Med. and Surg. Journal*, lxxxi, 1854, page 275), it appears the specific disease cholera was not generated by feeding dogs on choleraic vomit and evacuations, nor even on the blood drawn during life or taken from cholera cases after death. The

effect was the production of a non-specific diarrhœa. Their general health did not appear to deteriorate, or the poison to act otherwise than as a predisposing cause to their suffering from cholera, after exposure to its exhalations. The diarrhœa generated appeared to be very violent, with griping, vomiting and cramps, and great exhaustion, but the expelled matters were always feculent. In two dogs the disease proved fatal. The *résumé* from the experiments detailed, both by Dr. Lindsay and Mr. Marshall, thus appears to be that in all these cases diarrhœa more or less severe, and vomiting were produced. This was the general rule. In some cases the symptoms were so severe that death resulted, but fæcal matter was expelled to the last. The *post mortem* examinations of these cases were, however, defective. Moreover, in the few instances recorded where actual cholera is said to have been produced and to have been in the end fatal, I do not find it stated whether the urine was suppressed or not; such a circumstance, had it been present, would have gone far in my mind to prove that this diarrhœa was, after all, cholera, modified in different animals. The experiments, so far as they go, are then inconclusive; besides, they certainly present no relation which is constant, to the quantity of fæcal matters swallowed; although, as a rule, a large quantity was more certain in its products.

I am not aware of any experiments that tend directly to prove that the evacuations of typhus and other malignant fevers have ever been experimented with in the same way. We may, however, reason by analogy that in some cases yellow fever is propagated by the evacuations. The black stools passed in many instances of this disease differ in nothing from black vomit; and black vomit has been known, even when rubbed externally over the body, to generate the disease.

The evidence from retention of diseased fæces is, however, more conclusive. I have already alluded to those bad cases of typhus and yellow fever which are preceded by obstinate constipation. Among ourselves, the most familiar example of disease which, *per se*, proves the poisonous influence of diseased or putrid fæces, is afforded in that variety of *puerperal mania* which is so often induced by accumulations in the intestines. It should be remembered that the female after parturition is placed in those conditions most favourable to absorption, as there has been loss of blood to a greater or lesser extent, and invariably as a concomitant more or less nervous shock. Absorption of any

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offensive matters under these circumstances is greatly facilitated. Now these cases are generally accompanied by more or less head symptoms—the head may or may not be warmer, but a slight incoherent delirium at first, often passing into mania, presents itself. The face is sallow, and the conjunctiva is tinged with bile; the tongue is remarkably foul, the breath offensive, often fæcal. The abdomen is very tumid and swollen, and in one or other of the iliac fosse there is uneasiness, not to say tenderness, and on deep pressure frequently a large mass of fæces may be detected. The motions are peculiarly offensive, and in most cases emit that cadaveric odour which to my mind is so indicative of putrefaction in fæcal matters. The pulse is frequent, easily excited, from 100 to 120, and a shivering fit often occurs, thus denoting that the entire system is impregnated with the poison. The *locale* of the disease is often strongly manifested in the progress of the case. Rapid purgation may come on; the head symptoms will be ameliorated, if not actually removed; but the pulse will fall in frequency and increase in strength as the offensive matters are removed, an effect which, being generally the reverse of that observed after violent purgation, I look upon as almost diagnostic of this form of fæcal impregnation. It is clear in such cases that something more than fæcal accumulation is present here. The symptoms are those of a narcotic poison in its first effect with fever, and cannot but be partially explained if looked upon as due only to mechanical irritation. There are besides similar examples of disease occurring in non-puerperal cases, as in many instances of mania.

I have mentioned an extreme case, but the variety of minor affections of the same kind, differing only in degree, is legion. In these cases the poison is in the *primæ viæ* primarily; and so long as it remains, so long will the disease be kept up.

Since writing the above, my attention has been directed by Dr. Richardson to a report by Dr. Rennie, in a parliamentary blue book on *Further Correspondence on the subject of Convict Discipline and Transportation*, of the results obtained by overfeeding convicts in Western Australia. These men receive upon an average from 7 lb. 8 oz. to 7 lb. 14 oz. of food daily, from 59 to 67 oz. of which were solid food. This gross amount of food resulted in the production of a disease which affected the individual in various

ways, sometimes bringing on dyspepsia, dysentery, severe constipation, or other analogous bowel complaints; sometimes a severe kind of ophthalmia; sometimes a cutaneous eruption. These diseases yielded to powerful purgatives and low diet. In one case (that of a man named Nain), the patient took 5 purgative injections, 480 grs. of compound jalap, 8 oz. of Epsom salts, 32 other purgative pills, 1½ oz. castor oil, 5 drops of croton oil, 8 grs. of scammony, and 6 grs. of gamboge, before the cure was effected. During this period, he voided 30 lbs. of feculent matter in a state of decomposition.

The daily average number of prisoners was 504; of sick, 42. The total number of sick in one year was 2,290; of whom 959 had diseases of the digestive organs; 598, diseases of the eyes; and 633, diseases of skin, including ulcers and abscesses. These diseases, as showing their origin from the same cause, were cured by the same mode of treatment and low diet, and were frequently metastatic one to another.

But what I wish to call attention to, is the fermentative character of the disease which was set up in connexion with the putrid character of the evacuations passed. Dr. Rennie says: "With respect to the peculiar nature of the blood disease, I stated that, from finding on a microscopic examination of certain forms of skin-diseases, a low form of vegetation, resembling the yeast plant (one of the most primitive illustrations of organic life), there were rational grounds for supposing that the large and badly constituted diet might lead to fermentation and the development of this low form of vegetation, which, after becoming absorbed and circulating for a time in the blood, ultimately might become expelled in the form of local disease. As a proof of the fermentative process going on in the intestinal canal, I mentioned that one of the most common appearances which the evacuations presented in the early stages of dysentery was that of copious, frothy, watery stools in a high state of fermentation, and bearing a general resemblance to yeast." (p. 119.) In a table given at page 128, where he records the cases seriatim, the amount of medicine taken, and the characters of the stools, the fermenting process going on is well exemplified. In 4, the faeces are described as being in a high state of fermentation; in 6, putrid; in 2 only, tolerably healthy; in 3, the matters voided were like pea soup; and in the rest, pus, blood, mucus, shreds of membrane, etc., existed in abundance. The number of cases so tabulated amount to 22, affected with ophthalmic and skin

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diseases; half only, or 11, being affected with dysentery. This class of cases proves, I think satisfactorily, that faeces putrefy in the body if long kept there, and will give rise, if not removed in time, to serious blood disease.

PART II.

II. EFFECT OF FECAL MATTER WHEN TAKEN AFTER SOLUTION OR SUSPENSION IN WATER.

In entering upon the consideration of this subject, I feel bound to express the obligations under which, personally, we all lie to Dr. Snow, to whom the merit particularly belongs of having first called our attention to a source of disease previously ignored. Indeed, I find it difficult to express, in terms which I think would do justice to him, my sense of the importance of his discovery, and which he has also so ably enunciated, and proved, as I think, to demonstration, in his work on *Cholera*. Its truth is now admitted, with few exceptions, by most men of science. Besides, in its results, it has conferred already great advantages on the public, having originated the adoption of decisive measures in regard to the supply of water in this metropolis, and in other places. The results of a further development in this direction on the social condition of man may, like Jenner's discovery, be the means of preventing the spread of fatal disease, and saving thousands of lives to the nation. In this sense, I think our thanks to Dr. Snow cannot be too strongly expressed; and, for one, I feel I owe him a great debt of professional gratitude.

In speaking of *putrefaction* in the first part of this essay, I referred to the necessity for the presence of certain agencies to enable substances to pass into fermentation—*i. e.*, to absorb oxygen.

1. That *water* is sometimes necessary, is proved (among other circumstances) by the experiment made by Gay Lussac, who found that, if chloride of calcium were placed at the bottom of a jar of oxygen in which meat was suspended, the meat remained fresh many days; the chloride of calcium, from its affinity for moisture, keeping the gas in a perfectly dry state. Hay, straw, wool, if perfectly dry, will keep for any length of time; but, if moist, they will become slowly charred, undergoing a degree of oxidation which may pass into combustion if exposed to the atmosphere.

2. If, when water or moisture be present, a small quantity of fermenting matter be added, the process of fermentation will rapidly progress—much more rapidly than if time be given for its independent development. An experiment was brought before our notice, and detailed by Dr. Ayres, on the occasion of reading the first part of this paper, which applies so much to my subject that I cannot help again referring to it. Sir J. Pringle took the putrid yolk of an egg, into which he dipped a small thread. A bit of this thread was cut off, and put into a phial, with half the yolk of a new laid egg, diluted with water. The other half, with as much water, was put into another phial, and both, being corked, were put by the fire to putrefy. The result was, that the thread infected the first yolk very speedily; for the putrefaction was sooner perceived in the phial that contained it than in the other. (*Phil. Trans.* 1750, p. 554.) If, then, the ferment be thus supplied, the process in the water will progress most readily.

3. Water may become superoxygenated, both *(a)* by living animalcules and *(b)* by vegetable matters.

(a) By *animalcular life*. I have said before that oxygen is essential to bring about fermentation. Now water is, under certain circumstances, found to contain this gas in unusually large quantity. We all know that, while there exist in water certain kinds of animalcules which absorb oxygen and give out carbonic acid, thus exhaling a gas which, in course of time, will render that water unfit for animal life, so there are certain other animalcules which seem to act under the influence of light, as plants, giving out oxygen in large quantities. Thus (I quote from Liebig), Count Rumford noticed the fact some seventy years ago, that, if pieces of cotton, silk, and other organic substances, were placed in water, the water, after three or four days, became green, and was filled with minute spherical bodies, and evolved pure oxygen gas. No confervæ or plants of any kind were observed in this water. Again, "in the salt pans of the salt works of Rodenberg, in Hesse, a slimy transparent mass, which covers the bottom to the depth of from one to two inches, is found to consist entirely of such animalcules. This mass is everywhere interspersed with large air-bubbles, which ascend in great number through the supernatant fluid, when the pellicles enclosing them are torn by agitating the mass with a stick." "Pfaufuch, upon investigation, found this air to be pure oxygen gas, so that a wood splinter, the flame of

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which had been just extinguished, rekindled in a flame when immersed in it. Wohler found that this mass consisted of living infusoria. Messrs. Charles and A. Worren, in the *Transactions of the Academy of Brussels*, 1841, showed that water, with the co-operation of organic matter, evolved a gas containing as much as sixty-one per cent. of oxygen; and that this phenomenon was to be ascribed to the presence of *glamidonas pulviculus*, and some other green and red animalcules belonging to a still lower grade in creation." The same fact was confirmed by Liebig himself, who, after filtering a water in a trough in his garden, coloured green by the presence of various species of animalcules, filtered it through a fine sieve, to get rid of all confervæ and vegetable matters; and then, by exposing it to the light of the sun, in an inverted broken glass, the aperture of which was confined by water, found that, after a lapse of a fortnight, more than thirty cubic inches of gas had collected in the glass, which proved to be so rich in oxygen that a glowing splinter at once burst into a flame in it (*Liebig's Letters*, pp. 240, 41, 42.) It is true that, in these waters, there must be the necessary conditions of life present, contained in solution or suspension in the water, so that this may become a fit nidus for the development of the germ, fissa, or ovule of the future animal; but, in any case, we here see one way in which water highly saturated with oxygen may more readily and more rapidly cause animal or vegetable dead matter to pass into fermentation.

(b) By *vegetable life*. At any rate, if animalcules of the kind just now referred to be not present, to explain this hyperoxygenation of water, it may be due to the presence of certain water plants which have the property of keeping water fresh and continually oxygenated, although replete with animal life, such as fish, snails, etc., and other animalcules which consume oxygen, and though the water be not changed. Such are the various kinds of *valisneria* and *anacharis*, which emit oxygen gas. These facts are well known in these days, when so many keep aquaria. Many other water weeds have the same effect. Now, it is clear that more oxygen will remain in this water when the atmosphere is heavy and the barometer necessarily high; and even in cold weather, from the same reason. In this latter case, the water, especially at some depth, is often hotter than the external atmosphere; and in both these instances fermentation will take place more readily in it.

There is also this point in relation to this excess of oxygen

in water, not to be lost sight of. The very excess of animal life (I allude to those animals which consume oxygen) is proof that this gas abounds to support this increase of animal life. But, even in those countries where we have no such index of animal or vegetable life to guide us, by reason of the intense cold, oxygen may be in excess, and in like manner its abundance will explain the prevalence of epidemic disease —Asiatic cholera, for instance—in Moscow and St. Petersburgh during winter. The atmosphere at that season, from its greater weight and density, contains more oxygen in a given volume; and the porous snow necessarily contains also an increased proportion. Here then is a great supply of oxygen, and fermentation is only kept back by the intense cold. On the other hand, two habits, which I believe are common to inhabitants of snowy regions, assist to bring about this result. One is, that, owing to a want of proper water-closets, the slops are generally emptied in the neighbourhood of the houses. The other is, that, owing to the expense of fetching water from the river, and the natural unwillingness to face the intense cold longer than is absolutely necessary, the inhabitants prefer collecting the snow around their houses, and melting it down for drinking water; and thus water impregnated with fecal matters, or at any rate very impure, is drank; and hence, the moment it meets in the alimentary canal, or in the hot houses, the circumstances favourable to fermentation, this process is readily set up. Owing, moreover, to the excess of oxygen, it is very rapid when once it has begun, and thus any disease so induced becomes speedily fatal. To talk of emanations at temperatures as low as zero, and below that, which may be sensible to the organism, is, I deem, unphilosophical; and if we deny contagion to be in operation, the only place in which those circumstances of heat and moisture are present, and in which they may be developed, is in the alimentary canal, and through the ingesta introduced. So far as I know, this is the only reasonable explanation that can be afforded.

But this influence of a dry state of the atmosphere is not confined to cold weather. It has long been noticed in summer, although, so far as I know, not sufficiently insisted upon. Experience proves that, in some periods of the greatest intensity of an epidemic, the weather has been dry, and the barometer high, and the atmospheric temperature not necessarily low. These circumstances are, as I before stated, precisely those most likely to contaminate the water

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and eatables taken by a population; and the reverse of those which favour the evaporation of poisonous miasmata, namely, heat, moisture, and a fall of the barometer. It probably explains the reason why the Thames water is more fatal in dry weather with a high barometer, and this at a period of autumn when the temperature is not so elevated. To this fact also the immunity of some low districts may occasionally be referred, when the high suffer most. The poisonous particles, otherwise suspended in the atmosphere by the ammonia and moisture, are kept to the ground, and thus become part and parcel of the water we drink. It is remarkable how this dry state of the atmosphere was observed in the London epidemics of cholera. Thus Mr. Glaisher remarks: "In the year 1854, the pressure of the atmosphere was very great; the temperature generally high; sky overcast; direction of the wind, north-east and south-west; and the velocity of the air was less by one-half than its average for some time before; and, at the time of the greatest mortality from cholera, the barometer reading was remarkably high, and the temperature above its average. A thick atmosphere, though at times clear, everywhere prevailed; weak positive electricity; no rain. In low places, a dark mist and stagnant air, with a temperature in excess; temperature of the Thames very high; a high night London temperature; a small daily range; an absence of ozone; and no electricity." (*Board of Health Reports.*)

The three epidemics of 1832, 1848, and 1854, were attended with a particular state of the atmosphere, characterised by a prevalent mist, thin in high places, dense in low. During the height of the epidemic, in all cases, the reading of the barometer was remarkably high, and the atmosphere thick. In 1849 and 1854, the temperature was above its average; and a total absence of rain, and a stillness of air amounting almost to calm, accompanied the progress of the disease on each occasion (*Journal of Public Health*, No. IV, December 1855). As if, moreover, denoting the absence of ammonia in the air, Dr. R. D. Thomson mentions that the air in choleraic wards as well as the external air was *acid*, and alkaline only in sewers.

4. There is also a general remark which will explain the exemption of certain parts, even though these were possessed of waters containing the same general excess of oxygen. I believe, in London—and here I speak especially of the West End—that one of the reasons of the innocuity

of these waters was their alkalinity. My attention was forcibly called to this point by the case of a patient for whom I had ordered on one occasion the infusion of roses. I was soon afterwards sent for in a hurry to see my patient, who was supposed to have been poisoned, the lips and tongue having assumed a bright yellow brown colour. The medicine had been taken, and afterwards a glass of water. The whole was explained on testing the water, which was found very alkaline, owing to an excess of *lime*. The water in Dorset Square and Montague Square, at my own and my patient's house, as well as that of the Bryanstone Square pump, was strongly alkaline from the same cause. This innocuity of the water, even if it contains fermenting matters, is analogous to the fact observed by Pariset in Egypt. Pariset believes that the reason that the overflowing of the Nile proves so salubrious in its effects is, that its waters are *very alkaline;* and hence, as it spreads over the land, it reaches the dead, and temporarily neutralises the miasmata; and thus the plague is stayed (*British and Foreign Medico-Chirurgical Review*, p. 249).

If such be, however, the case with water containing excess of alkali, it is not so with sea water, especially if much diluted with fresh water, or fresh water with chloride of sodium. An experiment of Pringle proves that sea salt, *if in small quantity enough*, favours putrefaction. "One drachm of salt preserves two ounces of fresh beef in two ounces of water above thirty hours uncorrupted, in a heat equal to that of the human body; or, what amounts to the same thing, this quantity of salt keeps fresh meat about twenty hours longer sweet than pure water. But half a drachm of salt does not preserve it above two hours longer.... Now, I have since found that twenty-five grains have little or no antiseptic virtue, and that ten, fifteen, or even twenty, manifestly both hasten and heighten the corruption. It is, moreover, to be remarked, that, in warm infusions with these smaller quantities, the salt, instead of hardening the flesh as it does in a dry form in brine, or even in solutions such as our standard, here softens and relaxes the textures of the meat more than plain water, though much less than water with chalk or tartaceous powders" (Pringle, *Philosophical Transactions*, 1750). Pringle found, moreover, that, of the quantities ten, fifteen, and twenty grains, in two ounces of water, the former was the most putrefying in its effects. Now, sea water contains between twenty-five and twenty-six grains in two ounces;

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and therefore, if diluted with more water, as where the sea is in connexion with large rivers, so that the proportion of sea salt is still further diminished, it will putrefy much sooner. Hence, perhaps, the reason that cholera is more rife along seaports, where large rivers are also present, because putrefaction is more rapid. This view explains, moreover, the following commonly observed fact. It is known that a tough duck or curlew will eat very tender if steeped over night in weak brine or sea water. Tenderness of meat denotes incipient decomposition, although to a small degree. Fresh water will not, except in very warm weather, produce a similar effect. I have verified the same point by another experiment. Sea water from Brighton will keep longer sweet in stoppered bottles than sea water from Southend. Some of the former, collected on April 20th, of specific gravity 1018, was fresh up to the 10th June; some of the latter, collected May 12th, of specific gravity 1022, was putrid long antecedent to the 10th June. This is not surprising. The Southend waters, contaminated more or less by the sewage matters of the Thames, and yet containing less sea salt, would be, *ceteris paribus*, more readily decomposed. Besides, all sea water contains a large quantity of sulphates—from four in our channel to seven parts per 1000 in the Mediterranean; and these salts, in contact with animal matter, readily decompose, giving out sulphuretted hydrogen.

5. The existence of a poison in the water drunk may be inferred from the following considerations.

There must be in many cases something more than an emanation or miasma to explain the recurrence of some epidemics. The mere stench or overcrowding of a locality will not always account for the spread of disease. Take the cholera epidemic at Berlin in 1853. "In Prenzlau Street, and Gollnow Street, in Berlin, there are a great many overcrowded cottages, and yet the number of cases here was small. The solution of the puzzle is all the more difficult when it is considered that the houses in Büsching Street, Wine Street, and in front of the new King's Gate, stand much more open than the rest of the houses of the whole of the (ninth) medical district. There can be no question here of confined space and want of fresh air—circumstances which are usually supposed to favour the origination of cholera; and yet there were four to five times more cases here in proportion to the number of the inhabitants than in the narrow Gollnow Street, in which the circulation of air

is much impeded. . . . It certainly seems extremely surprising that, of the forty streets which compose the medical district, precisely the most openly situated of them were those which were most visited" (*Journal of Public Health*, vol. i, p. 280). Now, this something more is often, without doubt, *bad water*. Thus Scilla, a small village about fifteen minutes distance by sea from Valetta, in Malta, has always escaped cholera, while it has prevailed extensively in every other part of the island. The reason is this, all Malta (except this little village, which is supplied by tanks) is supplied by an aqueduct. And similar cases abound.

6. The *Board of Health Reports* show that the air was, during these cholera epidemics, in a weak *positive* electrical state. This, as before said, is precisely that state which attracts oxygen, and, as such, is most favourable to decomposition. It is to be regretted that the condition of the water was not also given. There is room in this direction for much inquiry. A blast of air, a direction of the wind, may determine the poisonous change in water: north and east winds bring usually positive, south and west bring negative electricity. Indeed, Dr. Bressler, in reference to the Berlin epidemic of cholera before referred to, proposes the question, whether, under certain circumstances, the admission of fresh air may not be prejudicial, as actually being the bearer of the miasma. The meeting of an epidemic may thus be synonymous with the meeting of a pernicious current—say a positive electricity, which developes the latent poison; and explains in this manner the healthiness of one place to-day and its unhealthiness to-morrow. Certain atmospheres, as well as waters or subsoils, in themselves harmless at one time, may, by virtue of their being the fittest nidi for the development of ferments, very rapidly multiply these, the moment they come into juxtaposition. This opinion is well set down by Surgeon J. H. Kerr Jones, of the 56th Regiment, in his evidence on the Bermuda fever (p. 62):—"When fever prevailed to a frightful extent among the troops in Upper Scindh, I have often at night been exposed with impunity for hours whilst wild fowl shooting, at what we considered the most concentrated sources of the poison, whose effects, even when infinitely diffused, were supposed to be deadly. The neighbourhood of such situations remained free from any disease. The troops who marched over them, and daily encamped there, continued in the enjoyment of health: it was *only when the epidemic influence prevailing on the rock of Jukkur was encountered*, that every *drain* and *cesspool*, or room abounding in exhalations from the human body, became impregnated with its poisonous property."

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7. It is probable that the *chemical rays* of light may in some way be concerned in the development of oxygen. According to recent experiments, these chemical rays vary much in quantity. A solution of quinine (the photographic test) becomes quite bluish and milky looking when these abound, and quite transparent when they are few in number; and hence the reason that photographs are so easy of execution at one time, and so difficult, if not impossible, at another. The effect of chemical rays on putrefaction has not, so far as I know, been studied; still, some facts may exemplify how an effect may be produced on them. A solution of chlorine in water, if exposed to light, will decompose the water, and liberate the oxygen. Some chlorides have an opposite effect. Thus, freshly precipitated chloride of silver will also, if exposed to light, decompose water; but the oxygen set free unites with the silver, forming oxide of silver; and thus the liquor assumes a black colour. Of the several rays of the spectrum, the violet produce this change more rapidly than the red on the moist chloride of silver. "Their characteristic effect is to promote those chemical decompositions in which oxygen is withdrawn from water and other oxides; and hence they are sometimes called *deoxidising* rays." (Graham's *Chemistry*, 1st. ed., p. 93.)

Recent inquiries, at any rate, prove that there is a connexion between these chemical rays and the electrical currents induced. How far, then, these two causes are co-operative, remains to be shown; but that they are often concurrently present, and in their effects materially influence decomposition, cannot be denied.

The seven conditions enumerated above all produce decomposition; and we may thence, I think, infer that, although their coexistence may not always be necessary for the development of every epidemic, their coexistence may materially increase the power of its action, as their disunion may diminish its intensity.

I proceed now to consider the effect of fæcal fermentation, in solution or suspension in water, in the production of *particular* diseases.

Cholera. It is not my intention to dwell at great length

on the subject of the spread of cholera by water impregnated with choleraic dejections. I conceive it is now generally admitted by all that cholera is most prevalent and fatal in the course of large rivers, and where the water supply is bad. Dr. Snow's work is full of examples on this point. I shall take from this work only three such examples, those of Newcastle-on-Tyne, South London, and Golden Square.

Newcastle and Gateshead. The following table exhibits the mortality from cholera in the periods 1851-2, 1849, and 1853:—

Newcastle.

Year.	Actual mortality.	Population.	Deaths to 10,000 population.	Remarks.
1831-2	801	42,760	187	No water works.
1849	295	71,847	41	Good water supplied.
1853	1533	86,114	178	Bad water supplied.

Gateshead.

1849	Comparative immunity			Water works and water good.
1853	433	26,000	166	Bad water.

Both Newcastle and Gateshead are supplied by the same water company. Previously to 1832, there were no water works; subsequently, these were made, and water was obtained from the Tyne, about a mile above the town, although the tide flows six miles upwards beyond it. In 1848 these water works were abandoned, and excellent water was supplied from a small stream ten miles above Newcastle, called Whittle Dean. In 1853, the water from Whittle Dean being insufficient for the wants of the town, the original water works of 1832 were reopened, and thus bad water mixed with the good, so bad as to contain 7·1 parts of organic matter per gallon. (Dr. Thomson says at its origin there was 15·6 total impurities, of which 4·5 were mechanical, 2·68 organic in solution, and 8·48 organic impurity.) Thus, according as the water supply was good or bad, so the mortality from cholera was low or high. Again, the greater number of deaths, viz., 1011 out of 1553, occurred at Newcastle from 13th to 23rd September, inclusive. The reason was this. Owing to the outcry, the company supplied, after the 15th, no more Tyne water; and although

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the Tyne water was not out of the pipes till the end of a day or two, the deaths decreased from the 19th.

Sept. 12th	38	Sept. 18th	103
„ 13th	59	„ 19th	111
„ 14th	90	„ 20th	85
„ 15th	106	„ 21st	68
„ 16th	114	„ 22nd	82
„ 17th	103	„ 23rd	60
	510		509

It is also to be remarked that places supplied with pump water, and not with that of the company, at most suffered from simple diarrhœa, and not from cholera. Thus, in the workhouse, supplied by the company, out of 440 inmates, the number of deaths was 7 out of 12 cases of cholera. In the barracks, with 590 inmates, and in Greenhow Terrace, supplied by wells, there was no cholera, only diarrhœa. (See Dr. Snow's work.)

South London District. This district was supplied with water from two sources, the Lambeth Company and the Southwark and Vauxhall Company. In 1849 the former company got its water from the Thames, near Hungerford Market; the Southwark and Vauxhall from the same river, near Battersea fields. In 1854 the water of the latter company was obtained from the same place, while the former procured it from Thames Ditton. In 1849 the mortality was nearly the same in the districts supplied by each company. In 1853, the deaths were, in those parts supplied by the Southwark and Vauxhall Company, 525; while only 94 occurred in those supplied by the Lambeth Company, and 33 in those districts supplied from pump wells, and other sources. This mortality being divided over two periods, the first four weeks give 286 cases against the Vauxhall Company to 14 against the Lambeth; while, for the remaining three weeks, the deaths were in the proportion of 8 against the former to 1 against the latter. In the General Report of the Board of Health, speaking of the supply of water in London, is the following paragraph: "Those supplied by the Southwark and Vauxhall and Chelsea Companies greatly surpass the others in badness. In the Southwark and Vauxhall water, the evidence of unfiltered contamination reaches its highest degree, revealing to the microscope not only swarms of infusorial life, *but particles of undigested food, referable to the discharges from human*

bowels." (p. 46.) And it is this last contamination which propagates especially the disease, and which is plentifully supplied by the admixture of sewage matter.

Golden Square. The last example to which I shall allude is that afforded by the Golden Square tragedy. As Dr. Snow remarks, "The mortality in this limited area (*i.e.*, within two hundred and fifty yards of the spot where Cambridge Street joins Broad Street) probably equals any that was ever caused in this country, and it was much more sudden, as the greater number of cases terminated in a few hours." (*Op. cit.*, p. 38.) Upwards of five hundred deaths occurred in ten days. I cannot follow the history of the cases, which have been so ably drawn up by Dr. Snow. I can only allude to a few points. The outbreak commenced on the night between the 31st August and September 1st, and was to be traced to the drinking of water from a particular pump in Broad Street. In the subdistrict of Golden Square, Berwick Street, and St. Ann's, Soho, the deaths registered for the week ending September 2nd were 6 in the first four days, 4 on Thursday 21st, and 79 on Friday and Saturday 22nd and 23rd. Into those occurring in these last three days, 83 in all, Dr. Snow made close inquiry. In 68 of these cases the patients had all partaken of the pump water. In a coffee-shop in the neighbourhood, frequented by mechanics, and supplied with this water at their dinner, by the 6th of September ten of its frequenters had died of cholera. It was partaken of by a gentleman and his brother in Poland Street, both of whom died. It was partaken of by a lady and her niece, living at Hampstead, who sent daily for this water from preference, and both died of cholera. Two remarkable instances of comparative exemption in the same locality of persons not partaking of this, but of other water, prove the same truth conversely. In the workhouse in Poland Street, out of 530 inmates, only 5 had cholera; the other cases of cholera were brought from without. Here the Broad Street pump water was never partaken of; but there was a well on the premises, besides a supply of water obtained from the Grand Junction Water Works. Secondly, in the brewery in Broad Street, out of 70 workmen employed, only two had slight attacks of cholera. The reason was, they drank little or no water, but malt liquor, and, besides, there was a well on the premises. The pump water was, then, the cause of this cholera; and it has now been shown that, on the day preceding the outbreak, a boy had had

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cholera in an opposite house, the water closet pipe of which passed about three feet from the well pipe, that the mortar work between the two pipes was defective, and that thus there was a communication allowing admixture of their contents.

So far, therefore, as these facts go, they appear to shew that the choleraic matters in solution in water do communicate the disease. But the mass of other evidence in the same direction, recorded by Dr. Snow, has proved this to demonstration. The immediate poison being a ferment, hitherto undiscoverable, because too minute even for the most powerful microscope of the day, Dr. Snow calls it a cholera cell; but, after all, the action of this cell is the same as that of a ferment. It is a distinction of words without a difference in meaning.

The examinations of waters in choleraic seasons may be thus detailed; and I have adopted a tabular form especially, as bringing the points of difference between the water from our companies, or from wells, or pumps, more saliently forward.

Water Companies.	*Pump Water or Wells.*
1. Contained organic matter, dead and living, annelida, entomostraceæ, infusoria, confervæ, dromidaceæ, diatomaceæ, *fungi*, hairs of animals, starchy matter, dead and decaying fragments of vegetable tissue.	1. The same, excepting hairs and starchy matter, among most wells. But *deep* wells, and spring water, were remarkably free from organic matter. In some there was total absence. In a few, as Wadden water, and Painham water, there were a few confervæ, dromidaceæ, and diatomaceæ, but no *fungi*.
2. No animal peculiar to cholera was found. One kind only gave room for suspicion, but it has been found since in harmless waters; viz., a brown, *fat, lenticular*, moving, spiral-like body.	2. *Ibid.* This body was also found in the Golden Square pump water.
3. Vibriones also abound.	3. The same.
4. The purest of the metropolitan waters was that of the Lambeth Water Company; the impurest was that of the Southwark and Vauxhall. In the latter, there were visible quantities of fæcal matters.	4. In some sewage matter was found. In the water of two pumps especially in Putney—in Price's Folly and Creek Buildings.

5. In some of the water of the Southwark and Vauxhall, Lambeth, and West Middlesex Companies, animalcules were found, which exist only in brackish waters, showing admixture of sea water, viz., the tintycnia trigma, melosura nemuloides, and coscinodiscus eccentricus.	5. Not found.

Typhus and Typhoid Fever. These are two diseases which have for centuries infected this country, and which may, I fear, be traced to a similar origin—cesspool water. In proof of this, I shall first adduce the example of the fever at Hastings, which I before detailed to the Society. The first case occurred in a large room in the uppermost story of one of the largest and best ventilated houses in St. Leonard's, on the Hastings side. There does not appear to have been any other cases of typhus in the place before, and I am also bound to add that this patient had not, so far as could be collected from inquiry in this point, been exposed to typhoid contagion. In this house, however, a woman—the cook to the family, and the inhabitant of this upper room—was seized with fever, which soon passed into malignant typhus. Owing to insufficient attendance, and by reason of the water-closet being placed two stories lower down, the evacuations from this woman's bowels, which were fearfully offensive, were frequently retained in the room for nearly a whole day before removal. The crisis in this fever was, in fact, a copious diarrhœa, during which the pulse fell from about 160 to 90 in the course of a few hours. Six persons who were living in that house caught the fever, and one gentleman died. All had feverish symptoms, accompanied with copious vomitings; though, in some, the disease fortunately stopped there. I learnt, subsequently, that after the house had been purified, two new visitors, who took the lower rooms, were also seized with fever. The origin of the disease was traced to a direct communication betwen the cistern of the water drunk by the inmates and the pipe from the water-closet. As the house was beautifully ventilated, and there was no communication between many of those who caught the fever, it is most probable that the disease was produced in the sick by the drinking of the polluted water. A similar result has been observed in other cases of typhus. My information, however, on this subject is necessarily meagre. I applied to

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Dr. Corrigan, of Dublin, to know whether such an effect had ever been noticed in the Richmond Fever Hospital. "In a fever hospital", he remarks, "there is such cleanliness observed, that there is little exposure to the action of faeces, and the purity of the water supplied would certainly give no room for this source of contagion." The Croydon typhoid fever, which proved so fatal, was due to the water being impregnated with drains of a very foetid character. This fact has been equally dwelt upon by Drs. Snow and Richardson. Croydon is gravelly in its soil, and thus perous to the transudation of liquid matters. The place, moreover, like most country places, abounds with cesspools. Up to the beginning of 1852 the water supplied was obtained from shallow wells and stagnant ponds. In 1852 these were abandoned, and the water supplied from a deep well, dug fifty feet in the chalk, worked by a steam engine. Towards the end of 1852, this supply was interfered with, owing to the new drainage works established by the Board of Health; the inhabitants had recourse to the old pumps, wells, and ponds, the former contaminated with cesspool matters, and the result was a virulent epidemic of typhoid fever. In the following account of an epidemic, quoted by Dr. Jenner in his Gulstonian lectures, the same origin probably existed. The facts are given on the authority of Dr. Hunt, of Buffalo.

At North Boston, Erie County, U.S., in 1843, resided nine families. Taking a tavern for the centre, seven of the nine lived within an area of one hundred rods in diameter. All the inhabitants, with the exception of the members of one family, were in the habit of frequenting the tavern. A feud existed between the master of that one and the tavern keeper. A man labouring under typhoid fever—a disease previously unknown at North Boston—took up his residence at the tavern September 21st, and died October 29th. Between October 19th and December 7th, twenty-eight persons in this little community had typhoid fever. Three families only escaped the disease, viz., the two residing the farthest from the tavern, and that of the man who had a quarrel with the tavern keeper, and, consequently, never visited at his house. Now, a fact of interest in this case is, that all the families in which the disease appeared drew their supply of water from the well of the tavern, while two out of three that escaped had their water from other sources. The man at feud with the tavern keeper was accused of having poisoned the well of

the tavern. He resided nearer than any of the others to the tavern. None who visited the village simply for the purpose of rendering assistance to the inhabitants contracted the disease. (Gulstonian Lectures by Dr. Jenner, *Medical Times*, March 12th, 1853, p. 26.)

It is true in this case, the necessary link, that the first patient's fæcal matters had been mixed with the well water, is wanting; and yet it seems to afford a probable explanation. Moreover, when we consider that the well and the cesspool are generally, in country places, in the same yard, and often close to one another, the admixture may sometimes occur through a porous soil, and in this manner occasionally explain the occurrence and spread of disease. Dr. Sutherland (*Report*, p. 19) remarks, "Much of the evil resulting from the close proximity of rivers and canals proceeds from the infiltration of the subsoil, and not merely from the aqueous vapour, which rises from the surface of the water itself." Speaking, moreover, of the Thames, he says, "Besides the evil resulting from imperfect declivity, the sewers are back watered at high tide, and actually become the means of distributing a polluted and unwholesome drainage through all their ramifications, by which the whole subsoil becomes infiltrated with impurities." It is also clear that any imperfection in a sewer, a broken brick, for instance, in connexion with a similar imperfection in a pump close by, may lead to the drinking of contaminated water by a whole neighbourhood. Now, I have remarked that such waters may lose all smell, and even appear to be quite clear, and yet be poisonous from matters held in solution. I remember the example of the common sewer of Fontainebleau, which passes through the park, which is of a gravelly and rocky soil, and which, at times, after about half a-mile's course as a rivulet, is in appearance perfectly clear, limpid, and inoffensive in odour.

The same fact is mentioned by the Committee on Dysentery of the American Medical Association (vol. x, p. 568). Speaking of Seconsia, in Calhoun County, it is remarked that the water in the streams was very low, emitting an unusual smell from a quantity of decayed fish having been thrown in it in July. A long time after *men* ceased to detect any offensive odour in it, some *horses* would not drink of it below the bridge, but hurried above it, and drank heartily of the stream there. The first case of dysentery occurred in this neighbourhood. Mr. Foote, in Drs. Arnott and Kaye's *Report on the Sanitary Condition of the Labouring Classes in and about the Metropolis*, remarks, "Two years ago a fever raged at Red Hill, which I attributed to the lodgment of the filth from privies, which I had removed at the time; the same thing occurred also at the Hyde, the fever prevailing there being of the typhoid type. Again, another medical officer writes that fever has been most prevalent in those courts and alleys where there is no free circulation of air, such, for instance, as Rosemary Lane, in which there are about twenty houses, in almost every one of which fever prevailed. The disease first made its appearance there in the month of August last; and, on my first visit, I found the intolerable nuisance of the overflowing of a cesspool, or privy, which continued for some time, there being no sewer to carry off the soil." (p. 4.) Now, a very little reflection explains how a sudden shower of rain would cause these fæcal matters to be carried down in the neighbourhood of wells, so that a very contaminated and impure water came to be drunk, through which fæcal matters might be absorbed in the system. Such may also be the case in Munich.

Diarrhœa is, as is well known to many, a disease frequently generated by the drinking of water contaminated with sewage matter. Indeed, where organic matter in solution in water abounds, this effect is usually produced. This was the case in Croydon, where a severe diarrhœa in the Park Lane School was traced by Messrs. Carpenter and Westall to the admixture of sewage matter with the well water drunk by the inmates. The frequency of diarrhœa in choleraic times, from the drinking of bad water, is also proverbial, such water containing offensive matters in solution, mostly derived from night soil, or other refuse matter. This same effect is commonly produced by the water of many of the inland lakes of Lower Canada, in America. These are, for the most part, very deep, and abound with fish of all kinds, whereas the rivers which issue from them are very narrow, so that the surface only of a lake is removed. The waters are generally brown in colour. When a boy, I remember, half a tumbler of the water of one of these, about fifty miles from Quebec, in the summer season, was, in our happy ignorance of the cause, the usual aperient —no doubt the animal, vegetable, and fæcal matters, in solution, contributing, along with other organic matters in a state of fermentation, to produce purgation. The effects, in many instances, resemble those occasionally produced by vegetable matters in particular states of

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decomposition, also in solution in water; and probably hereafter it may be found that in both cases the ferments are identical—protein bodies, such as caseine, albumen, and gluten, which also produce analogous diseases. The following example, taken from Pereira's *Treatise on Food*, illustrates this point. In the course of a trial for damages, at the Nottingham assizes, it was proved that a very severe epidemic of dysentery, which had occurred among some cattle, was caused by their having partaken of water impregnated with the putrescent vegetable refuse from a starch manufactory. All the fish and frogs contained in it died. The amount of disease in the cattle, moreover, bore a direct proportion to the amount of putrescent organic matter contained in the water, and taken by the animals. The mere putridity of the water could not have produced the disease. The water of ponds in fields is often very putrid, and yet full of frogs and fish, and the cattle partake of it with impunity. There was, therefore, some ferment, most probably gluten, in this refuse, which, developing in the water a poison, generated disease on all who partook of it. This is the only explanation which offers itself; otherwise we must suppose that, the fæcal discharges of an affected animal having first mingled with the water, the water fermented the morbid principle, and the disease spread in consequence; and this, after all, is not an improbable supposition. This effect is analogous to that kind of diarrhœa produced in infants whom it is attempted to bring up by hand, and in whom the food, whether animal or vegetable, passes off in a state of putrefaction in most offensive stools, the foster of which it is almost as difficult to describe as their production is difficult to restrain, even by the most energetic medicines.

Dysentery has been proved, in many instances, to have been produced by fæcal matters in solution. The previous case, detailed under the head diarrhœa, as an instance of the second supposition, is correct. Bad water is a frequent cause, at any rate, if contaminated by organic matter in a state of decomposition. Dr. Snow alluded, in his book on *Cholera*, to the occurrence of dysentery from bad water in Millbank prison. Dr. Bryson mentions cases in which dysentery was produced by the waters of the Yangote Kiang, the Canton river, and other rivers in China—and in which nearly all the patients were affected with great numbers of lumbrici. Zimmermann describes this appearance of worms in the evacuations of the patients affected with dysentery in the epidemic of 1765, in Switzerland, as a very bad sign—*signe funeste*. These worms were no doubt taken as ova into the system chiefly in water, and afterwards developed in the body. A fact came to my notice, in which two deaths occurred from the use of impure water at Filey in Yorkshire. The drainage at Filey, as late as September and October last, was very bad. It is the practice of the fishermen also to wash their fish in water within the town. These drains, at certain periods, therefore, in their downward course to the sea, emit a fearful odour. At other times, however, it is not so. On one of these latter occasions, when this sewage water was free from smell, through the carelessness of a domestic, a portion of it was administered to a family, and two deaths resulted very speedily. The symptoms were magnified by some into cholera; but, from all I could learn, they were rather those of dysentery. The other members of the family also suffered, but the effects produced were, fortunately, not fatal. From what I have been able to learn on the subject of the dysentery in the Crimea, I think the presence of drain or fæcal matter in solution in the water drunk had a great deal to do with the production of dysentery. It very readily yielded to the drinking of bitter ale; negatively, I presume, by the substitution of an uncontaminated drink for water, which, through the rainy season, washing down the fæcal excrementations of a camp, must have been more or less impregnated therewith.

Plague, although, in most cases, to be traced to emanations from dead bodies in those cases where it appears to have been generated sporadically, is occasionally, so far as we know, produced by solution of sewage matter in water in those countries where the disease is usually rife, and where it might be said to be endemically prevalent. The waters, in a low district, are, for this very reason, more deleterious in a country without common sewers than in higher districts, where the admixture is not so easy. The observation that fæcal matters are capable of retaining the poison of plague, belongs to an old writer, Mercurialis; he states distinctly, that the poison of plague may be preserved in the excrements, as well as in clothes, and this without any actual injury to patients themselves, but that it cannot be preserved in living matters. (*De Peste*, p. 54.) Cairo is, moreover, a hotbed for plague. Now, in the midst of this town there flows a large open canal, or common sewer, —for it is nothing else, if we look to the carrion and fæcal filth it contains,—and yet as the Nile overflows, and mixes with

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this sewer, portions of it are drunk with the water, and this gives rise to plague. And it is remarkable to notice how fearfully this disease spreads where the supply of water is bad or scarce, and where, necessarily, the water becomes contaminated the more readily. Such was the case in the fearful epidemic which broke out in Malta in 1813, and which never yielded to the most severe prophylactic measures that could be adopted, but was only stopped by the compulsory removal of the inhabitants into another part of the island, where we may suppose their food and drink were no longer contaminated. Indeed, to non-contagionists, the above is the only practicable explanation.

Yellow Fever. In regard to yellow fever, and its origin from bad water, there are, perhaps, no very strong facts to prove it. Still there are some negative facts, which may give a colour to it; one in particular, to which my attention was drawn in reading Dr. Richardson's able paper in the *Journal of Public Health* on the subject of Water Supply in this Metropolis, and from which article I have largely drawn in the argument of this paper.

In the year 1853, a very fatal fever broke out in Bermuda, which destroyed at least 663 persons, from July 1st to December 31st, out of a population of 13,800 persons—*i. e.*, in round numbers, 1 in 20. The mortality was greatest in St. George's parish and Hamilton. In St. George's and Hamilton parishes, the surface drainage only is attended to; there are no sewers; and the use of cesspools is universal. Hamilton has a population of 1900 persons, in 426 houses, four persons to each house, each house being provided with a tank to receive rain water. There are three or four wells in the town, the water from which is very bad, and used only by the lower orders of people. The cesspools are from fifteen to twenty feet deep, and never cleaned out. The shallow cesspits are cleaned out once a year, and then the contents buried in a hole or emptied on the beach. All dirty and waste water is generally thrown in the yard. The soil of Hamilton is of a light calcareous nature. The soil of St. George is of two distinct qualities, one part composed of a stiff reddish clay, with a limestone bottom; the other and greater portion, light and sandy, particularly porous, admitting immediate absorption of the heaviest fall of rain. In St. George's, there is no surface drainage or sewerage whatever; the population is 1440, in 440 houses—*i. e.*, six in each house. Cesspits are used, but the night soil is frequently deposited on the surface in all

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directions. Water-butts are very uncommon, and many houses have no tanks. There are two or three wells, with very bad water. In this manner, a vast amount of night soil penetrates through the porous soil, or, floated to the sea, gives rise to the most offensive odour in the waters on that shore. For the very same reason, and owing to the proximity of the cesspools and the tanks, these are frequently mixed, and the water becomes very offensive. It was thus that the tank water at Fort Cunningham at last could not be drunk. As an evidence of the impregnation of the soil with fæcal matters, it was remarked that the submarine soil where the *Thames* hulk ship was placed was contaminated, so that the odour evolved during its violent commotion in a gale of wind was well nigh intolerable. In the *Minutes of Examination of the Sanitary Condition of Ireland* (No. 1, art. 3), it is stated: "The water in nearly all the tanks was found impregnated with sulphuretted hydrogen gas, and was only used by the inmates for culinary purposes, the water for drinking being procured from other sources." And again: "The tanks are also too close to the houses and to the privies, and thus receive impurities which affect the water." (p. 74.) This state of things (although especially applicable to Rows 3 and 4 of the mechanics' houses, being nine dwellings on the glacis) is yet sufficient to prove the character of the water partaken of, at least for culinary purposes, in which manner much poison could have been transferred to the alimentary canal. It is not everything used for alimentation, or every vessel cleaned which is previously submitted to a temperature of 212°, at which temperature ferments are destroyed. And it is a peculiar property of such localities, when once encountering an epidemic influence, to develope the poisonous ferment very rapidly, as before shown, in referring to Surgeon Jones's opinion. (See page 28). Hence one reason why, after a time, an infected locality becomes very dangerous, and all more or less pass under its influence. In the Boa Vista fever, although the natives suffered but little at first, afterwards they did suffer, and then more severely than the whites—probably because the whole waters drunk by this town were so impregnated with fæcal matters as to be too poisonous to be resisted. A very rainy season preceded this outbreak of fever.

I might have extended this paper in speaking of bad water in its effects in producing some other diseases; but,

as Dr. Richardson has so ably done this already, in his paper on the Supply of Water in London, I feel this is unnecessary now. I must however say, that it is possible the effects of the same poison in water or as an emanation may produce occasionally a different effect. But this is no proof of their non-identity. It is quite possible, where two allied diseases coexist, that the one may be produced by the ingestion of the same poison in water, the other by its inspiration as an emanation; and thus we may in the end be able to carry this distinction one step further. If we judge from the effects of medicine taken by the mouth, and inspired as a gas, the difference is marked, varying often not only in degree, but also in kind. Ether, chloroform, and alcohol, inspired, will often produce, in very small quantities (a few drops), effects as powerful as ounces taken by the mouth. The effects, however, vary in kind. Carbonic acid is a well known instance. The tincture of sesquichloride of iron inspired will produce faintness and vomiting, which it will not do taken by the mouth. The powerful and almost celestial feelings of inspired opium and cannabis Indica are frequently not even in degree producible by any dose taken by the mouth. In like manner, it is not impossible that the same animal poison, according as it is inspired or taken by the mouth, may produce very different results.

Such are some of the grounds which lead me to believe that fæcal matters may undergo in water changes such that, in their accidental ingestion by the mouth, they may prove injurious to the organism, and, either without further transformation or by additional changes taking place in the alimentary canal, prove the cause of disease. There is, however, this difficulty in tracing out these changes; it is not always possible to prove to demonstration that the emanations from morbid fæcal matters may not have likewise co-operated. Facts are often indistinctly recorded, where, with a little of Dr. Snow's perseverance and exactitude, precise ground for accurate conclusions might have been given. This is especially the case when biassed men are employed, or when the medical element is not sufficiently mixed in a board of commissioners, called upon to investigate the causes of epidemics. Much, however, I trust, has been stated to prove that fæcal matters in solution often produce fatal results, in cholera, dysentery, diarrhœa, and typhoid fever, and probably also in yellow and other bad intermittent fevers. If this be so, then the removal

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of any sources of this infection, especially in the supply of water provided to communities, is imperatively called for, and should be enforced by the most stringent sanitary regulations.

PART III.

INJURIOUS INFLUENCE OF FÆCAL EMANATIONS.

In my first paper, I alluded to some of the contingent phenomena which accompany the evaporation or emanation of miasmata. I dwelt particularly on four of these: 1. A comparatively high temperature for the period of the year. 2. A light atmosphere. 3. Moisture in the air. 4. Ammonia, always present in the decomposition of animal and vegetable matters, and in contagious diseases.

The co-existence of the first two phenomena is also insisted upon by an able writer as co-operative in coal mine explosions. I allude to the very interesting paper by Mr. Dobson of Cambridge, on the influence of revolving storms in the escape of inflammable gas in coal mines. The propinquity of these storms is always marked by a *rise in the temperature and a fall in the barometer*. These storms have generally a south-west direction, and as they pass over a country, so, under the diminished atmospheric temperature, the inflammable gas in the mines escapes. In an opposite state of atmosphere, i. e., a fall in the temperature and a rise in the barometer, explosions seldom occur. And so it is with emanations productive of disease; only that moisture and ammonia are superadded. Indeed, I think it may be stated as a law in nature, that, whereas a moderately low temperature, dry weather, and a heavy state of the atmosphere, favour the deposition towards the ground or into water of poisonous miasmata, so a warm temperature, damp weather, and a light atmosphere, favour the ascent of these as emanations. But I believe more than this, that at certain low temperatures emanations are impossible. When I made this statement in my last paper, the truth of it was called in question; yet the statement is in accordance with fact and experiment; for then the fourth condition which I spoke of, is impossible, viz., the

evolution of ammonia. Neither vegetable or animal matters will decompose at a temperature below 32° Fahr., and even many degrees above that temperature. The ammonia which will not *rise* in vapour will be kept to the surface of the earth, even if found at all; and the same is true of watery vapour, though not to the same degree. A cubic inch of air, which at 0 Fahr. can only contain 0·856 grains of moisture, at 95° Fahr. contains 17·009 grains. The amount contained at 11° Fahr. is almost 0, while at 50° Fahr. 2.5 grains are contained. Hence the reason that in wet or damp weather the offensive vapours of a town are greatest. These odours, as I stated in my former paper, are, if smelt powerfully, conversely a sign of rain. What holds for vapour is equally true, therefore, of miasmatic emanations. This view explains readily why it is that in very cold weather, when the atmosphere is always most dense and heaviest, driest and most free from ammonia, we have precisely those circumstances present which are most unfavourable to the spread of disease arising from poisonous emanations, etc., which, experience proves, usually disappear at such periods. We may, therefore, I think, safely conclude that in proportion as the barometer is high or low, and the atmosphere dry or moist, so will miasmata remain on the ground or rise, and the diseases arising therefrom decrease or increase. But I think we may be justified in going further still, and reasoning from these antecedents, to lay down *à posteriori* this additional rule, that if a disease spreads in very cold weather, that weather being dry and the barometer high, the said disease cannot be generated by *miasmata* in solution with air, but is either contagious or propagated by poisoned water or other ingesta; and *vice versâ*, that if a disease spreads in damp hottish weather, with low barometer, it is most probably generated by *miasmata*.

So true is the concurrence of dampness in the generation of many epidemic diseases, that Dr. Barton, of the United States of America, has been led therefrom to form a theory on yellow fever, which is very ably supported by Dr. Hunt, of Buffalo, in the recent number of the *Transactions of the American Medical Association*. Without the conjunction of dampness, and what he calls *terrene* causes, no epidemic can occur. These *terrene* causes are any which may give rise to miasmata, such as upheaval of soil, decaying vegetable and animal matters, filth, low stagnant recesses, etc., etc. This theory has, moreover, the advantage of reconciling facts which were before in apparent contradiction, but which now appear to be in strict accordance one with another.

My friend Dr. Snow, I believe, while he does not deny that emanations may give rise to cholera, yet believes they have been raised in the air by watery vapour, in which water they had acquired their poisonous character, having been first voided in the evacuations of affected persons. This may or may not be true, but it at any rate directs attention to *watery vapour* as a chief agent in the transmission of the poison.

Together with this watery vapour, and ammonia, there are probably other gaseous compounds which, by their presence and quantity, influence or modify the character of the malarious poison in its action upon man. Unfortunately their precise nature has been of late but little studied specially, which is the more to be regretted, since organic chemistry has progressed so much. No doubt, like the perfumes in the atmosphere, of which we may become sensible from the sense of smell, and yet which we may be unable to detect by chemical agents, they are too subtle to be made out. Still there are particular symptoms which concomitant gases produce, which so far resemble these in their effects, that we are warranted, I think, in referring some of the results produced by contagious poisons, at least, to their concurrent action. This is especially true with regard to sulphuretted hydrogen; but carburetted hydrogen and also phosphuretted hydrogen may produce analogous effects. Of the two poisonous gases which occur in common sewers, described by Thénard, and before alluded to, the results of the first (oxygen, 14; nitrogen, 81; carbonic acid, 2; and sulphuretted hydrogen, 3) are not necessarily fatal, unless their inspiration is long continued; and the symptoms are those then of poisoning by sulphuretted hydrogen, being gradual prostration of the physical powers, giddiness, sickness, and general debility, finally emaciation and low fever; but the *post mortem* appearances are very similar to those observed in the worst forms of typhus. The second variety of gas found in common sewers (oxygen, 2; carbonic acid, 4; and nitrogen, 94; with more or less ammonia) produces its effects more suddenly, being of course quite irrespirable if it contain much carbonate of ammonia; or, if this be absent, killing by asphyxia, from the oxygen being in insufficient quantity.

A great deal is as yet unknown as to the effects of car-

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buretted hydrogen and phosphuretted hydrogen when in a more or less diluted state in the atmosphere. That both should prove fatal if breathed singly can be readily understood, but in what manner they prove injurious when diluted with various proportions of atmospheric air, is not made out. Carburetted hydrogen breathed in small quantities has been recommended in cases of phthisis, catarrh, and hæmoptysis, though upon what theoretical grounds I cannot tell. As it is always present among the gases disengaged from swamps and stagnant waters, although in these cases mixed always with carbonic acid and nitrogen, it has been considered by some as the active agent in the production of fever from malaria. I believe this opinion is entirely without proof; but it is probable some of the effects of it might be inferred from the diseases peculiar to coal miners.

Of phosphuretted hydrogen still less is known. There are three compounds of phosphorus and hydrogen at least. The more inflammable, the phosphuretted hydrogen, which is emitted in the putrefaction of organic bodies containing phosphorus, which is the cause of the peculiar odour of decayed fish. Phosphorus we know to be a deadly poison; and if we were to reason from the analogy of arseniuretted hydrogen, we should be led to attribute very deleterious influences to any emanation containing it in admixture.

In connexion with the action of phosphuretted hydrogen, it is interesting to remark that a peculiar low fever is often said to be induced by the decomposition of fish and seaweed on the sea-shore in some watering places at certain seasons of the year, in which emanations the phosphuretted hydrogen exists. Liebig denies that this gas is ever generated in the decomposition of animal bodies, either in disease or in the putrefaction of dead bodies; and what is more important (he adds), "the human body contains no phosphorus in such a state as to yield by any process during life or after death phosphuretted hydrogen gas." (Liebig's *Letters*, Spont. Combust.) The presence of some phosphorised vapour is however undoubted, as it exists occasionally around parts in a state of ulceration, or rapid decomposition, as in dissecting rooms when accompanied with light, or in rare cases of phthisis with luminous breath. At any rate, from Dr. Ayre's experiments, we may infer that these gases are present in the atmosphere around putrefied fæcal matters.

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According to M. Pariset, however, "The immediate products of decomposition are not simply elementary bodies, as hydrogen, carbon, and nitrogen, but, on the contrary, vapours, the organised condensable molecules of which are capable of being completely disorganised by a combination of heat and moisture, drawn along by gases, aqueous vapours, currents of air and dust, dispersed without being destroyed by pure water. Desgenettes believed that, expelled by sulphuric acid, they are only neutralised by the powerful alkaline bases, lime, potass, soda, ammonia, cinders of vegetables. They are condensed, on the contrary, and not decomposed by porous bodies, among which fine sand may be classed. While they preserve their peculiar organisation they can serve as ferments, and consequently act upon living beings to disorganise them, finding in them the degree of heat and humidity which ought to destroy them; they are not destroyed and do not undergo these metamorphoses but by making the being who has received them a partaker, whose life is thus compromised, sometimes destroyed. . . . When the elevation of temperature makes the ferments evaporate, man receives them by the superficial absorbent vessels, or by those of the lungs in the act of respiration." (*British and Foreign Medical Review*, vol. xxiv, p. 249.)

The precise amount of heat required for the development of this fermentation varies probably with each disease; it may, however, be generally stated, that a very high temperature destroys it, as well as a very cold. In yellow fever, Chisholm makes the temperature from above 60 to 90, below or above which it is inert.

There can be no doubt, however, that something more is required even in the due development of these diseases, and it is probably to be found in some *electric condition*. We know, for instance, that every man possesses electricity in his organism. The skin gives positive, the mucous membranes negative electricity. The atmosphere likewise possesses one kind of electricity, while the earth possesses the opposite. Man lives thus between these two in a kind of intermediate state, in which both electricities are in equilibrium, and thus escapes injury. This is especially true in dry weather. But there are at least three causes which may interrupt his immunity—all connected with several natural processes in action.

1. *Vegetation* developes positive electricity. Now, as before seen, this kind of electricity favours decomposition. Hence, if it be abundant, as in those instances where vege-

tation is rank and rapid, in a moist atmosphere, then disease may be rapidly developed.

2. *Evaporation* also developes in the same way positive electricity with the same deleterious influences; the earth remaining negative. Hence usually pestilential miasmata arise with evaporation; when this is greatest, disease is most rife, as when a hot season follows heavy rains. It may be opposed to this view, that positive electricity also augments with height, and yet mountainous regions are, as a rule, healthiest; but then, the temperature is also lowered, the air more rarified, and therefore containing less oxygen in a given volume, and lastly, more agitated by winds. Hence the miasmata, being weaker and not stationary, cease to be injurious.

3. Under certain circumstances, *clouds heavily charged with electricity* approach the earth. These, by disturbing unduly the equilibrium of man's organism, prove injurious. We may explain in this manner the peculiar sensations experienced by some persons in thunderous weather. If this disturbance persist long, and especially if the air contain much moisture, so that the electricity is easily communicated to persons, then the chemico-vital relations of the body may be seriously influenced, and disease result. Mists and damps are frequently associated with typhus; and in typhus, the normal electricities of the skin and mucous membranes are reversed. It may hereafter, perhaps, be shewn that the fermentative and diseased changes of mucous membranes are connected in this disease with the kind of electricity they have acquired. (See *Brit. and For. Medical Review.*)

We are now in a position to speak generally upon this part of our subject, reserving the individual instances where specific disease is generated for more particular inquiry when we speak in order of the individual diseases themselves. That town refuse and night soil give rise to the most fatal emanations is, I think, fully established by the researches of Mr. Grainger, Drs. Lewis and Sutherland; and yet, as Mr. Grainger informs us, many of even the educated classes believe now that they are innocuous and also curative of disease; innocuous, because many have been exposed to them, and yet have escaped disease; and curative, because the atmosphere of a cesspool is said to cure hooping-cough, and that of a cow stable consumption. So far may popular prejudice deceive even the most educated.

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Here are a few instances taken from the *Reports on Cholera* of the Board of Health (p. 41). Immediately opposite Christ Church workhouse, Spitalfields, belonging to Whitechapel union, and only separated from it by a narrow lane, a few feet wide, there was in 1848 a manufactory of artificial manure, in which bullock's blood and night soil were desiccated by dry heat on a kiln, or sometimes by mere exposure of the compost to sun and air, causing a most powerful stench. The workhouse contained about four hundred paupers. Whenever the works were actively carried on, particularly when the wind blew in the direction of the house, there were produced numerous cases of fever of an intractable and typhoid form; a typhoid tendency to measles, small-pox, and other infantile diseases; and for some time a most intractable and unmanageable and fatal form of aphthæ. From this cause alone twelve children died in one quarter. In Dec. 1848, when cholera had already appeared in the union, sixty of the children were seized with violent diarrhœa in the early morning. The proprietor was compelled to close his establishment, and disease ceased among the inmates. Five months afterwards, the works were recommenced. In a day or two subsequently, the wind blowing from the manufactory, a most powerful stench pervaded the workhouse. The night following, forty boys were seized with severe diarrhœa, while the girls, whose dormitories faced in another direction, escaped; and the suppression of the nuisance resulted in the disappearance of the diarrhœa, and it has not since recurred. A similar effect was observed in St. George's, Southwark, in the summer of 1847, where a similar manufactory was established, and which also gave rise to most offensive effluvia. Diarrhœa, extensive and severe in type, soon appeared, which only entirely gave way when the nuisance was suppressed.

In the Potteries of Kensington there were kept, in 1849, 3,000 pigs, and as the process of fat boiling was also extensively carried on, an area of half a mile round was tainted by foul odours. The inhabitants there lived in a state of misery and filth which is indescribable. The houses were close to a pool of stagnant water, called the Ocean, which was covered with filthy slime, and bubbled with a poisonous gas. This Ocean was fed by the drainage of the pigstyes and privies. In this place, out of 1,000 inhabitants, in ten months of 1849, 50 died; 29 of fever, and 21 of cholera, *i. e.*, a mortality of 6 per cent.

per annum. Some twelve or thirteen hundred feet off, says Dr. Lewis, is situated a row of clean respectable houses, called Crafter Terrace, Latimer Road; the situation, though low, is clear and airy. On Saturday and Sunday, the 8th and 9th Sept. 1849, the inhabitants complained of an intolerable odour, the north-east wind blowing directly upon the terrace from the Potteries. Till this time, there had been no cholera among the inhabitants of the place. The next day, the disease broke out. Indeed, wherever there are fæcal emanations, it may be laid down as a rule that the place is unhealthy, and its effects debilitating to a degree. In such places, if a poisonous ferment be introduced, it will speedily develope and increase, and produce disease among those who are exposed to its action. Dr. Sutherland gives an instance in point in the case of the Witham suburb of the town of Hull, a space of some three acres, two acres of which are used as a deposit of the night soil and other manure. The town of Hull generally gives as the average age of death in other parishes, 23; in Witham it is 18.

All who visit among the poor must admit this; and here it may be as well to answer an objection to the argument made use of by Parent-Duchatelet, in regard to the healthiness of the inhabitants who live near Montfaucon in Paris. We are all aware that Montfaucon is the great receptacle of the fæcal evacuations of Paris, and dead horses, etc., which are all brought there in carts. Now it is alleged, that if fever could be generated from such a cause, the health of the workmen employed would be very bad, and they would be very obnoxious to fever, which is contrary to the fact. Indeed, if their state of health was to be taken as a criterion of its salubrity, this trade would be one of the healthiest for workmen. These *débardeurs*, it is stated by that author, are very rarely liable to intermittent fever; and those amongst them who have it were ancient soldiers of Africa, Spain, and Russia. It is not true that they are liable to cramps. Their colics are due to bad wine. The mania with which some are affected (those of Bercy) is due to white wine mixed with perry, drunk to the extent of six *litres* (about seven quarts) daily. Atonic ulcers are very rare among them; the only disease to which they are liable is *grenouille*, a peculiar affection of the extremities when placed in water. Typhus is very rare in this district as compared with others. The epidemic that prevailed in 1814 stopped on April 17th of the same year, and has not since recurred, though Montfaucon still exists.

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Duchatelet published his work in 1836; and I have not seen any statistics to prove the comparative amount of typhus and low fevers in Montfaucon now as compared with other districts. Assuming that these statements are true, and even applicable to the present day, the exemption is to be otherwise explained. These *débardeurs* are, as compared with other Parisian workmen, *unusually well fed*. They have animal food—most Parisian workmen live exclusively on vegetables: and although this animal food is horseflesh, and therefore may be distasteful to an Englishman, it is not the less a wholesome food. Now wholesome food is as powerful a prophylactic against fever, as bad food and fatigue, and mental depression, are favourable to its development. I believe Dr. Alison looks upon such influences as directly causative of fever. Dr. Corrigan, in writing to me on this subject, says: "We have lost six or seven clinical clerks within the last three years, and in every case, I believe, from exposure while the system was in a depressed state. In one case, the student went into the wards after sitting up all night; in another, after great exhaustion from three or four hours skating, etc.; and so impressed are our old porters with this, that they constantly urge upon the young clerks not to go into the fever wards when relaxed, exhausted, or tired out; and I always warn them *against going in fasting*."

Again, whatever effects the emanations have on the spot, it is notorious that puerperal fever is frequently generated in the lying-in wards of St. Louis's Hospital, in the Faubourg St. Antoine, when the wind blows from Montfaucon. This fact I have before mentioned, in my paper on Puerperal Fever. Whether these emanations acquire in their transit through the air the amount of oxygen and moisture necessary to ferment and putrefy, I cannot say, but the fact is undeniable. The spread of contagious diseases generally through fomites and the emanations arising therefrom, if soiled, is asserted on too many sides to admit of complete denial; and the liability of washerwomen to catch the diseases of those for whom they wash is almost universally admitted, and yet remarkable instances are given to the contrary. Washerwomen, says Dr. Corrigan, certainly do not suffer from their employment; nor do the nurses or ward maids employed in changing the linen, making the beds, etc. This contradiction of facts can be reconciled by two suppositions founded on fact, that either the parties exposed to the emanations therefrom are not fit recipients,

or in great measure to the chlorine employed, but particularly the strong alkali, the soda with which they wash, which destroys the poison effectively.

We will now notice some individual diseases which are capable of being produced by fœcal emanations.

Cholera. Pettenkofer is one of those, I believe, who states that diarrhœic fluids will, during choleraic seasons, develope through fermentation cholera poison; that this change depends much upon the nature of the soil—a damp soil favouring this fermentation, which a rocky soil in measure prevents; and that this cholera virus developes, according to the idiosyncrasy of the patient seized, cholera, cholerine, or simple diarrhœa. Whether this view be admitted or not, it cannot be denied that emanations from *cholera stools* will sometimes produce cholera. It has, I think, been fully proved by Drs. Alison and W. Budd, as is clearly shewn in the papers written by these gentlemen, and respectively published in the *Edinburgh Medical Journal*, Dec. 1855, and the Association Journal of 1854, that the poison of cholera is multiplied rapidly in the stools and fœcal matters generally, and acts chiefly in consequence of a particular decomposition which it subsequently undergoes in these, becoming therein so concentrated, that an emanation therefrom may produce in a recipient the disease. I think that one fact adduced is conclusive upon this point. Although the same water was drunk by all the inmates in a large workhouse, those only who made use of the same privies or receptacles for cholera dejections caught the disorder, while the recurrence of cholera seizures was effectually prevented by guarding the avenues leading to these, and by preventing persons not affected with cholera from using them. The miseries endured by our troops in the Crimea, who were obliged to encamp over the same filthy *locale* where the Russians affected with cholera had previously been quartered, is a sad confirmation of the same tale. Certain it is, that in Dr. Lindsay's hands, experiments made by him with the emanations from evacuations and blood, and from clothes saturated by the evacuations and breath of cholera patients, were followed by specific results. In his experiments, cholera was simultaneously developed in four animals, and proved fatal in two of these.

I am quite aware that instances at direct variance with these facts may be cited. Thus, in Mr. Marshall's paper before referred to, we read that Dr. Sarmichen, M. Foy, and

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others, had purposely inhaled the breath of cholera patients with impunity. Drs. Deynert and Mavroyen did the same at Moscow. Persons have slept in wards as well as upon cholera beds, and have even worn the linen of cholera patients with impunity. Again, dogs have been made to inhale effluvia from choleraic evacuations and blood, and from clothes worn by and saturated by the perspiration and breath of these patients. Rabbits have been placed in close places with choleraic stools about them, but escaped. But these apparent contradictions are yet, I believe, to be reconciled. There cannot be a doubt, I think, from Mr. Glaisher's experiments, that the air of choleraic wards is sometimes changed. I have before alluded to Dr. R. D. Thomson's experiments in which he found that the air of a cholera ward was acid; also that the external air in choleraic seasons was alkaline only in the sewers. Whether this be a constant change present or not, is open to future inquiry. Still the disparity of action would point to a change in the chemical character of the surrounding medium; but if the concurrent circumstances, the intensity of the poison, the period of the epidemic, the fitness of the recipient, etc., and the varied contingencies to which I referred in my first paper will not account for this, I am sure the hygrometrical state of the atmosphere will. I again repeat that, if the atmosphere be very moist and warm, it will easily convey infectious particles; and if it be dry and cold, it will as effectually prevent diffusion of the cholera poison as a solid metallic or other not porous substance.

Cases, at any rate, are not wanting in which fœtid emanations in choleraic periods have given rise to cholera. I have already alluded to some; but I may mention a few more, selected from the *Reports of the Board of Health*, which I prefer making use of as being official.

I have already instanced the case of the Kensington Potteries; I have also mentioned Witham in Hull. Dr. Sutherland says that 91 deaths from cholera occurred here on the outskirts of a triangular space of little more than two hundred yards. Dr. Sutherland adds: "I have never known an open neighbourhood of this size yield so large a number of deaths." The report goes on to allude to Merthyr Tydvil, Dowlais, and Pen-y-daran, all in the same neighbourhood, where cholera was very rife and most destructive. The instance of Brest prison is also very instructive. This prison contains 2,662 inmates, distributed in four wards and in an infirmary. The four wards have twenty-seven water-

closets, so that the prisoners in each row may be enabled to reach the closet without being unchained. These water-closets communicate with a drain which opens into the harbour of Brest. At low water, the south-west winds (remark here the *damp* winds) blow up the unguarded drain, and force back the mephitic vapours into the very wards. The infirmary and condemned cells are free from this inconvenience: 189 cases, with 113 deaths, occurred in the prison; a mortality on the seizures of 59 per cent. Out of 2,445 prisoners in the wards, 165, or an average of 6·7 per cent. on the whole number, were attacked with cholera: while in the infirmary and condemned cell, out of 217 persons, only 3 were attacked, or 1·3 per cent. The same result occurred in 1832. Then 53 prisoners died in the wards, and one only in the infirmary. (p. 47.)

Of the eight deaths in Hampstead, four took place in localities swarming with night soil and manure; and those persons generally suffered most in Marylebone who lived over stables and cowhouses. (See *Board of Health Reports.*)

I might prolong this part of my paper almost indefinitely, so numerous are the instances recorded; enough, however, has been said to prove the proposition, and this will be sufficient.

Dysentery. The facts collected in regard to dysentery in some epidemics point to a similar origin for the contagion. Most of the older writers have stated the same thing, and believed that the stools were contagious. "You often see," says Hildoanus Podoanus, "the disease attacking those who use a clyster-pipe previously employed by a dysenteric patient, and not well cleansed." Around a night-chair or a water-closet, the source of contagion may persist for a time, and infect all those who employ them. Fabricius Hildanus, in his work *De Dysenteriâ*, also believes in this infection from stools. (*British and Foreign Medical Review*, vol. xxiv, p. 357.)

This same fact is recorded by Zimmermann (*Ib.*, p. 139) as follows. After the battle of Dettingen, the dysentery made its appearance in the English army, and committed great ravages in it during the months of July and August. The hospital was in the village of Feckenheim, about a league from the camp. During the time that the army lay near Hanau, about 1,500 sick, besides those wounded on the field of battle, were brought from the camp into this hospital, and amongst these the greatest part were ill of the dysentery; the air was by this means infected to such a degree, that not only the rest of the patients, but even the apothecary, nurses, and the other servants, with most of the inhabitants of the village, were infected. (Zimmermann *on Dysentery*, p. 137.) Pringle says that this epidemic arose apparently from sudden changes of temperature and great exposure of the troops, and afterwards became contagious from development of typhus or hospital fever, but that, when the troops left the village, which had been converted into a hospital, and which was filled with the dirty straw covered with the dysenteric excretions, the disease ceased immediately to be propagated by contagion.

"Now," says Zimmermann, "the contagious power of the dysentery lies chiefly in the excrements; for the mere smell of them has often communicated the dysentery to men in perfect health and even beasts." And further on, he adds: "And even though one stops one's nose, one is not secure from infection, for the putrid vapours adhere to one's clothes, and when they are in high degree contagious, are thus communicated from one person to another; while at the same time he that has the clothes on his back is perhaps not in the least infected." (Zimmermann *on Dysentery*, p. 19.)

Himertus also admits this source of contagion. Vignes remarks: "We have seen many sick contract the disease in going to the common places where the dysenterics render their stool. (p. 205.)" And Chomel holds the same opinion. (*British and Foreign Medical Review*, vol. xxiv, p. 357.)

The case mentioned by Lind in his first paper on Infection, where a patient with chronic flux is said to have infected all those who used the same necessary with him, is believed by the author of the review above quoted, to be apocryphal; but to my mind it is quite within the pale of belief. (*Ibid.*)

The following is another instance of the special effects of stools in continuing the disease.

A very severe epidemic of dysentery broke out at Morrovelho, among the miners of a large gold mine in that locality. All the patients admitted into the hospital for other complaints, wounds, catarrhs, etc., were seized soon after their admission with it, and many died. My informant (Mr. T. P. Champion), a visitor in the hospital, always walked through the wards every day, and, observing that the stench from the patients' evacuations left in open pans under their beds was very great, he mentioned it to Captain Cotesworth of the Royal Navy, at that time chief-

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commissioner of the Catabranca mine, another mine in the neighbourhood. This gentleman then stated to him that no doubt the disease was aggravated by this custom. He instanced as proof the case of H.M.S. *Dublin*, in which the same complaint being rife in the ship, the men attacked were, because of the supposed contagion of the stools, kept apart and quite separate from the other men, and also prevented from going to the same closet. My informant mentioned this fact to Mr. Crickett, the chief commissioner at Morrovelho. After speaking to the surgeon, orders were given to remove these pans, to purify the closets, and to keep the wards sweet. The good effects were soon apparent in the marked diminution of the cases of dysentery, and finally in the speedy disappearance of the disease.

Dr. James Bird has informed me that the same fact was observed in the cases in hospital during the first Punjaub expedition; the emanations from the stools reproducing the dysentery in other cases, but, what is more singular, developing the hospital gangrene among the patients labouring under wounds to a fearful degree. During this period, the air was hot and moist. The mortality from disease in this expedition was very large, as compared with that of the second Punjaub expedition, during which period the weather was cold and dry, and the health of the troops good. This difference proves the danger and the influence of moist heat in spreading disease.

Typhus and Typhoid Fever. In the general remarks which preceded this paper, I already instanced the case of Christchurch Workhouse, Spitalfields, where typhus and typhoid fever were generated from this cause. I may quote here Mr. Grainger's evidence on this point. "A large body of evidence which I have received from medical practitioners both in London and other populous towns, as Manchester, Liverpool, and Nottingham, distinctly proves that persons habitually exposed to such an atmosphere [that of privies and cesspools] are thereby predisposed in an especial degree to fever and other sickness; and that, in courts and alleys, those persons who reside in the houses immediately adjoining foul privies, all other circumstances as to food, lodging, etc., being equal, suffer more from typhus than the other inhabitants. So much is this the case, that houses so situated have been pointed out to me by the medical officer as being the constant seat of fever, families after families coming to reside in them, and all in succession being attacked." (*Report on Cholera*, p. 87.)

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My own experience among the poor proves the same fact; added to which, in many instances, with the removal of the nuisance, so the disease has disappeared. An interesting and forcible instance was related by Dr. Thudichum, at a former meeting of this Society. Munich is remarkable for typhus; indeed, it is almost impossible for a family to live in it without some members catching the disease. Pettenkofer offers the following explanation. Two-thirds of the town possess no drainage, and the fæcal accumulations of the inmates, gathered in pails, are emptied occasionally in large holes in the adjoining yards, and left to accumulate and putrefy therein. Hence not only is the air polluted, but the ground in many places is saturated with liquid fæcal matter, whenever sufficiently porous to allow of its transudation. In these localities, therefore, typhoid disease is very rife; and, during cholera epidemics, the cholera has proved very fatal. This example is open, however, to the objection, that the disease may, from the very saturation of the ground, have been also produced by the impurity of the water drunk there when contaminated with choleraic matters in the wells; and that, as such, it is not a perfectly pure instance of typhus communicated by fæcal emanations.

Still, researches are not wanting to prove that emanations from cesspools have produced typhus. The cause of the disease is thus stated in Dr. Arnott and Kay's Report:—"The disease was most severe in those courts and alleys where there was no circulation of air; such, for instance, as Johnson's Change, in Rosemary Lane, in which there are about twenty houses, in almost every one of which fever prevailed. The disease first made its appearance there in the month of August last, and, on my first visit, I found the intolerable nuisance of the overflowing of a cesspool or privy, which continued for some time, there being no sewer to carry off the soil." (Arnott and Kay's *Report of the Sanitary State of the Labouring Classes.*) A case of typhus, produced from this cause, was mentioned to me by a medical friend. Being called upon to visit a patient affected with pleurisy, he was very much affected on entering the room by a most offensive cesspool odour, which entered the room by an open window, from a back yard in which the cesspool was exposed. The patient was removed to the hospital; but the same evening my friend became seriously affected with typhus, with black tongue, shivering, etc., etc., under which disease he was long ill. The

preceding good health, the instant nausea and uneasiness produced on entering the patient's room, and the rapid supervention in six hours or so of typhus, leave no doubt but that the cesspool odour was the exciting cause.

But upon this point it is needless to enlarge. Foul drains are notorious as at least being very frequently concurrent causes of typhus; but their effect in rendering suddenly malignant many of the exanthemata and other diseases, as scarlatina, erysipelas, measles, childbed fever, etc., when those labouring under these diseases are exposed to them, is so universally acknowledged, that any further remarks to prove this influence by a narration of facts becomes unnecessary.

Plague. That emanations from putrid animal matter will produce plague, is shown by the following extracts from Pariset. "In the winter 1823-4, the Pasha built a cotton factory at Kelioub, a small town four leagues north of Cairo. The foundations of this factory were laid among ancient and recent tombs. One day, about noon, a mason complained of headache. He was sent home, and at four o'clock he was dead. He was not visited, but eight persons who composed his family died the same evening, with both buboes and carbuncles. Kelioub was soon affected, and, of 5,000 inhabitants, lost 2,000. . . . Lately, a Copt rebuilt his house, and, on arriving at the burial-place, the workmen, to the number of four, took the plague." (Pariset, p. 938-9; *British and Foreign Medical Review*, vol. xxiv, p. 249.) That fæcal matter may retain the plague poison, is but a natural conclusion, and one much strengthened by a fact adduced by Mercurialis. Particular facts, however, in direct illustration of the position, are wanting.

Yellow Fever is the last division to which I shall refer in this paper. In the report of the Bermuda fever of 1850, we find the following statement, from the evidence of Captain R. W. Lacy. It is very analogous to the origin of cholera, before spoken of, at Brest. After speaking of the remarkable humidity of Bermuda, and in relation to the casemate barracks at Ireland Island, he adds: "On the north-west, and adjacent to the barracks, is a privy for the accommodation of the non-commissioned officers and men, the ineffective drainage of which, arising from its faulty construction, of its being beyond the influence of the tides, cause the soil, after ascending a large shaft, to accumulate at the foot of walls of the fortifications, emitting gaseous exhalations of the most pernicious and offensive nature, which taint the

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atmosphere of the barrack. To the north-east is a building of a similar description, for the officers, almost equally objectionable." (p. 90.) Upon this point, Mr. Anderson states: "Regarding the locality occupied by the troops at Ireland Island, I consider that the barrack privy, which accommodates a garrison of about three hundred men, must exercise a bad influence upon its sanitary condition. A hollow shaft, about fifty feet high, and averaging about twenty-four feet by four, or ninety-six superficial feet of section, conducts the soil from the top of the rampart to an open ditch leading to the sea. The shaft is always filled with the most noisome gases, continually escaping; and the ditch with the decomposing soil. The whole surrounding atmosphere is affected, and that not only as regards the barracks, but as regards part of the naval stores." (p. 77.) The same fact is alluded to in the Minutes of the Sanitary Commission, consisting of the naval storekeeper, deputy inspector of hospitals, and clerk of the works. (p. 74.) In confirmation of this point, it may be said, that the mortality was 36·8 out of 505 cases in Ireland Island, as a ratio to 1000 of strength. The measure of this mortality is not, however, so great as that which would refer much more to the influence of bad water suspended; since, in St. George's and Hamilton, the proportion to 1000 strength was respectively, 831 cases to 184·2 deaths, and 436 cases to 39·4 deaths.

PART IV.

RULES OF TREATMENT TO BE ADOPTED.

If the views which I have enunciated are correct, and the development of the diseases mentioned, whether communicated by fæcal matter in substance, in solution in water, or through a moist atmosphere, is due to a process of fermentation which generates the poison, it is clear that the remedies to be adopted are those which arrest fermentation, or, at any rate, those which, if they do not exactly arrest it, either absorb or modify the character of the poisonous sporules or gases formed during the process of fermentation, and so impede their action. We have, moreover, to consider the treatment in two points of view.

First, in its *curative* effect, as relating to an individual already affected.

Secondly, in its *prophylatic* effect, as relating to communities, to prevent their being affected. Both points of view have the closest relation, although they are best separately considered.

1. *Curative Effect of Treatment.* The remedies here applicable are those called antiseptics; and they consist of all those substances which chemically act on the ferments, because they have an affinity for them, and thus bring about an equilibrium in the fermenting body. "Among these we may enumerate alkalies, mineral acids, concentrated vegetable acids, volatile oils, alcohol, sea salt (in excess). The most effective is sulphurous acid, the metallic salts, especially those of mercury. Arsenious acid does not prevent the putrefaction of blood, nor the ordinary alcoholic fermentation of sugar, but entirely suppresses the putrefaction of the skin and gelatinous tissues." (Liebig's *Letters on Chemistry.)* Chlorine retards putrefaction, as before stated, by forming with dead animal matter a white pearly compound, which is almost imputrescible; phosphorus and deutoxide of hydrogen, by absorbing oxygen; creasote and ammonia also retard putrefaction, as before shewn. We may also get a practical lesson from the processes employed for the disinfection of manures, which at least prevent the generation of emanations from them, if they do not completely arrest fermentation. "The disinfection of manure has for its object to destroy the offensive odour, and to cause the phosphates to be retained in a form capable of being assimilated by the plants" (Liebig and Kopp's *Report*, p. 439, 1850); and a great variety of processes have been adopted. One of these has for its object the formation of charcoal, which may be procured largely in several ways. The slime and mud of the Seine in Paris, as also of the Thames, contains very much animal and vegetable matter intermixed with it, which, if burnt, constitutes a very efficient kind of charcoal for this purpose. This is Mr. Salmon's method. Mr. Alfred Samson found that the cinders of peat or turf, or the simple refuse of carbonised peat, had the same effect. The bran obtained from sawing wood, the refuse of oak which has been used for tanning leather, the mould on the Parisian strata, answer the same purpose; and, when fæcal matters are mixed with simple clay, it is only necessary to carbonise this admixture, and we obtain at once a perfect disinfecting powder. Hydrochloric acid

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is also said to act as a powerful disinfectant, by fixing the ammonia. The tribasic phosphate of soda has also been praised as a disinfectant. Calvond, in his *Journal de Pharmacie*, quoted by Liebig and Kopp *(Op. cit.* and *Journal de Pharmacie*, xvii, p. 281), finds that the methods usually employed do not fully satisfy any of the conditions required, but that the mother liquor of salt-works can be used with great advantage, and most perfectly, for disinfection.

It is impossible to look over this list of antiseptics and disinfectants without being struck with the fact, that among them are to be found most of those remedies which we have been in the habit of hearing so vaunted in practice for the cure of the diseases spoken of in this paper. I will instance a few of these: *sulphuric acid*, a mineral acid, found to be most useful in cholera; also *cajuput*, an essential oil, in the first epidemic in this country; *chalk*, an alkaline earth, in the premonitory and other diarrhœas; *saline injections, common salt*, and its internal administration in *large* quantities, as in the case of the late Mr. Carmichael of Dublin; *mercury*, according to Dr. Ayre's plan; and lastly, *charcoal*, which proved so invaluable a remedy among the choleraic patients in Canada in 1832. The disease was then at its height, when a stranger made his appearance in the colony, and administered charcoal largely; and the result was, that cures were very frequent, and the epidemic soon disappeared. The effect was so marked, that this stranger was believed by the ignorant to be a saint, having succeeded where the doctors failed. I have also been told that phosphorus proved in many cases, in the hands of a London practitioner, a most efficacious remedy. Dysentery is another disease in which antiseptic remedies have been given with success, such as sulphate of copper and alum, metallic salts; not to speak of small doses of calomel, another metallic salt, opium and carbonate of soda: the last an alkali. Charcoal has also been found most efficacious in dysenteries and diarrhœas, especially when complicated with intermittent fever. It is known in Smyrna to exert sometimes a curative effect in intermittent fevers, where quinine has failed; and this fact is confirmed by the experience of the learned Dr. Calvert, Calsagno, Macadino, Morsin of Nissoria, Burza of Palermo, Borland, and Tully. (See *Edinburgh Medical and Surgical Journal*, vol. x.) Perhaps, indeed, bark itself acts as an antiseptic. From some experiments made by Dr. M'Bride, it would appear that, if putrefaction has not proceeded to a very great

degree, it may be arrested by infusion of bark. Thus, Dr. M'Bride placed a putrid rat in a strong infusion of bark: it was completely deodorised thereby. If the putridity, however, was far advanced, the infusion failed. Certainly the change effected in many low fevers by bark is well explained on this view. Pariset believes that the reason that the plague does not produce a greater mortality in Egypt is, that the Nile water is very alkaline, and that spreading, in the annual inundations, over the country, it neutralises the miasmata. Most of the remedies I have mentioned have been employed in fever, and each would no doubt find some advocate in this Society. Charcoal is less often given in fever in this country than it might be. I am satisfied that, in my hands, it has been a means, under Providence, of doing much good in cases of fever, particularly when these have been accompanied with very offensive stools, especially in the remittent fever of infants.

It would be useless in me, however, to extend on this point. If the diseases in question be induced by fermentation, and especially in those cases where that fermentation is of fæcal origin, the remedies which common sense would indicate are those which arrest this action, namely, antiseptics and disinfectants. This, it appears to me, is the only philosophical treatment, and so far it is that which theory and practice alike support. Till, however, we know more of the peculiar chemical nature of each poison, we must, when once the broad principle is laid down, prosecute by empirical experiment. It may be, and it no doubt is so, that there is a specific antiseptic for every disease of the kind specified, and for many more. Sulphuric acid may be the best in cholera; mercury or wine in fever, as the case may be: furthermore, each may be susceptible of a similar explanation to that given for arsenic, which may be supposed to prove sometimes the best remedy for skin-disease, because it specifically arrests fermentation in the skin. But, at any rate, the remedies to be sought for are, theoretically, to be found among those which, by arresting fermentation in the system, prevent its extension.

While I state this, however, I must guard myself from an inference which may be made. I do not say that all fevers, or even the diseases here mentioned as fermentative, may not be cured by remedies which act otherwise than as antiseptics. This is quite possible, and I am bound to admit it. I merely suggest one mode in which many remedies may act.

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2. *Prophylactic Effect of Treatment.* The treatment must be *prophylactic*, as well as *curative*. In a sanitary point of view, the remedies used must not only aim to cure the local malady in the patient's body, but to prevent its diffusion through a town, or wherever a number of individuals are congregated together. Now, as means to this end, our attention has of late been directed to two agents, both of which have been recommended—*charcoal* and *fresh sea water*.

(a) Charcoal. I have before spoken of charcoal as an antiseptic, although, from Dr. Stenhouse's inquiries, it appears to be a deodoriser from an opposite property, by reason of the large quantity of oxygen contained in its pores, which it absorbs, together with the miasmata and bad smells of decaying substances. These last are rapidly burnt and destroyed; so that, in point of fact, charcoal is not purely an antiseptic, but a *destructive* of decaying matters. A series of very valuable papers have been contributed, on this subject, to the *Journal of Public Health*, which have fully brought out the advantages of this agent as a disinfectant and purifier. Dr. Stenhouse's plan of air-filters, applied over offensive gulley-holes, would go far towards removing the foul odours of our London streets; and a similar kind of apparatus, to cover the apertures of water-closets in private houses, but especially in hospitals, would very much increase the comforts of their residents. In like manner, if bodies were buried in charcoal, according to Dr. Richardson's suggestion, all odour from this source would be removed, and not conveyed by solution, after heavy rains, within the common sewers. But this will not suffice. The sewers themselves will continue to emit these odours. All gulley-holes cannot be closed by charcoal air-filters, and the mud and rain must pass downwards into them through some openings; and it is therefore necessary, if possible, that something should be devised to deodorise the common sewers themselves. The employment of hydrochloric acid, chloride of lime, and pure charcoal, would involve too much expense—the first two in particular. In a very able paper by Mr. Durden in the *Journal of Public Health* (vol. i, p. 213), on the Preparation of Charcoal, the preferable mode, because the cheapest, would seem to be by the action of sulphuric acid on sawdust. The chief objection, however, to this plan, is the great uncertainty of

procuring the *raw material*, the sawdust, in sufficient quantity—a difficulty on which many a city company, whose profits, as set forth in their prospectuses, were to have been extraordinarily great, has foundered. But I think that we need only follow out in London Mr. Salmon's plan, already adopted so successfully in Paris. Assuredly, old Father Thames contains animal and vegetable matter in greater abundance than the Seine in its slime and mud; and since, when this is burnt, it yields a charcoal in a very fine state of powder, and which possesses the disinfecting property to an extraordinary degree, we have at our doors a source whence charcoal can be obtained in abundance and of excellent quality, and whence it could be very cheaply collected and prepared.

If once obtained in sufficient quantity, nothing could be easier than its transmission, by suspension in water, into the common sewers, from certain high localities before selected and established. We should do what we are now doing when we flush the sewers, only we should deodorise at the same time, instead of forcing upwards through the pipes of water-closets, and all the gulley-holes in town, those pestilential odours which are so fearfully obnoxious, and which originate or aggravate disease.

I am not aware that any but one objection of weight has been made to this introduction of charcoal, and it is that in course of time the sewers would become clogged up, and in this manner the flow through them arrested. I believe that this supposed effect is greatly exaggerated. The fine mud and broken up fæcal matters have not usually this effect. And if the supply of water passed through the drains at the same time be great, so that the current in the sewers is increased, and the size of the sewers be sufficient, I think no such effect would follow. Indeed, if obstruction did occur, perhaps in the end it would prove of advantage, because it would compel builders and the parochial authorities to repair and widen the narrow and faulty sewers, and to build any new sewers required upon an improved and more scientific plan.

(b) Water. I now come to the next point, the supply of water required for the suspension and diffusion of this deodorising powder. Under this plan, impure water would offer no impediment. The most offensive and impure water loses all odour by admixture with charcoal. All that would be needed would be a large reservoir with a smooth bottom, from which the water, mixed gradually with charcoal and

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agitated by machinery (such, for instance, as a paddle wheel) could be emptied in quantity and at fixed intervals into the sewers. The foulest Thames water, or the Serpentine water of the parks, would suffice. I think this supply would be found sufficient; so that there would be no need to bring sea water into London according to Mr. Fuller's plan, which I believe is upon the whole very objectionable, at least so far as relates to the watering of the streets by it. From the facts before recorded on the subject of sea water, putrefaction and foul odours would be augmented, at least in the hot weather. I may add a few additional facts on this point. In a high temperature, at least so far as my experiments go (I speak of a temperature above 60°), animal matters will putrefy more readily and quickly in sea water than in fresh water. I have often noticed this result in vivaria. If a snail or a fish died in a *fresh water* vivarium, it might remain in the basin three or four days and longer, and the water did not sensibly suffer; the remaining animals escaped with impunity. In the *marine* vivarium, if a periwinkle or anemone died, in two or three days the water smelt strongly, and the remaining animals all died. This result I have frequently noticed in the same conservatory, where both vivaria were exposed to the same influences. I have also noticed a fact which, so far as *chloride of sodium* is concerned, seems to prove that a small quantity of this salt rather favours decomposition. I was preparing some of Liebig's beef tea: the temperature was tepid, and it was apparently fit to drink. In this state I thought to make it more palatable by the addition of a small quantity of salt. I added this, and the result was that it suddenly frothed up, and became very offensive; putrefaction had begun. Matters to be kept must be highly salted, and the temperature must be low, otherwise they will putrefy as readily, if not more so, than if not salted. In the instance of the Bermuda fever, to which I alluded before in my last paper, I instanced the case of the mud in which the convict hulk the "Thames" was fixed; although the water over it during the tidal rise was changed twice daily, the odour was very powerful from decomposing animal, especially fæcal, and vegetable matters. Sea weed will readily putrefy under a hot sun, though saturated with sea water. I therefore cannot help thinking that the beneficial effects of sea water have been overrated. Still I think that, if undiluted with much water, it does not in itself favour the extension of some epidemic influences. As a rule,

watering places, and particularly those where there is no fresh water river at hand, are healthiest; and except where cases are continually imported, or where bad water is drunk, epidemics, such as cholera, for instance, are not propagated. The coexistence, however, of a river, by the admixture of the waters of which the sea is unduly diluted, more especially if offensive sewage forms any part of this admixture, has quite an opposite effect. In such localities, some epidemics when present rapidly extend, and are very fatal. I see, then, no good result likely to be obtained by the substitution of *sea* for *fresh* water for the purpose of watering the streets.

I have said that the most impure fresh water would become purified and deodorised by the charcoal admixture. But the adoption of this plan should not be limited to the common sewers. There are *two* sources of infection *without* the sewers to which the deodorising measure should be applied. 1. The ordinary water-closets in domiciles should from time to time have charcoal thrown down into them; and whatever is inconsistent with its transmission, such as syphons or old fashioned traps, removed. If this were done, the source of annoyance in our houses from these odours would be removed. 2. We all know to our cost how offensive are mews, particularly in summer; and as before stated, during the late epidemic of cholera, it was the inhabitants of these localities who suffered most. If, in these, alternate layers of charcoal and stable refuse were interposed, the odour would in great measure disappear. I am aware, however, that John Bull could only be compelled to do this by an act of Parliament.

In any general plan, however, which should be adopted, it is expedient that the sewage matter now deodorised should be diverted from the Thames and carried out of town. But I think it is one of the advantages accruing from the employment of charcoal, that, once deodorised, there is no need of conveying it to such great distances from London. A distant removal has its advantages as affording larger space for operations, but then the tunnelling of the main sewers to such a locality is peculiarly expensive. If the deodorising process be effected in the sewers, a Montfaucon in London, even as near as that in Paris, would cease to be so great a nuisance; and the air being no longer tainted by vile odours, and not capable of being wafted back on the town, all disadvantages of proximity would cease. How near this proximity should be is

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then, after all, resolved into a matter of convenience to the public.

To *agriculturists*, the advantage of such a ready made manure at the very doors of every large town would be infinite. Human manure is inferior to none, and equal to guano, and could be procured at a price considerably less. To the *inhabitants of towns*, a comparatively pure atmosphere, a less frequent recurrence of pestilence, and a more benignant type of disease would, under God's blessing, be the well earned reward.

RICHARDS, PRINTER, 37, GREAT QUEEN STREET.

ON THE

WEIGHT AND DIMENSIONS OF THE HEART

IN

HEALTH AND DISEASE.

By THOMAS B. PEACOCK, M.D.,
ASSISTANT PHYSICIAN TO ST THOMAS'S HOSPITAL,
AND PHYSICIAN TO THE CITY OF LONDON HOSPITAL FOR DISEASES OF THE CHEST.

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WEIGHT AND DIMENSIONS OF THE HEART

IN

HEALTH AND DISEASE.

The following paper contains a series of observations with calculations based upon them, intended to illustrate the weight and dimensions of the heart in health, and the changes which it undergoes in different forms of disease.

The observations have all been collected by myself within the last few years; some have already been published in connection with a series of tables of the weight of the brain,[1] but most of them have not previously appeared in print, and all the weights of the diseased heart and the measurements, are now first collected and analyzed. For the purpose of presenting the facts in a more complete form, and in order to furnish the means of ready comparison between the results here arrived at, and the data from which they are deduced, I have preferred to give the whole of the observations together, rather than to refer to the former papers for those previously published.

The mode in which the observations were made is as follows:—The heart having been removed from the body, and the aorta and pulmonary artery cut across about an inch above their origin, the left ventricle was laid open by an incision commencing near the aortic orifice, and continued across the apex to the posterior wall, so as fully to expose the cavity, without interfering with the attachments of the valves. The right ventricle was exposed by a similar incision extending from near the origin of the pulmonary artery to the apex, and the auricles were laid open transversely. The organ was then deprived of coagulum, washed, wiped, and weighed.

The measurement of the girth of the heart externally was taken with a piece of tape or string, from the line of the septum in front, to the corresponding situation behind. The capacity of the orifices

[1] Monthly Journal of Medical Science, vol. vii. (New Series, vol. i.) 1846.

was ascertained by the passage of graduated balls numbered from 1 to 15, and measuring from 21 to 63 French lines—each number being 3 French lines more in circumference than the former. The capacity of the orifices having been ascertained, and the state

WEIGHT AND DIMENSIONS OF HEALTHY HEARTS.

No.	Age.	Sex.	Weight of Body.	Weight of Heart.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of cavity of Right V.	Length of cavity of Left V.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.			Thickness of Septum.	Circumference of Apertures.				Cause of Death.
									Base.	Mid-point.	Apex.	Base.	Mid-point.	Apex.		Tricuspid	Pulmonic.	Mitral.	Aortic.	
			lb	oz. dr.	lines.	lines.	lines.	lines.							lines.	lines.	lines.	lines.	lines.	
1	7 days	F.	7	0 13	25	19			2 lines			2 lines				13	13	13	13	
2	10 months	M.		1 10	29	25			1 line							22	19	22	19	Acute hydrocephalus and pneumonia.
3	1 year	F.	15	2 0	34	32	22	19	2	2	1½	3	3	1½		28	22	25	17½	Bronchial phthisis.
4	3 years	F.	22	1 10	35	35	21	21	1 line			3	3	2	4	28	22	25	17½	Bronchial phthisis.
5	3	M.		2 15	35	35			1 line			3½	4	1½	3	26	21	25	18	Burn, 4 hours.
6	4½	M.		2 9	36	30	25	22	1½	1½	1	3	4	2		31	24	28	22	Diseased kidneys and dropsy; empyema.
7	8	F.	56	5 1	42	36	36	30	1	2	1	3	4	1½		36	25	30	20	Phthisis, 4 valves to pulmonary orifice.
8	8	F.	24	3 0	36	30	27	24	1	1½	1	3	4	2		33	30	33	27	Burn of abdomen.
9	11	M.	43	4 6	38	37	34	28	2	2	1	3½	3½	2		36	27	30	24	Abscess of liver; diseased mesenteric glands.
10	13	F.	46½	6 2					1½	2	1½	5	5	2		39	33	36	24	Fractured skull.
11	18	F.		7 15	52	48	34	31	1½	2	1	4½	5	1½		42	36	42	30	Fever; enlarged spleen.
12	20	M.	97	11 0	49	48	48	39	2	2	1½	7	7	3	6	48	42	45	30	Phthisis and morbus renum.
13	24	M.		8 8	50	46	40	34	1½	1½	1½	4	7	2	6	43	29	37	26	Phthisis.
14	24	M.		9 1½	60	60	34	32	2	2	1½	5	6	2½	6	45	39	42	36	Meningitis.
15	24	M.		9 0	60	54	42	40	2	1½	1	5	6	3	5	60	42	48	39	Ascites and diseased liver.
16	25	M.		8 8	60	38	35	32	2	2	1	6	5	2	6	60	34	42	31	Fever; foramen ovale not entirely closed.
17	26	M.	101	8 11	54	48	45	36	1½ line			4	6	2	6	54	45	51	42	Disd. kidneys and bladder.
18	27	M.	120	11 12	52	52	45	42	2	2½	1½	5	6	3		60	42	48	36	Ulceration of intestines.
19	30	M.		8 15½			40	36	1½	2	1	5	7	2	7	50	32	38	28	Fever, cirrhosis hepatis.
20	31	M.		9 4	48	53	46	37	2	2	2	5½	3½	2	3½	51	45	48	36	Abscess of brain.
21	37	M.		6 4½	50	42	36	36	2	2	1½	6	7	2½	6½	54	43	51	42	Phthisis.
22	66	M.		10 8	60	46	45	37	2	2	1	6	5	2½	5	51	39	48	36	Phthisis.
23	20	F.		7 4	49	38	40	32	2	2	1	5	7	2½	6	51	39	39	36	Fever.
24	24	F.		10 11	60	48	48	42	1½	3	1½	6	8	2	4	42	33	37	29	Variola.
25	47	F.		10 3			42	36	3½	2½	2	3½	5	2		60	45	48	36	Ac. capillary bronchitis.
26	60	F.	87	11 0	52	40	42	36	2	2	1	7	5	2½		54	39	45	30	Ac. asthenic bronchitis supervening on chronic.
27	66	F.	70	7 0	48	42	42	27	1½ line			4	7	2		40	34	42	30	Bronchitis.
												6	7	2		51	42	48	33	Phthisis.

of the valves observed, the orifices were laid open, and the length of the ventricular cavities measured from the attachments of the aortic and pulmonic valves to the apex, and the thickness of the parieties ascertained at three points; near the base, where the walls begin to narrow; at the mid-point between the base and apex; and at the

WEIGHT OF HEALTHY HEARTS—MALES.

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death.
		lb.	oz. dr.	
28	6 mths.		0 13½	
29	2 „	24	2 10	Burn, 21 days.
30	2 yrs.	31½	2 12	Burn, 6 hours.
31	4		4 4	Burn, 12 hours.
32	5	21	3 0	Coma, succeeding cholera.
33	8	48	6 4	Scarlatina.
34	10	45	4 4	Cholera.
35	11	42	8 8	Typhoid fever.
36	11		5 2	Typhoid fever.
37	11	38	4 0	Chorea.
38	11		6 0	Phthisis.
39	14		9 0	Amputation of toe; morbus renum.
40	14		6 8	Typhoid fever.
41	15	88	8 0	Laceration of internal organs from injury.
42	15		6 0	Phthisis; copious hæmoptysis.
43	16	47	5 8	Phthisis.
44	16		7 0	Bronchial phthisis.
45	16		6 8	Typhoid fever.
46	17		8 8	Typhoid fever.
47	19		11 0	Typhoid fever.
48	20	100	10 0	Diphtheritis.
49	21		8 8	Strumous pyelitis.
50	21	106	9 8	Phthisis.
51	22	127	8 0	Phthisis.
52	24		8 0	Phthisis.
53	24		10 0	Fever.
54	24	90	7 8	Phthisis.
55	24		10 4	Phthisis.
56	25	103	10 0	Phthisis.
57	25	125	10 8	Fever; white spot on pericardium.
58	25		9 0	Fever.
59	26		8 12	Lumbar abscess.
60	27	145	9 0	Phthisis.
61	27		11 4	Variola.
62	28	108	8 8	Fever.
63	28	97	9 8	Phthisis; softening of brain.
64	28	103	10 0	Fever.
65	28	97	7 8	Morbus renum.
66	29		11 0	Morbus renum.
67	29		11 0	Phthisis; white spot on pericardium.
68	32		11 8	Double pneumonia.
69	32		11 8	Morbus renum.
70	33		10 8	Ulceration of cartilages of knee.

apex. The septum was cut across about half way between the base and apex, and measured across its centre.

No organs were weighed in which there was an undue proportion of fat on the surface, and none were measured which, whether from disease or post-mortem change, were very flaccid. The weight em-

WEIGHTS OF HEALTHY HEARTS—MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death.
	Years.	lbs.	oz. dr.	
71	33		9 0	Diseased liver.
72	33	99	8 0	Phthisis; aorta atheromatous.
73	33		11 0	Apoplexy; aorta atheromatous.
74	35		8 0	Morbus renum.
75	35	100	7 8	Phthisis.
76	35	87	7 4	Morbus renum.
77	35		6 12	Phthisis.
78	35		11 8	Laceration of aorta, from violent muscular exertion.
79	37	112	8 0	Fever, white spots on pericardium.
80	37	90	9 10	Abscess on buttock and secondary deposits.
81	37		9 0	Pneumonia.
82	37	84	9 4	Phthisis.
83	37		9 0	Fever.
84	38		8 8	Fever.
85	38		10 0	Meningitis; pneumonia.
86	38	100	10 12	Fever, chr. bronchitis.
87	38		8 8	Erysipelas of head.
88	38		11 0	Delirium tremens; softening of brain.
89	39		6 0	Carcinoma pylori.
90	39		11 0	Phthisis.
91	39		9 12	Phthisis.
92	40		11 0	Phthisis, two valves to aortic orifice.
93	40		11 12	Fever.
94	40		11 8	Cirrhosis hepatis.
95	40	130	11 8	Delirium tremens.
96	40		10 4	Phthisis.
97	41		9 8	Phthisis.
98	42	115	11 12	Pneumonia.
99	42	114	8 8	Phthisis.
100	45	79	9 8	Phthisis.
101	45		10 0	Phthisis.
102	46	90	10 0	Injury; purulent deposits; white spot on pericardium.
103	47	129	10 0	Phthisis.
104	50		9 0	Fever.
105	50		9 0	Fever.
106	51		9 12	Pleuropneumonia.
107	52		10 8	Bronchitis; white spots on pericardium.
108	53	115	10 8	Complicated dislocation of astragalus; white spot on pericardium.
109	53	103	5 0	Cirrhosis hepatis and morbus renum.
110	54	146	11 8	Meningitis and apoplexy.

ployed is avoirdupois, and the measure French lines and inches—the French line being 0,888 of an English inch, and the French inch equal to 1.065 English inch, or rather more than $\frac{1}{8}$ in. longer. The French line is equal to 2.25 millimetres. The French measures were made use of as having been those employed by M. Bizot and some other observers.

WEIGHTS OF HEALTHY HEARTS—MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death
	Years.	lb.	oz. dr.	
111	54		9 0	Pleuritis; peritonitis.
112	55	80	3 8	Tertiary symptoms.
113	55		8 0	Phthisis.
114	56		11 8	Cut throat.
115	56	84	7 0	Phthisis.
116	60	102	11 8	Disease of stomach.
117	60	101	10 0	Fractured ribs; pneumonia.
118	60	129	11 0	Phthisis; white spot on pericardium.
119	60		11 8	Phthisis.
120	60		11 0	Chronic dysentery.
121	62	99	10 8	Phthisis.
122	70		11 8	Fever; white spot on pericardium.

WEIGHT OF HEALTHY HEARTS—FEMALES.

123	10 wks.		1 3	
124	1 year and $\frac{3}{4}$th	13½	1 8	Subacute hydrocephalus.
125	5 years	28	2 14	Cholera.
126	6		2 12	Burn, 14 hours.
127	7	32	2 3	Fever.
128	7		3 0	Fever.
129	11	36	3 14	Cholera.
130	17	59	4 8	Phthisis, necrosis.
131	17		7 0	Phthisis.
132	18	113	9 8	Fever.
133	18		10 8	Bronchitis; phthisis.
134	19		7 12	Typhoid fever.
135	19		8 0	Cholera.
136	20	90	9 0	Phthisis.
137	21	86	5 12	Phthisis.
138	21		10 8	Morbus renum.
139	21	97	10 0	Phthisis.
140	22		10 4	Fever.
141	23	85	6 12	Phthisis.
142	23		8 8	Disease of ear and brain.
143	24		11 0	Acute capillary bronchitis.
144	25	86	9 12	Pneumonia.
145	25		5 8	Phthisis.
146	25	66	8 0	Tertiary syphilis.
147	25	117	9 0	Variola.

The tables include 183 observations of the weights of organs regarded as healthy, and 145 observations of the weights of hearts presenting either primary or secondary disease. The measurements are of 27 healthy and 41 diseased organs. The whole series is a careful selection from a larger number of observations, of which those only are given which are regarded as accurate and complete.

WEIGHT OF HEALTHY HEARTS—FEMALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death.
	Years.	lb.	oz. dr.	
148	26		9 0	Acute phthisis.
149	28	97	9 8	Fever.
150	28		7 4	Morbus renum.
151	28		7 12	Phthisis.
152	28		7 4	Morbus renum.
153	29		9 8	Disease of liver.
154	30		8 8	Pneumonia; morbus renum.
155	30		10 0	Cholera.
156	31	104½	9 0	Delirium tremens.
157	31	89	9 12	Fever.
158	32	107	9 12	
159	33	94	8 8	Fever.
160	34	88	7 8	Phthisis.
161	34	103	11 0	Phthisis.
162	34	71	6 8	Morbus renum.
163	35	58	5 8	Phthisis.
164	36		8 8	Ac. supervening or chronic bronchitis.
165	36		10 12	Fever.
166	38	79	11 0	Phthisis.
167	39		8 8	Scrofulous abscess of liver.
168	39		10 0	Pleuritis; morbus renum.
169	39	73	7 7	Phthisis.
170	39	95	9 0	Phthisis; puerperal mania.
171	39		9 8	Morbus renum.
172	40		9 0	Sudden death during convalescence from fever.
173	40		9 4	Pneumonia; gangrene of os uteri after fever.
174	40	75	8 0	Relapsing fever.
175	45	109¾	11 0	Phthisis.
176	45	91	8 0	Tubercular peritonitis.
177	47	109	9 12	Morbus renum.
178	50		7 0	Phthisis.
179	51		8 8	Phthisis; white spot on pericardium.
180	52	74	8 0	Fever.
181	53	101	10 12	Phthisis.
182	56		10 8	Fever.
183	58	66	8 0	Gangrene of leg from obliteration of aorta.

WEIGHTS AND MEASUREMENTS OF DISEASED HEARTS.

No.	Age.	Sex.	Weight of Body.	Weight of Heart.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle.	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.			Thickness of Septum.	Circumference of Apertures.				Cause of Death, and Form of Disease.
									Base.	Mid Pt.	Apex.	Base.	Mid Pt.	Apex.		Tricuspid.	Pulmonic.	Mitral.	Aortic.	
	Yrs.		lb.	oz. dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	
1	25	F.		9 0	54	40	44	32	4	4	2	4	4	2		51	39	45	39	Chronic bronchitis, with deformed spine.
2	5	F.		3 12						to		4	4	2		39		33	25	Abnormal septum in right ventricle.
3	15	M.		10 0	54	42			2	to	5½	3½	to	4½		39	13	36	33	Abnormal septum in right ventricle; aorta arising from both ventricles; pulmonary or. contracted.
4	20	M.		12 0	66	42			4	7	3	6	5	3		45	12	36	30	Great contraction of pulmonary orifice, with open foramen ovale.
5	8½	F.		6 3	54	42			4	3	1	5	6	2		42	36	39	19	Open foramen ovale; thickening of valves.
6	45	M.	149	17 3	56	58	47	52	2	2	2	6	9	3	11	58	43	56	39	Double pneumonia; no valvular disease.
7	33	M.		12 3	60	54	42	40	1½	1½	1½	6	6	1½		60	48	51	39	Pleuropneumonia; some tendency to excavation at apex of left ventricle.
8	35	M.	78	10 12	54	48	42	36	3	3	2	6	8	3		60	42	48	39	Diseased hip; malformation by defect of aortic valves, with thickening of valves.
9	44	M.	127	12 12	54	50	48	40	2	1½	1	5	7	2	7	42	35	39	34	Fever.
10	40	M.		12 3	60	50	48	41	2½	2	1	5½	6	3	6½	54	48	54	42	Fever and bronchitis; slight thickening of valves.
11	50	M.		14 0	60	56	48	40	2	2	2	4	7	3	5	60	48	51	42	Erysipelas of head; slight thickening of aortic and mitral valves.
12	65	M.		12 3			36	36	3	2½	1	7	7	2½	6	54	48	48	39	Intussusception; valves somewhat thickened, and aorta dilated.
13	65	M.	137	12 0	48	39	51	40	2	2	1	6	8	2	6	57	42	45	39	Fever, with icterus and hæmorrhage from bowels; no material valvular disease.
14	65	M.		40 12	96	86	62	51	3	2½	2	8	11	3½	10	63	54	60	42	Sudden death; slight atheromatous degeneration of aortic and mitral valves, and of aorta.
15	71	M.		21 0	72	66	48	42	2	3	1½	10	11	2½	8	60	42	51	36	Bronchitis; thickening of aortic and mitral valves; dilatation of aorta.
16	72	M.		14 0	76	54	51	36	3	4½	1½	6	6	2½	4	60	45	54	36	Chronic bronchitis; atheromatous disease of aorta and pulmonary artery; thickening of valves.
17	78	M.		18 0	54	66	42	36	3	5	2	6	9	4		60	45	54	39	Scalp wound; thickening of aortic and mitral valves; great dilatation of aorta.
18	60	F.		14 0	60	60	48	42	2	2	2	5	7	2½		60	39	45	33	Some thickening of aortic valves; disease and dilatation of aorta; fatty degeneration.
19	62	M.		14 3				36	2	3	1½	8	9	4		57	42	48	33	Contraction of aortic orifice, and thickening of the valves; rupture of septum ventriculorum.
20	62	M.		16 0			42	36		2½		6½	7	3½		54	42	48	36	Mass of bone projecting into the aortic orifice; valves ossified; recent pericarditis.
21	24	M.		15 3	62	62	50	49	2	1½	½	7½	7½	1½		57	45	51	42	Apoplexy; aortic valves much thickened and decurtated; atheromatous dis. of mitral valves and of aorta.
22	75	F.		10 12	48	45	40	38	2	2	2	8	8	3						Extreme contraction of aortic orifice from malformation and ossification of valves; death from strangulated hernia.
23	33	M.		17 12	54	62	60	48	2	3	2	6	10	3		60	42	54	30	Injury of aortic valves from violent exertion.
24	32	M.		16 0			56	42	2	2¼	¾	4	7	1½		60	48	60	42	Incompetency of aortic valves, with recent endocarditis and fibro-cartilaginous degeneration of muscular structure.
25	42	M.		16 0	53	63	50	44	1¾	2½	1	6½	5½	2		60	45	51	36	Extensive disease of aortic valves; obstruction and incompetency; aortic disease.

WEIGHTS AND MEASUREMENTS

No.	Age.	Sex.	Weight of Body.	Weight of Heart.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle.	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.		
									Base.	Mid Pt.	Apex.	Base.	Mid Pt.	Apex.
	Yrs.		lb.	oz. dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
26	50	M.		23 0	66	60	66	50	$2\frac{1}{2}$	$3\frac{1}{2}$	$2\frac{1}{2}$	7	7	3
27	55	M.		34 0	90	81	72	60	$2\frac{1}{2}$	5	2	10	$5\frac{1}{2}$	1
28	56	M.		15 4	58	55	48	36		$3\frac{1}{2}$		6	5	$3\frac{1}{2}$
29	57	M.		11 8	44	60	38	33	2	3	1	7	7	$2\frac{1}{2}$
30	46	F.	91	16 0			52	52	2	2	$1\frac{1}{2}$	6	7	3
31	27	F.		8 8	60	48	42	36	2	2	1	4	5	2
32	23	F.		10 8	59	47	44	39	3	2	1	5	5	2
33	39	F.		15 12	75	52	51	42	2	3	$1\frac{1}{2}$	5	5	2
34	40	F.		12 8	66	50	48	42	2	2	1	6	7	3
35	68	F.		12 0	54	48	45	36	2	2	2	5	5	2
36	8	F.		10 8	60	48	36	36	2	2	$1\frac{1}{2}$	5	5	3
37	11	F.		7 8	41	45	33	33	$1\frac{1}{2}$	2	$1\frac{1}{2}$	5	$6\frac{1}{2}$	$2\frac{1}{2}$
38	12	F.		12 0	54	60	36	42	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	5	5	$2\frac{1}{2}$
39	18	F.		13 8	72	54			2	2	1	6	6	3
40	39	F.		22 0						4		9	11	3
41	63	F.		22 0	55	55	43	42	2	2	$1\frac{1}{2}$	7	10	2
42	37	F		9 0					$1\frac{1}{2}$	$1\frac{1}{2}$	$1\frac{1}{2}$	4	4	4

OF DISEASED HEARTS—*continued.*

Thickness of Septum.	Circumference of Apertures.				Cause of Death, and Form of Disease.
	Tricuspid.	Pulmonic.	Mitral.	Aortic.	
lines.	lines.	lines.	lines.	lines.	
	60	48	54	45	Retroversion and perforation of aortic valves; some disease of mitral and of aorta.
	60	51	60	45	Incompetency from adhesion of valves and dilatation of orifice; aortic disease.
	54	45	54	39	Incompetency; recent endocarditis.
9	48	39	42	36	Softening of brain and apoplexy; old pericarditis; thickening and incompetency of aortic valves; dilatation of aorta.
		37		29	Thickening and decurtation, and incompetency of aortic valves; aorta dilated and diseased.
4	57	39	39	30	Peritonitis, from passage of gall-stones. Pericardium entirely adherent from old attachments; mitral valve thickened and rigid.
6		29	22	25	Great contraction of aperture, and induration and thickening of mitral valve.
5	54	39		33	Sudden death. Very great contraction of mitral aperture, and induration and thickening of valves.
5	40	30	24	27	Fever. Great contraction of mitral aperture, and some thickening of aortic valves.
	54	36		30	Great contraction of mitral aperture, and induration and thickening of valves.
	36	33	45	30	Free regurgitation through mitral aperture, from dilatation of the orifice; valves slightly opaque and thick. L. aur.; $1\frac{1}{2}$ to 2 l. thick.
5	42	30	36	30	Combined aortic and mitral disease, chiefly mitral; sequence of rheumatism of two years' duration.
	48	33	36	30	Great aortic and mitral disease; sequence of rheumatic fever 9 years before.
5	42	36	42	27	Thickening and contraction of mitral, with some of aortic aperture and valves; old pericarditis; sequence of rheumatism 15 months before.
	51	39		30	Very great contraction of mitral aperture, with thickening of the aortic valves; aorta atheromatous.
	51	32	43	35	Contraction of mitral aperture; crescentic thickening of aortic valves; tricuspid valve thickened; chronic bronchitis.
	21	34	18	30	Very great contraction of tricuspid and mitral valves; slight thickening of aortic valves.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
43	14		16 12	Dilatation and hypertrophy of walls of left ventricle, without valvular disease; recent lymph on pericardium.
44	18		23 0	Destruction of aortic valves occasioning incompetency; malformation.
45	18		12 8	Pericarditis; purulent deposits after amputation.
46	22		12 0	Compound fracture.
47	25		12 0	Laceration of walls of heart from injury.
48	22		12 0	Phthisis; some thickening of tricuspid valve; hypertrophy and dilatation of right ventricle.
49	24		14 8	Combined mitral and aortic valvular disease.
50	23		13 0	Pneumonia; hypertrophy of left ventricle, without valvular disease.
51	21		24 0	Incompetency of aortic valves, with thickening of mitral valves.
52	25		12 0	Phthisis.
53	29		14 4	Left ventricle hypertrophied and dilated; aorta dilated.
54	26		15 0	Incompetency of aortic valves, with thickening and opacity of mitral.
55	26		13 0	Delirium tremens; some thickening and opacity of left valves.
56	27		15 0	Fever; chronic bronchitis of left valves and emphysema.
57	29		12 8	Phthisis; no valvular disease.
58	29		14 0	Cholera; great contraction, thickening, and induration of mitral valve.
59	38		21 0	Aneurism of septum of left ventricle, with hypertrophy and dilatation of both ventricles.
60	38		12 0	Phthisis and pneumonia; some opacity of pulmonary valves.
61	39		21 0	Apoplexy; great hypertrophy of left ventricle, but no valvular disease; aorta much dilated and diseased.
62	32		12 8	Phthisis; no valvular disease.
63	33		21 0	Incompetency of aortic valves, from malformation by defect; thickening; morbus renum.
64	36		13 0	Pneumonia; hypertrophy of left ventricle; white spots on pericardium; no valvular disease.
65	38		15 0	Chronic bronchitis and emphysema; no valvular disease.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
66	30		24 0	Phthisis, pneumonia; aneurism of transverse arch; no valvular disease.
67	37		14 8	Delirium tremens; slight opacity and thickening of left valves.
68	34		16 0	Fever; no valvular disease.
69	32		14 8	Incompetency of aortic valves; diseased kidneys and spleen.
70	33		19 0	No material valvular disease; dilatation of aorta.
71	38		11 8	Phthisis; aorta dilated and diseased; some thickening of walls of right ventricle.
72	34		20 0	Hypertrophy and dilatation of right ventricle, without valvular disease.
73	30		14 8	Pneumonia; hypertrophy and dilatation of left ventricle; without valvular disease.
74	30		13 8	Chronic bronchitis; no valvular disease.
75	36		23 8	Aortic valves incompetent; some thickening of mitral and diseased aorta.
76	36		17 8	Mitral valvular disease; some thickening of aortic valves; hypertrophy, and dilatation both of right and left ventricles; hemiplegia.
77	33		14 0	Morbus renum; recent pericarditis; no material valvular disease.
78	34		22 4	Incompetency of aortic valves; slight thickening of mitral.
79	36		12 0	Fever; no valvular disease.
80	36		24 0	Incompetency of aortic valves.
81	32		18 0	Old pericarditis and disease of aorta, without material valvular disease; dysentery after typhoid fever.
82	40		12 0	Aortic valvular obstruction; malformation.
83	40		16 0	Recent pericarditis; aortic and mitral valves somewhat thickened; aorta dilated and diseased.
84	40		15 0	Fever, with jaundice; some thickening of mitral valves, and hypertrophy of left ventricle.
85	44		15 8	Phthisis after fever; no valvular disease.
86	45		11 8	Fever; white spots on pericardium; some thickening and opacity of aortic and mitral valves.
87	42		14 12	Chronic bronchitis; some thickening of aortic and mitral valves.
88	45		13 8	Fever; hypertrophy, with dilatation of left ventricle; no valvular disease.
89	43		13 8	Chronic bronchitis and emphysema; no valvular disease.
90	40		16 0	Pericardium universally adherent; aortic valves healthy; mitral somewhat thick.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
91	42		13 6	Fever ; no valvular disease.
92	43		14 8	Recent pericarditis ; chronic bronchitis; hypertrophy of right and left ventricles.
93	44		17 4	Old pericarditis ; no valvular disease ; great hypertrophy of left ventricle.
94	45		16 4	Chronic bronchitis ; thickening of mitral and aortic valves ; dilatation of aorta.
95	46		10 0	Phthisis ; white spots on pericardium ; no valvular disease ; aneurism of aorta.
96	44		20 0	Obstructive and regurgitant disease of aortic valves; rupture of aorta ; dissecting aneurism.
97	40		16 8	Obstruction of regurgitant disease of aortic valves.
98	50		11 12	Pleuropneumonia ; old pericarditis ; thickening of mitral valve.
99	51		13 0	Pericarditis ; bronchitis ; aneurism of coronary artery ; dilatation of cavities, with atheromatous disease of aortic and mitral valves, and of aorta.
100	51		13 12	Fever; no valvular disease.
101	51		21 8	Extensive thickening, and contraction of mitral, aortic, tricuspid and pulmonic valves ; thickness, 4, 5, and 2 lines ; great hypertrophy of right ventricle, old pericarditis ; morbus renum.
102	53		11 8	Chronic bronchitis ; white spots on pericardium and adhesions.
103	55		23 0	Aortic valves incompetent and some disease of aorta.
104	52		12 8	Chronic phthisis ; no valvular disease.
105	58		12 8	Hemiplegia ; no valvular disease.
106	50		23 8	Aorta much dilated and diseased ; no material valvular disease.
107	59		14 8	Aortic valves thickened and ossified, and aorta diseased.
108	53		10 8	Compound dislocation of astragalus ; recent pericarditis.
109	57		13 0	Cut throat—no valvular disease ; dilatation and hypertrophy of left ventricle ; aorta dilated and diseased.
110	52		14 8	Morbus renum ; mitral thickened; left ventricle hypertrophied.
111	51		21 8	Mitral valvular disease, with some thickening of aortic valves and diseased aorta.
112	58		23 0	Much obstruction at aortic orifice and some regurgitation ; mitral valve slightly thickened ; aorta dilated.

WEIGHT OF DISEASED HEARTS, MALES—*continued.*

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
113	67		13 0	Chronic bronchitis and emphysema ; thickening of mitral valve and hypertrophy of left ventricle.
114	60		11 8	Ulcer of stomach ; pneumonia ; opacity of aortic and mitral valves; aorta dilated and diseased.
115	57		16 0	Phthisis ; valves healthy, but mass of bone projecting into upper part of orifice from walls of aorta.
116	65		18 4	Apoplexy ; thickening of mitral and aortic valves ; hypertrophy and dilatation of left ventricle ; diseased aorta.
117	62		14 0	No valvular disease ; hypertrophy with dilatation.
118	71		23 0	Diseased prostate ; no valvular disease ; hypertrophy, with dilatation of right and left ventricle ; aorta dilated.
119	74		19 8	Convulsions ; thickening and ossification of aortic valves.
120	78		21 0	Thickening and ossification of aortic valves ; some thickening of mitral ; dilatation of aorta.
121	30		14 8	Fractured arm and delirium tremens ; white spots on pericardium ; no material valvular disease.

WEIGHT OF DISEASED HEARTS, FEMALES.

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
		lbs.	oz. dr.	
122	5 mos.	11	2 4	Malformation ; aperture in septum of ventricles, with contraction of tricuspid aperture.
123	14 yrs	95	11 12	Morbus renum.
124	17		11 0	Fever ; some thickening of mitral valve, with hypertrophy and dilatation of left ventricle.

WEIGHT OF DISEASED HEARTS, FEMALES—*continued*.

No.	Age.	Weight of Body.	Weight of Heart.	Cause of Death, and kind of Disease.
	Years.	lbs.	oz. dr.	
125	16		11 8	Lobular pneumonia; thickening and opacity of mitral valve.
126	54		15 8	Morbus renum; no material valvular disease.
127	29		11 8	Fatty degeneration of heart and kidneys.
128	21		13 0	Great contraction of mitral orifice and thickening of valves, as well as of pulmonic and tricuspid; aortic orifice small.
129	28		12 0	Morbus renum; coma; hypertrophy, with dilatation of left ventricle.
130	29		8 8	Phthisis; aneurisma aortæ.
131	34		15 0	Phthisis; pneumonia; general hypertrophy and dilatation without valvular disease.
132	34		12 8	Chronic bronchitis and emphysema; hypertrophy of right ventricle; no material valvular disease.
133	36		12 0	Fever; no valvular disease.
134	36		13 12	Morbus renum; no valvular disease.
135	30		12 8	Phthisis; no valvular disease; aorta greatly dilated.
136	40		17 0	Combined mitral, aortic, and tricuspid valvular disease, chiefly aortic; regurgitation.
137	44		23 0	Great incompetency of aortic valves; orifice dilated and valves thickened and indurated.
138	50		11 8	Chronic bronchitis; no valvular disease; hypertrophy and dilatation of right ventricle.
139	53		23 0	Combined mitral and aortic valvular disease; pleurisy.
140	57		12 8	Chronic phthisis.
141	60		14 0	Dissecting aneurism of descending aorta; no valvular disease.
142	60		17 8	Dilatation of aorta; and great hypertrophy and dilatation of left ventricle, without valvular disease.
143	66		18 0	Injury of head; no valvular disease; dilatation of aorta; hypertrophy and dilatation of left ventricle.
144	66		8 12	Recent endocarditis destroying aortic valves; pneumonia; meningitis.
145	67		13 8	Contraction of aortic orifice, and induration and thickening of valves.
146	70		13 0	Apoplexy; aortic valves thick and rigid; aorta dilated and atheromatous; hypertrophy of left ventricle.

Part I.—*Weights of the Healthy Heart.*

Table I.

Showing the weight of the healthy heart in males and females, at different periods of life, as deduced from 155 observations; males 94; females 61.

Ages.	Males.		Females.	
	Nos. Weighed.	Mean Weight.	Nos. Weighed.	Mean Weight.
Years.		oz. dr.		oz. dr.
15 to 20	9	8 $2\frac{2}{3}$	9	8 $1\frac{5}{9}$
20 to 30	27	9 $0\frac{4}{9}$	21	8 $10\frac{8}{9}$
30 to 40	31	9 $7\frac{13}{21}$	19	8 $13\frac{1}{19}$
40 to 50	9	9 $11\frac{1}{3}$	5	9 3
50 to 60	15	9 12	6	9 $7\frac{1}{3}$
60 to 70	3	10 $13\frac{1}{3}$	1	7 0
	94		61	

Table II.

Showing the different weights of the healthy heart in males and females, between 20 and 55 years of age, deduced from 125 observations.

Weights.	Males.		Females.	
	Nos. Weighed.	Per Centage.	Nos. Weighed.	Per Centage.
oz. oz. drs.				
5 to 6	2	5·2	3	12·2
6 to 7	2		3	
7 to 8	10	39·4	10	44·8
8 to 9	20		12	
9 to 10	18	39·4	13	42·8
10 to 11	12		8	
11 to 11 12	12	15·7		
	76		49	

	oz. dr.
Average weight of the healthy heart in 76 males from 20 to 55 years of age,	9 $8\frac{15}{76}$
Ditto ditto in 49 females ditto	8 $13\frac{6}{49}$

From the above table all organs weighing more than 11 oz. 12 dr. are excluded; if the cases, in which, without other disease, it weighed 12 oz. be calculated, the observations are extended to 83 males and 51 females, and the average weight of the heart in males is 9 oz. $11\frac{815}{83}$ dr.; in females 8 oz. 15 dr.

Table III.

Weight of the healthy heart, in persons from 20 to 55 years of age, in cases of phthisis, and of all others, exclusive of bronchitis and morbus renum, specifying the average weight in acute and chronic diseases, separately.

	Nos. Weighed	Mean Weight	Extremes	Mean Age
		oz. dr.	oz. dr. oz. dr.	
Males.				Years.
Phthisis, . .	27	9 $3\frac{1}{3}\frac{1}{3}$	11 0 and 6 $4\frac{1}{2}$	34·03
All others, . .	44	9 $9\frac{1}{3}\frac{1}{3}$		36
Acute cases only,	30	9 $13\frac{1}{3}\frac{1}{3}$	11 12 „ 8 8	36
Chronic cases only,	14	8 $14\frac{1}{14}\frac{1}{2}$	11 8 „ 5 0	35·9
Females.				
Phthisis, . .	17	8 $6\frac{1}{17}$	11 0 „ 5 8	34·4
All others, . .	23	9 $2\frac{2}{23}$		31·4
Acute cases only,	16	9 $5\frac{5}{16}$	11 0 „ 7 4	34·3
Chronic cases only,	6 [1]	8 $9\frac{6}{6}$	9 8 „ 8 0	30·2

The above table contains only the cases in which the weight of the heart did not exceed 11 oz. 12 dr.; if those in which it weighed 12 oz. be included, the result will stand as follows:—

Males,	Phthisis,	30	9 oz. $7\frac{13}{30}$ dr.
„	Others,	47	9 „ $12\frac{6}{47}$ „
Females,	Phthisis,	17	8 „ $6\frac{1}{17}$ „
„	Others,	24	9 „ $4\frac{7}{24}$ „

Table IV.

Weights of the heart in all cases of phthisis, bronchitis, and morbus renum, compared with the weights of the healthy heart in all other cases, hypertrophy, pericarditis, and morbus aortæ excluded.

Weights.	Phthisis.		Bronchitis.		Morbus Renum.		All others.	
	Males.	Fem.	Males.	Fem.	Males.	Fem.	Males.	Fem.
5 oz. to 6 oz		3					2	
6 „ to 7 „	2	2				1		
7 „ to 8 „	6	3			3	2	1	5
8 „ to 9 „	2	4		2	1		17	7
9 „ to 10 „	10	1				3	8	9
10 „ to 11 „	6	4	1	1	1	1	4	2
11 „ to 12 „	4		1	1	1	2	10	
12 „ to 13 „	3	2	2	1				
13 „ to 14 „			3			1		
14 „ to 15 „		1	3		1			
15 „ to 16 „	1					1		
Mean age, . .	34·1	35·3	46·4	38·4	30·1	33·4	36·5	31·4

[1] One case, in which the disease causing death is not stated, is excluded from this calculation.

Table V.

Weights of the heart in males and females under 20 years of age.

Age.	Males. No.	Mean Weight.	Heaviest.	Lightest.	Age.	Females. No.	Mean Weight.	Heaviest.	Lightest.
		oz. dr.	oz. dr.	oz. dr.			oz. dr.	oz. dr.	oz. dr.
6 months	1	0 $13\frac{1}{2}$			7 days	1	0 13		
10 months	1	1 10			$2\frac{1}{2}$ months	1	1 3		
2 and $2\frac{1}{2}$ yrs.	2		2 12	2 10	3 months	1	1 0		
3 years	1	2 15			1 year	1	2 0		
4 and $4\frac{1}{2}$ yrs.	2		4 4	2 9	$1\frac{1}{2}$ years	1	1 8		
3 years	1	3 0			3 years	1	1 10		
8 „	1	6 4			5 „	1	2 14		
10 „	1	4 4			6 „	1	2 12		
11 „	5	5 $10\frac{1}{2}$	8 8	4 0	7 „	2		3 0	2 3
14 „	2		9 0	6 8	8 „	2		5 1	3 0
15 „	2		8 0	6 0	11 „	1	3 14		
16 „	3	6 $5\frac{1}{3}$	7 0	5 8	13 „	1	6 2		
17 „	1	8 8			17 „	2		7 0	4 8
19 „	1	11 0			18 „	3	9 5	10 8	7 15
					19 „	2		8 0	7 12
	24					21			

Of the observations included in this table, all but 11 were cases of acute disease, and the results would not have been materially different had the cases of chronic disease been excluded from the calculation. The difference in the weights of the heart at different ages, in young persons, is chiefly due to the relative vigour of the children and the greater or less rapidity with which their growth proceeds; and in this respect the rate of growth of the heart corresponds with that of the brain, and indeed of the body generally.

INFERENCES.

1. The weight of the healthy heart in persons from 20 to 55 years of age, averages in males 9 oz. 8 dr., and in females 8 oz. 13 dr. The mean difference between the weights of the organ in the two sexes being thus 11 drachms. This calculation is based only upon the observations in which the heart weighed less than 12 oz., if those in which it attained the weight of 12 oz. be added, the average becomes somewhat higher, or 9 oz. 11 dr. in males, and 8 oz. 15 dr. in females, and the difference between the weights in the two sexes is 12 drachms.

Calculations of this description must always be to a certain extent arbitrary, for as the heart is found in some cases to be considerably above its ordinary weight, without the proportion of its walls and cavities being materially altered, or the organ being otherwise diseased, it is not easy to say at what point it ceases to be healthy. The estimates of the weight of the healthy heart here given differ in some degree from those of other observers. Bouillaud[1] infers that the average weight of the heart is from 8 to 9 oz. (systeme usuel, or from 8 oz. 10 dr., to 9 oz. 11 dr. avoird.), and may amount to 10 or 11 oz. (10 oz. 12 dr., and 11 oz. 14 dr. avoird.), but his observations refer to only 14 cases, and include individuals of both sexes and of various ages from 16 to 38. The mean weight of 8 adult hearts given in his table is 9 oz. 2 dr. (9 oz. 15 dr. avoird.), the extremes being 7 oz. 6 dr. (8 oz. 5 dr. avoird.), and 11 oz. 3 dr. (12 oz. 4 dr. avoird.)

Dr Clendinning[2] estimates the average weight of the male heart in persons from 20 to 60 years of age, at 8½ oz. avoird.; the female heart at 7⅔ oz. avoird., and his calculations are based upon 118 observations (58 males and 60 females). Dr Reid's[3] researches give the average weight of the heart in 89 males, from 25 to 55 years of age, as 11 oz. 1 dr., and in 53 females, as 9 oz.; the difference being 2¼ ounces.

2. The weight of the healthy heart differs in different forms of disease, being greater in persons who die after short periods of illness, and less in those who have suffered from protracted and emaciating diseases.

This result is illustrated in table 3, from which it will be seen that the weight of the heart in adult males who sank under acute diseases, averaged 9 oz. 13 dr., while in those who had died of chronic diseases, phthisis, bronchitis, and morbus renum being excepted, it averaged 8 oz. 14 dr. In females the heart weighed in those who had died of acute diseases, 9 oz. 5 dr., and in those who sank from chronic diseases, 8 oz. 9 drachms.

Dr Reid found the mean weight of the heart in 9 adult males, who had died from accidents, to be 12 oz. 6 dr., while, as before stated, in the adult males who sank from disease, it weighed on an average 11 oz. 1 dr.; but in both these calculations the weights of hearts are included, which exceeded what I have regarded as the limit of health, either in cases of accident or disease.

3. The general inference to be deduced from these observations, would appear to be that in adult males who have died from acute diseases, or from the effects of accidents, the ordinary weight of the healthy heart is from 9 to 11 oz. avoird., and in those who have

[1] Maladies du Cœur, 2me Ed., 1841, t. i., p. 25.
[2] Med. Chir. Trans., vol. xxi., 1838, p. 55.
[3] Lond. and Ed. Monthly Journal of Medical Science, 1843. Also, Physiological, Pathological, and Anatomical Researches, Ed. 1848, p. 376.

died from chronic diseases from 8 to 10 oz. In females the ordinary weight of the heart in acute cases may be regarded as from 8 to 10 oz., and in chronic diseases from 7 to 9 oz. Occasionally, however, in persons of small and delicate frame, who have died from exhausting diseases, such as cancer of the stomach or chronic affections of the liver, the heart will be found to weigh only 5 or 6 oz.; and in large and powerful persons, of the male sex, who have been suddenly killed or have died after a very short illness, the organ may weigh 12 oz. or perhaps even more, without exceeding the limit of health.

4. The heart increases in weight with the advance of life.

This inference is borne out by the facts collected in table 2, for the exception formed by the single case in advanced life, in females, is unimportant. It is also confirmed by the observations of Drs Clendinning and Reid, and by a table, compiled from their data, in Dr Sharpey's edition of Quain's Anatomy.[1] It is not, however, clear, to what period of life this increase extends, and whether in very advanced age, there is not, as is shown to be the case with the brain, a more or less marked decline in weight.[2] Hospitals do not afford the means of ascertaining satisfactorily the weight of the heart in aged persons, for comparatively few old people die in them, and of these a large proportion labour under some form of cardiac disease; it is therefore very desirable that medical men having charge of public charitable institutions for the aged, should direct their attention to this point. From some observations which I have made, I have been led to suppose, that when entirely free from disease, the heart, so far from continuing to increase in weight to the term of life, undergoes a decrease in advanced age; and I have more especially noticed this to be the case in females, in whom the decrease of weight of the brain in advanced life, is noticed at the earliest period and to the greatest extent. The opposite result arrived at by other observers, I cannot but suspect to have been due to organs, not strictly healthy, having been included in their calculations.

5. *Weight of the Heart in Phthisis, Bronchitis, and Disease of the Kidneys.*—The weight of the heart in persons who have died of phthisis, is less than in those who have sank from other diseases; but the decrease of weight after death from that disease is usually not so marked as in persons who have died from other chronic affections, unconnected with disease of the lungs.

This inference is deduced from the facts which appear in table 3, there being, however, an exception as to the relative weight of the heart in phthisis and in other chronic diseases, in females; but the latter cases are too few in number to form a satisfactory basis for generalization.

[1] Vol. ii., p. 1124.
[2] See observations by Dr Reid and myself, in Lond. and Ed. Journal, 1843, and 1846, and by myself in the Lond. Journal of Medicine.

The conclusion here drawn as to the relative weight of the heart in phthisis and in other diseases, differs considerably from that arrived at by Dr Clendinning, that the weight of the heart in phthisis is greater than its weight in other diseases, in which the organ is healthy, I have therefore carefully compared the two series of observations with the view of ascertaining the cause of the discrepancy. After re-calculating the data collected by Dr Clendinning, separating from them all the weights which exceeded the limit of health, and classifying the remainder according to the acute or chronic character of the disease occasioning death, the results are as follows :—

		oz.	dr.
Males.	Weight of the heart in cases of phthisis	9	9
,,	,, ,, of acute disease only	9	1
,,	,, ,, of chronic disease only	8	0
Females.	Weight of the heart in cases of phthisis	7	13
,,	,, ,, of acute diseases only	8	8
,,	,, ,, of chronic diseases only	7	8

It will thus be seen that it is only in reference to males that the observations collected by Dr Clendinning, bear out the inference which he has drawn from them, that in phthisis the heart is heavier than in other diseases; while in females, the results are similar to those deduced from the larger series of data contained in the paper, and show that the weight of the heart in phthisis, is ordinarily less than in acute diseases, but greater than in other chronic diseases, unconnected with obstruction in the lungs. The correct explanation most probably is, that in phthisis there is a tendency to enlargement of the heart, which, though counteracted to a greater or less extent by the emaciation, prevents the organ declining so much in weight as would have been the case were no source of obstruction present. This inference, though different both from the conclusions of MM. Louis and Bizot and Dr Clendinning, is quite compatible with the correctness of their observations, and the different results arrived at by them and by myself, is doubtless due to their having compared the condition of the heart in phthisis with its state in *all* other diseases, instead of with its weight and size in *chronic* diseases *only*. There is certainly nothing in phthisis which prevents the heart becoming considerably hypertrophied, as is shown by two cases included in Dr Clendinning's paper in which it weighed 13 oz. 4 dr. and 13 oz. 8 dr. in males; and by others in my own in which it weighed 15 oz. 8 dr. in a male, and 15 oz. in a female.

6. The facts collected in table 4 show, that in chronic bronchitis the heart ordinarily acquires a considerable increase of weight. In two cases only of persons who had died of this disease did the organ possess its average weight in chronic diseases, while in 9, 8 males and one female, it exceeded the weight regarded as the extreme limit

of health; and, in three of them, all of whom were males, it weighed from 15 to 16 oz. Though the number of cases contained in the table is so few, yet the results are too marked to be regarded as accidental.

7. The condition of the heart in cases of disease of the kidneys is also shown in table 4; and from this it will be seen that in 7 cases, or 4 males and 3 females, the weight of the organ was below the average in other chronic diseases; while in 11, 3 males and 8 females, it exceeded the average, attaining in one male the weight of 14 oz. 8 dr., and in 2 females of 13 oz. 12 dr., and 15 oz. 8 dr. The facts, therefore, confirm, to a certain extent, the observations of Dr Bright,[1] that the disease of the kidneys has a tendency to produce enlargement of the heart, unconnected with valvular disease, or disease of the aorta.

Part II.—*Weight of Diseased Hearts.*

Table VI.

Weight of the heart in cases in which it exceeded the limit of health, but in which there was no obvious source of obstruction; and in cases of old adhesions of pericardium, and disease of the aorta (including atheromatous deposit, dilatation, and aneurism), unconnected with valvular defect.

Weights.	Hypertrophy without Obstruction.		Adhesions of the Pericardium.		Aortic Disease.			
	Males.	Age.	Males.	Age.	Males.	Age.	Females.	Age.
8 oz. 8 dr.					1	46*	1	29*
10 ,, 0 ,,								
11 ,, to 12 oz.			1	50	2	38* and 60		
		Mean.						
12 ,, to 13 ,,	5	37·2			2	57 and 65	1	30
13 ,, to 14 ,,	5	50					1	60
14 ,, to 15 ,,	4	46·7			1	29		
15 ,, to 16 ,,	1	34	1	40	1	45		
16 ,, to 20 ,,	2	45 and 34	2	44 and 32	4	38 to 78 Mean 56·4.	2	60 and 66
20 ,, to 24 ,,					5	30* to 71 Mean 52·2.		
40 ,, 12 dr.	1	65						

[1] Guy's Hospital Reports, vol. i., 1836, p. 380.

In 11 out of the 18 cases of hypertrophy included in the table it is stated in the reports that there was no valvular disease, and in the others any slight thickening, opacity or atheroma which existed was inadequate to explain the increase of weight. In all the cases, also, there was an absence of material chronic disease of the lungs and aorta.

In two females the hearts weighed 11 oz. and 12 oz. in persons of 17 and 36 years of age: in the former, though there was slight thickening of the mitral valves, it was inadequate to explain the unusual weight of the heart for a person of the age, and in the second there was no valvular disease.

From the table all the cases in which there was recent pericarditis are excluded, and one, in which the pericardium was universally adherent by old attachments, but in which there was chronic bronchitis, is also omitted. In the case of the male, 32 years of age, in whom the heart weighed 18 oz., there was not only adhesion but extensive ossification of the pericardium. The cases marked with an asterisk were aneurisms. In two of these in which the heart weighed 8 oz. 8 dr. and 24 oz., the patients were phthisical, and this was also the case in the female in whom the heart weighed 12 oz. 8 dr., and the aorta was dilated.

Table VII.

Weight of the heart in cases of aortic valvular disease, whether obstructive, regurgitant, or both.

Weights.	Males.			Females.		
	Obstruct.	Obst. and Regurgit.	Ages.	Obstruct.	Obst. and Regurgit.	Ages.
			Years.			Years.
8 oz. and 12 dr.					1	66
10 „ and 12 „				1		75
11 oz. to 12 oz.	1	1	40 and 57			
12 „ to 13 „				1		70
14 „ to 15 „	2	2	44.7			
15 „ to 16 „	3	3	45.5		1	46
16 „ to 20 „	1	3	47.7	1		67
20 „ to 25 „	1	8	44.5		1	44
28 „		1	18			
34 „		1	55			

In several of these cases there was also dilatation or atheromatous disease of the aorta, and, generally, some thickening of the mitral valve.

Table VIII.

Weight of the heart in cases of mitral valvular disease.

Weights.	Males.	Age.	Females.	Age.
		Years.		Years.
8 oz. and 8 dr.			1	27
10 „ and 8 „			1	23
11 „			1	17
12 „ and 12 oz. 8 dr.			2	62 and 40
14 „ and 14 „ 8 „	2	29 and 52		
15 „ and 12 dr.			1	39
17 „ and 8 „	1	36		
18 „			1	21

Of the cases included in this table the male in whom the heart weighed 14 oz., and the two females in whom it weighed 8 oz. 8 dr. and 11 oz. died respectively of attacks of cholera, peritonitis and fever.

In one case not entered in the table, there was free regurgitation through the left auriculo-ventricular aperture from dilatation, without disease of the valves; and the organ weighed 10 oz. 8 dr.; the subject being a girl 8 years of age. In several cases the tricuspid valves were also slightly thickened.

Table IX.

Weight of the heart in cases of combined aortic and mitral valvular disease.

Weights.	Males.	Age.	Fem.	Age.
oz. dr.				
7 8			1	11
12 0			1	12
13 8			1	18
14 8	1	24		
17 0			1	40
18 4	1	65		
21 8	1	51		
22 0			2	39 and 63
23 0			1	53

In the three cases in which the organ attained the greatest weight, the predominant valvular disease was mitral. In that in which the heart weighed 21 oz. 8 dr., the pericardium was also universally

adherent, and there was considerable thickening of the tricuspid valves, and disease of the aorta; and in some of the other cases the tricuspid valves were also slightly thickened.

INFERENCES.

1. The weight of the heart may very greatly exceed the limit of health without the existence of any material valvular, aortic, or pulmonic disease, adhesions of the pericardium or other obvious source of obstruction.

Thus it will be seen in Table VI. that in 18 males, the weight of the heart exceeded 12 oz., and in 5 cases it amounted to 15 oz., 16 oz., 17 oz. 8 dr., 20 oz. and 40 oz. 12 dr., the last being the heaviest heart weighed. In all these cases there existed no source of obstruction, either in the heart itself or in the aorta or lungs, which appeared sufficient to explain the great increase of weight, and in 11 of them it is expressly stated in the reports, that there was no valvular disease. In the case in which the heart weighed 40 oz. and 12 dr., though there was slight atheromatous change in the aortic and mitral valves and in the coats of the aorta, the lungs were healthy, and nothing sufficient to explain the hypertrophy was detected.

The process by which the heart attains this great increase of weight in cases where there is no marked obstruction to the circulation, is most probably a slow one, but the data given show that there is no relation between the age of the individual and the amount of hypertrophy. The heart is found to weigh very considerably above the average in comparatively young persons, and the more extreme degrees of hypertrophy are not more common in advanced age, than in persons at earlier periods of life.

Only two cases which could be regarded as instances of simple hypertrophy in females are included in the tables, and, in neither of these did the organ exceed 12 oz. in weight;—facts which show the infrequency of this form of disease in females, and the very slight increase of weight which the female heart attains, when there is no serious source of obstruction.

2. The tables contain only four cases in which there were old adhesions of the pericardium, without valvular, aortic, or pulmonic disease, or recent pericarditis;—and this number is too small to warrant any decided inferences. It will, however, be seen that in one instance the weight of the heart did not exceed the limit of health, being only 11 oz. 12 dr., while, in the other three cases, it was very considerably greater, or 16 oz., 17 oz. 4 dr., and 18 oz. These facts do not confirm the opinion that adhesions of the pericardium, so far from leading to enlargement of the heart, rather tend to produce atrophy; but I have examined other hearts in which the pericardium was entirely adherent by old cellular attachments, without the organ being larger than natural. The person in whom the heart weighed 18 oz. was a male, 32 years of age, and, in this instance not only was the pericardium much thickened and adherent, but there was also a considerable formation of bone.

3. The heart most generally acquires very great increase of weight in cases of disease of the aorta, whether consisting in atheromatous deposit and ossification in the coats, dilatation, or sacculated aneurism; but in none of the cases of this description did the heart attain so great a weight as in one of those in which there existed no obvious source of obstruction to explain the enlargement.

The last column in Table VI. shows that in one female the weight of the heart did not attain the average, being only 8 oz. 8 dr. In one male also it only slightly exceeded the average, being 10 oz.; and in two other males it did not exceed the limit of health, weighing in each 11 oz. 8 dr. In the remaining cases (seventeen in number—thirteen males and four females), it very considerably passed that point, weighing from 12 oz. to 24 oz. in males, and from 12 oz. 8 dr. to 18 oz. in females; the greatest weights being, in males, 16 oz. 4 dr., 18 oz. 4 dr., 19 oz., 21 oz., 23 oz., 23 oz. 8 dr., and 24 oz.; and in females, 17 oz. 8 dr., and 18 oz. It must, however, be observed, that in the male in which the heart weighed only 10 oz., in a second in which it weighed 24 oz., and in the female in whom it weighed 8 oz. 8 dr. in addition to the aneurisms, the lungs were tuberculous; and in the female in whom the heart weighed 12 oz. 8 dr., the aorta was dilated and the lungs tuberculous, so that, in these instances, the hypertrophy of the heart resulting from the aortic obstruction, may have been to some extent counteracted by the emaciation from phthisis.

As in the cases of hypertrophy only, so also in this form of disease, there is no just relation between the ages of the individuals and the increase in the weight of the heart.

4. The heart usually becomes very greatly enlarged in cases of aortic valvular disease, whether obstructive or regurgitant, or both; and the increase of weight is apparently greater in these cases than in those of disease of the aorta; but in no instance did the organ acquire from this cause so great a weight as in one of the cases of hypertrophy unconnected with obvious obstruction.

These inferences are deduced from Table VII., from which it appears that in males the weight of the heart exceeded the average in all the cases; but, in two instances, in one of which there was obstruction only, in the other regurgitation, it did not pass the extreme limit of health, weighing only 11 oz. 8 dr., and 12 oz. In seven other cases of obstruction its weight ranged from 14 to 21 oz., the greatest weights being 16 oz., 16 oz., 19 oz., and 21 oz.; and in eighteen cases of regurgitation, from 14 oz. to 34 oz.; the greatest weights being 20 oz., 21 oz., 22 oz. 4 dr., in three cases 23 oz., 23 oz. 8 dr., in two cases 24 oz., 28 oz., and 34 oz.

In females, the increase of weight in cases of aortic valvular disease also obtains, though to a less degree. If a case in which one of the segments was destroyed by acute endocarditis, and in which death

rapidly ensued, and the organ weighed only 8 oz. 12 dr., be excluded, the lightest heart in the table weighed 10 oz. 12 dr., or within the limit of health; yet, in this instance, there was most extensive thickening and ossification of the valves, probably originating in malformation, and causing extreme obstruction: and as the subject of the case was 75 years of age, the progress of the disease must have been very slow. In two other cases of obstructive disease the heart weighed 13 oz. and 18 oz. 8 dr., and in two cases of regurgitation 16 oz. and 23 oz.

It will thus be seen that, though the weight of the heart in cases of aortic valvular disease was greater than in the cases of disease of the aorta, the heaviest heart in the former weighed nearly 7 oz. less than in the remarkable case of hypertrophy without valvular or aortic disease before mentioned.

The table also shows that the weight of the heart in cases of aortic valvular disease is by no means proportionate to the age of the subjects; the organ having attained a weight of 28 oz. in a boy of 18, and 23 oz. in a female of 44.

It is not possible to ascertain the respective effects of obstructive and regurgitant disease in increasing the weight of the heart, for the two generally coexist, and the latter is very frequently only the final stage of the former; the influence which is due to each cause cannot therefore generally be assigned. In the case, however, in which the heart weighed 17¾ oz. in a male 33 years of age, the disease was the result of rupture of the angle of attachment of two of the valves, from violent muscular exertion, 27 months before death, and though there must have been some obstruction to the flow of blood from the ventricle into the aorta, the great increase of weight must have been chiefly due to the incompetency of the valves. A still more remarkable instance of enlargement from regurgitation, is afforded by a case of rupture of the valves, related by Dr Quain, in which the heart acquired the weight of 22½ oz. in a period of two years which elapsed between the occurrence of the accident and the death of the individual. In both these cases the heart was most probably healthy till the accidents occurred.

In all cases of regurgitant disease, on whatever cause dependent, the increase of weight probably takes place rapidly; thus, in the boy of 18, in whom the organ weighed 28 oz., the duration of illness had only been two years and ten months; on the contrary, in cases of obstructive disease, the enlargement of the heart is probably a slower process. A striking example of the length of time during which obstruction may exist without the heart acquiring great increase of weight, is afforded by the case of the elderly female, before referred to, in whom the organ weighed 10 oz. 12 dr., but in this instance, it seems probable, that a decrease of weight may have occurred with the advance of life. The case, however, with many others which might be quoted, shows the absence of any just relation between the degree of obstruction and the amount of hypertrophy.

5. In cases of mitral valvular disease the heart ordinarily exceeds the limit of health, but does not attain so great an increase of weight as in cases of aortic or aortic valvular disease.

Of the cases included in Table VIII., three, that of the male in whom the heart weighed 14 oz., and two females, in whom it weighed 8 oz. 8 dr., and 11 oz., death did not result from the direct effect of the disease, but from other causes. If, therefore, these cases be excepted, it will be seen that in one female the heart weighed only 10 oz. 10 dr., or within the limit of health, while in the six other cases it greatly exceeded that point, weighing, in males, from 14 oz. 8 dr. to 17 oz. 8 dr., and in females from 12 oz. to 18 oz. These weights are, however, much less than those in the cases of aortic and aortic valvular disease, and still less than that of the heart in which the hypertrophy was not dependent on obvious obstruction. It will also be observed, that in these cases there is not the great difference in weight between the male and female heart which is observed in other forms of disease, but the cases are so few, that the inferences cannot be fully depended upon.

The weight of the heart in cases of mitral valvular disease bears no certain proportion to the age of the individual, as is shown by the subject of the case in which the heart weighed 18 oz. being a female only 21 years of age.

6. In disease of the mitral and aortic valves combined, the weight of the heart is generally intermediate between that in cases of aortic and mitral disease alone; the organ being lighter than in aortic valvular disease, and heavier than in mitral valvular disease.

It will be seen from Table IX., that if the case in which the heart weighed 7 oz. 8 dr., in a girl 11 years of age, be omitted, in all the observations, including two females of 12 and 18 years of age, the weights of the heart exceeded the extreme limit of health; and that in three males it weighed from 14 oz. 8 dr. to 21 oz. 8 dr., and in three females from 17 oz. to 23 oz.

7. The tables contain only one observation of the weight of the heart in extensive disease of the mitral and tricuspid valves. In this instance the disease was probably congenital. The organ weighed 9 oz., and the subject was 37 years of age, but she was peculiarly ill-formed and stunted, and did not appear more than 16 or 18 years old.

8. In the tables the weights of four hearts are given in which there were congenital malformations. Of these cases that which presented the least important deviation from the natural structure, was one in which the foramen ovale was unclosed, in a girl 8½ years of age, and the heart weighed 6 oz. 8 dr. A second case was that of a girl aged 5 years, who died of hemorrhage from the throat or stomach, during an attack of scarlet fever, and the only malformation was the existence of a septum dividing the infundibular portion of the right ventricle from the sinus, and the heart weighed 3 oz. 12 dr. In a third case, that of a boy 15 years of age, in addition to a similar source of obstruction in the right ventricle, the

pulmonary artery was small and its valves malformed, and there was an aperture in the septum ventriculorum, by which the aorta freely communicated with the right, as well as with the left ventricle; in this instance the heart weighed 10 oz. In three other cases (two of which are not included in the tables), the pulmonary orifice was the seat of obstruction. In one of these, a young man, 20 years of age, it was very greatly contracted from the adhesion and thickening of the valves, and the foramen ovale was largely open, and the heart weighed 12 oz. In another, a female, 19 years of age, the pulmonary orifice was greatly contracted from malformation of the valves, the tricuspid valves were also diseased; the aorta arose from both ventricles, and the ductus arteriorus was still pervious; and the heart weighed 17½ oz. The subject of the third case was an infant nearly 12 months old, in which the pulmonary artery was entirely obliterated, the aorta arose from both ventricles, and the blood was transmitted to the lungs through the ductus arteriosus. The heart in this case weighed 3 oz. 8 dr. The last instance of malformation is one in which, in a female infant aged 5 months, there were two small apertures in the septum ventriculorum, with thickening of the tricuspid valves, causing contraction of the orifice, and the heart weighed 2 oz. 4 dr. A comparison of the weights of the heart in these cases with that of the healthy organ in infancy and early life, as given in Table V., will show how greatly it exceeded the ordinary weight in healthy children at similar ages; indeed, in the female 19 years of age, it very considerably exceeded the extreme limit of health in adults.

General Remarks on the Weight of the Diseased Heart.

The weights of the diseased heart in the above tables are, so far as I am aware, the most extensive series yet published; indeed with the exception of M. Bouillaud, I do not know any writer who has specially collected the weights of diseased organs. M. Bouillaud's observations are, however, very few in number, amounting to only 11 cases in which the heart was hypertrophied, and 7 which he regarded as cases of atrophy. Of the latter, in five males, the weights ranged from 5 oz. 6 dr. in a person 18 years of age, to 7 oz. in one of 20, and the causes of death were marasmus, typhoid, and the effects of having taken nitric acid. The two females were aged 45 and 30, and died respectively of schirrus of the pylorus, and of disease of the liver, and the hearts weighed 4 oz. 5 dr., and 5 oz. 13 dr. Of the observations in the paper, the lightest male heart weighed 5 oz. in a person 53 years of age, who died of cirrhosis hepatis, combined with disease of the kidneys. In a second case the heart weighed 6 oz. in a person 39 years of age, who died of cancer of the pylorus, and in a third, the heart had the same weight in a man 29 years old, the cause of whose death is not recorded. The lightest female hearts weighed 5 oz. 8 dr. in two persons 25 and 35

years of age, and 5 oz. 12 dr. in a third, 21 years old, and all these died of phthisis.

The weight of the heart in the cases of hypertrophy collected by M. Bouillaud, are as follows:—

Hypertrophy apparently without Valvular disease.

1. Male,	60 years of age,	. .	12 oz. 15 dr.	avoirdupois.
2. „	50 „	. .	13 „ 11 „	„
3. Female,	75 „	. .	14 „ 10 „	„
4. Male,	67 „	. .	18 „ 3 „	„
. „	69 „	. .	17 „ 10 „	„

Aortic Valvular obstruction and probably Regurgitation.

6. Female,	54 years of age,	. .	24 oz. 4 dr.	avoirdupois.

Mitral Valvular Regurgitation.

7. Male,	47 years of age,	. .	22 oz. 3 dr.	avoirdupois.

Mitral Valvular obstruction and Regurgitation.

8. Female,	56 years of age,	. .	12 oz. 15 dr.	avoirdupois.
. Male,	53 „	. .	16 „ 3 „	„

Combined Mitral and Aortic Valvular disease.

10. Female,	17 to 18 years,	. .	11 oz. 14 dr.	avoirdupois.
11. Male,	40 years of age,	. .	18 „ 10 „	„

In the tables in the paper the weight of only one heart is given in which there was regurgitation through the left auriculo-ventricular aperture, without disease of the valves, and in this case the patient was a girl only 8 years of age;—so that no comparison can be instituted between the weight of the heart in M. Bouillaud's 7th observation and my own; but, with this exception, it will be seen that the weights of the heart, in the different forms of disease in the comparatively small number of observations published by M. Bouillaud, correspond with those deduced from my own much more numerous data; though in none of his observations did the heart attain so great a weight as in several of my own.[1]

In Dr Clendinning's tables the lightest male heart weighed 6 oz., in three cases, in persons who died of ascites, mania, and phthisis, at the ages of 24, 41, and 43; and the lightest female hearts weighed 4 oz. 8 dr., 5 oz., 5 oz. 8 dr., and 5 oz. 4 dr., in persons aged respectively 32, 31, 20, and 26, who died of epilepsy with ulceration of the intestines, chronic bronchitis, typhus, and phthisis.

[1] In M. Bouillaud's treatise the weights are given in grammes, and also in the pound, ounce, and gros. The pound being the poids de marc of 7560·6 grains troy, and the ounce and gros respectively the medicinal weight of 472·5 and 59·1 grains. The weights given above, are avoirdupois, calculated from the gramme, as equal to 15·434 troy grains.

The heaviest hearts in males weighed 24 oz., 24 oz. 8 dr., 26 oz.[1] and 40 oz. 8 dr., in persons 48, 34, 33, and 33 years of age; but, except in reference to the last case, in which there was aneurism of the aorta near the heart, the nature of the disease causing death is not reported. It is curious that the heart in the last case should be precisely the same weight as the heaviest which I have weighed. In females the greatest weights given by Dr Clendinning are 15½ oz. and 17½ oz. in persons 23 and 50 years of age; but though the foramen ovale is stated to have been open in the former case, the precise kind of disease is not mentioned in either.

M. Bouillaud[2] concludes his observations with the remark that, in cases of hypertrophy, the weight of the heart may amount to nearly three times that of the organ of usual dimensions, and five times that of the most atrophied heart. It will, however, be seen, from the data here published, that this estimate is too low. As in males the average weight of the heart is 9 oz. 8 dr. (oz, if the cases in which the weight of the organ was 12 oz. be included, 9 oz. 11 dr.), and the extreme weights of the organs examined were 5 oz. and 40 oz. 12 dr., the weight in the most hypertrophied heart was upwards of four times that of the average organ, and eight times that of the lightest heart weighed. In females, the range of weight in the heart, though sufficiently remarkable, is less than in males. The mean weight of the heart in females is 8 oz. 13 dr. (or 8 oz. 15 dr.), and the extreme weights are 5 oz. 8 dr. and 23 oz., so that the most hypertrophied heart was nearly three times the weight of the average organ, and four times that of the lightest heart. The greatest weight recorded was in a case of hypertrophy without material valvular disease; and the lightest hearts were found in cases of cancer of the pylorus and disease of the liver—an observation which accords with the results of M. Louis[3] as to the small size of the heart in cases of this description.

M. Bouillaud, in referring to the statement of Lobstein, that a heart examined by him weighed 2 lbs. (or 34 oz. avoird.), suggests that the organ had most probably been weighed before being deprived of its coagulum and blood; but this weight is so far from being extreme, that there is no reason to doubt the correctness of M. Lobstein's report. Dr Hope[4] mentions having examined, at St George's Hospital, a heart which weighed 2½ lbs., which, if, as is probable, the weight employed was avoirdupois, equals the heaviest organs weighed by Dr Clendinning and myself.

[1] This case is evidently twice inserted in the Tables.
[2] Traité Clinique des Maladies du Cœur 2me Ed. 1841, Tome i. p. 68.
[3] Sydenham's Society's Translation, p. 52.
[4] Diseases of the Heart, 3d Ed., 1839, p. 238.

TABLE X.—Showing the dimensions of the healthy heart *in males*, specifying in separate columns, the cases of phthisis, bronchitis, and of other diseases, unconnected with pulmonary disease, together with the dimensions of the heart in persons dying of chronic diseases *only*, unconnected with disease in the lungs. The table includes the mean of all the observations, together with the dimensions of the lightest and heaviest hearts examined.

	Phthisis.			Bronchitis.			Other.			Chronic cases only.		
	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest	Mean.	Lightest	Heaviest
	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.	Hors.
Circumference of the heart,	94·5	92	97	118·6	116	130	103·1	98	87	104·4	98	120
Girth of right ventricle,	49·5	50	49	67·3	66	76	55·6	54	43	57·2	54	60
„ left ventricle,	45·	42	43	51·3	50	54	47·5	44	39	47·2	44	60
Length of the cavity of right ventricle,	43·3	38	48	50	51	51	42·5	42	51	40	42	54
„ left ventricle,	37·3	36	39	40·6	46	36	37·	36	40	34·4	36	32
Thickness of walls of right ventricle base,	2	2	2	2·5	2	3	1·91	2	2	1·8	2	2
„ midpoint,	2	2	2	3	2·6	4·5	1·80	2	2	1·50	2·25	3
„ apex,	1·3	1·25	1·5	1·3	1·6	1·5	1·57	2·5	1	1·5	2	1·5
„ of left ventricle base,	5·3	6	4	5·5	6	6	5·1	6	6	4·7	5	6
„ midpoint,	6·3	5·5	7	5·8	5·5	6	5·95	6	6	5·8	6	6
„ apex,	2·3	2·5	2	2·5	2·5	2	2·5	3	2	2·8	3	3
„ of septum,	5·8	5	6	5·68	5	6	5·77	6	6	5·6	6	5
Circumf. of right auriculo-ventric. aper.,	49·3	51	43	58·6	62	64	54·4	57	57	57·7	57	60
„ of pulmonic,	38·6	39	39	46	45	45	40	39	42	40·3	39	42
„ of left auriculo ventricular,	45·3	40	57	54	54	54	44·3	42	45	47·3	42	48
„ of aortic,	34·6	36	26	39	39	36	35·5	33	39	36·4	33	39

The observations in cases of phthisis are three; the ages 20, 31, and 37; and the weights of the heart, 6 oz. 4½ dr., 9 oz. 4 dr., and 11 oz.
The number of cases of bronchitis, etc., is also three, the ages 38, 40, 72, and the weights of the heart 11 oz., 12 oz. 8 dr., and 14 oz.
The number of other cases is twelve, the ages ranging from 23 to 66, and the mean age 52·8; the weights of the heart range from 6 oz., 9½ dr. to 12 oz., and the mean weight is 9 oz. 4·7 dr.
The number of cases of chronic disease only are five, the ages 23 to 35, the weights of the heart from 6 oz. 9½ dr. to 9 oz. 1·5 dr.

TABLE XI.—Dimensions of the healthy heart *in females*, specifying separately the dimensions in cases of phthisis, bronchitis, and of all other diseases, unconnected with pulmonary disease.

	Phthisis.			Bronchitis.			Other.		
	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of the heart,	102·6	90·	108·	93·	94·	92·	107·8	103.	120·
Girth of the right ventricle,	46·	42·	60·	53·	54·	52·	59·	60·	72·
,, left ventricle,	40·6	42·	48·	40·	40·	40·	48·8	48·	48·
Length of the cavity of the right ventricle,	43·3	42·	48·	42·6	44·	42·	49·7	42·	48·
,, left ventricle,	36·6	37·	42·	34·6	32·	36·	37·5	39·	39·
Thickness of walls of right ventricle, base,	1·6	1·5	2·	2·67	4·	3·	1·91	1·	2·
,, ,, midpoint,	2·3	1·5	3·	1·67	2·	2·	1·67	1·5	1·5
,, ,, apex,	1·3	1·5	1·5	1·67	2·	1·	1·67	1·5	1·
,, of left ventricle, base,	4·8	6·	3·5	5·2	4·	4·	4·9	4·	5·
,, ,, midpoint,	5·6	7·	5·	6·	4·	7·	5·8	5·	6·
,, ,, apex,	3·16	2·	2·	2·25	2·	2·	2·7	2·5	2·5
,, of septum,	3·5						4·7	4·	6·
Circumf. of right auriculo-ventricular apert.	54·	51·	60.	47·5	51·	40·	52·1	54·	51·
,, of pulmonic,	42·	42·	45·	37·7	39·	34·	39·	39·	42·
,, of left auriculo-ventricular,	45·	48·	48·	44·2	45·	42.	45·5	45·	45·
,, of aortic,	34·	33·	36·	34·5	39·	30.	34·	33·	36·

The cases of phthisis in the table are three in number, the ages 20, 24, and 66; the weights of the heart 7 oz., 9 oz. 5 dr., and 10 oz. 11 dr.
The cases of bronchitis are four, the ages 25 to 60, mean 47, and the weights of the heart 9 oz. to 11 oz., mean 10 oz. 2·7 dr.
The other cases, unconnected with pulmonary disease, are eight in number; the ages 20 to 64, the mean age 35·3, and the weights of the heart 7 oz. to 10 oz. 10 dr., the mean weight 8 oz. 4 dr.
The cases of chronic disease only have not been separated in this table, as they are only two in number.

PART III.—*Dimensions of the Healthy Heart.*

INFERENCES.

1. In males, the average circumference of the healthy heart, in persons of adult age, dying of diseases unconnected with any pulmonary affection, measured externally, was 103·1 lines, and the extreme circumference in different cases was from 87 to 120 lines.

The girth of the right ventricle averaged 55·6 lines, that of the left ventricle 47·5 lines; but these dimensions varied greatly in different cases, the girth of the right ventricle having a range of from 48 to 60 lines, that of the left ventricle of from 39 to 60 lines.

The mean length of the cavity of the right ventricle was 42·5 lines, that of the left ventricle 37 lines. These measurements also varied—the length of the right ventricle ranging from 34 to 51 lines, that of the left ventricle from 32 to 44 lines.

The parieties of the right ventricle had an average thickness at about the middle of its anterior wall of 1·93 lines; and decreased slightly in width towards the pulmonic orifice, and about half a line towards the apex, having thus a thickness of 1·81 lines in the former situation, and of 1·37 at the apex.

The walls of the left ventricle had a medium width at about the mid-point of 5·95 lines, at the base of 5·1 lines, and at the apex of 2·5 lines. The thickness of the parieties of the ventricles exceeded or fell short of these dimensions, without passing the limit of health, in the right ventricle by half a line, and in the left by about a line and a half. The thickness of the right ventricle ranged from 1·5 to 2·5 lines; that of the left ventricle from 5 to 8 lines.

The septum of the ventricles had an average thickness at the middle of 5·77 lines, and its width ranged from 3·75 to 7 lines, generally corresponding in thickness with the parieties of the left ventricle.

The right auriculo-ventricular aperture had a mean circumference of 54·4 lines; the pulmonic orifice of 40 lines; the left auriculo-ventricular aperture of 44·3 lines, and the aortic orifice of 35·5 lines; but the dimensions of the orifices also varied greatly in different cases, the right auriculo-ventricular aperture having a range of from 45 to 60 lines, the pulmonic of from 34 to 45; the left auriculo-ventricular aperture of from 38 to 51, and the aortic orifice of from 28 to 48.

2. In females the dimensions of the heart are ordinarily less than in males, but the difference is rather apparent in the diminished thickness of the walls of the ventricles and in the circumference of the orifices, than in the capacity of the cavities.

In adult females the circumference of the heart in persons dying of various affections, unconnected with disease in the lungs, had a

mean of 107·8 lines, and in different cases it attained extremes of 87 to 120 lines.

The girth of the right ventricle averaged 59 lines, and varied from 49 to 72 lines in different cases. The girth of the left ventricle averaged 48·8 lines, and ranged from 38 to 60 lines.

The medium length of the cavity of the right ventricle was 43·7 lines, and it ranged from 34 to 48 lines. The left ventricle had a mean length of 37·5 lines, and extremes of 32 and 44 lines.

The thickness of the walls of the right ventricle was near the base 1·81 lines, at the midpoint 1·87 lines, and near the apex 1·18 lines; and the greatest thickness varied in different cases from 1·5 to 2·5 lines. The parieties of the left ventricle had a medium width near the base of 4·9 lines, at the midpoint of 5·8 lines, and at the apex of 2·7 lines, the greatest width ranging in different cases from 5 to 8 lines.

The septum averaged 4·7 lines in thickness, and its width in various cases was from 4 to 6 lines.

The right auriculo-ventricular aperture had an average circumference of 52·1 lines, the pulmonic orifice of 39 lines, the left auriculo-ventricular aperture of 45·5 lines, and the aortic orifice of 34 lines; and the variations in the capacity of the different orifices, was, in the right auriculo-ventricular aperture from 42 to 57 lines, in the pulmonic from 33 to 42, in the left auriculo-ventricular from 37 to 54, and in the aortic orifice from 29 to 36. It will thus be seen that while in the two sexes the dimensions of the whole heart, and the size of the cavities did not differ materially, in females the walls were somewhat thinner, and the orifices less capacious than in males. The comparison is not, however, a just one, for, while a larger proportion of the males the dimensions of whose hearts are given, died of chronic diseases,—the females, with two exceptions, all died after short periods of illness. Had the respective proportions of cases of acute and chronic disease in the two sexes been similar, the dimensions of the heart would most probably have shown a greater difference. It would, however, appear that in the female heart the cavities are somewhat larger, relative to the thickness of the walls, than in males.

3. From these observations it thus appears that the girth of the right ventricle, measured externally, exceeded that of the left, in males by 1-6th, and in females by 1-5th. The length of the cavity of the right ventricle exceeded that of the left ventricle, in males by about 1-7th, and in females by about 1-6th. In both sexes the thickness of the walls of the right ventricle is about 1-3d that of the parieties of the left ventricle. The thickness of the septum is intermediate between that of the external walls of the right and left ventricles.

In males the pulmonic aperture was about 1-8th more in circumference than the aortic; the left auriculo-ventricular orifice about 1-4th more than the aortic; and the right auriculo-ventricular opening one-half larger. In females, the difference between the circumference of the aortic and of the other orifices, is somewhat greater.

4. In the two sexes, the length of the heart measured from the base to the apex was greater than its breadth measured across the broadest part. These dimensions were taken in comparatively few cases: in these the mean length of the organ was 49 lines, and the mean breadth 43 lines.

The walls of the right auricle measured at the middle of the sinus had a medium thickness of half a line to one line, but from the arrangement of the *musculi pectinati*, it is not easy to estimate their width. The walls of the left auricle had an average thickness of 1 to 1½ lines.

The right coronary artery in the cases in which it was measured had a circumference of from 5 to 8 lines; ordinarily this artery gives off the anterior branch immediately after its origin, but not unfrequently that vessel arises separately from the right sinus of Valsalva. The left coronary artery is generally larger than the right. In one case I have seen the two coronary arteries arise by a common trunk, and in another both arteries arose from the same sinus. The orifice of the coronary vein has generally a circumference of about 10 lines.

The fossa of the foramen ovale has a mean size of 8·6 lines in its longest, and 6·8 lines in its shortest diameter, but the dimensions vary considerably. The fold on the left side ordinarily overlaps the isthmus by 2 or 3 lines, and, when a valvular opening remains, it is usually 2 to 3 lines wide.

5. The heart is ordinarily greater in persons who have died of bronchitis and other pulmonary diseases, phthisis excepted, than in persons who have died of affections in which there was no obstruction in the lungs. The enlargement generally consists in hypertrophy and dilatation of both the right ventricle and left ventricles;—the cavity of the right ventricle being enlarged, and its walls increased in thickness, while the left ventricle, though increased in size, retains the usual thickness of its walls.

The circumference of the heart in cases of bronchitis in males, averaged 118·6 lines, and ranged from 110 to 130 lines.

The girth of the right ventricle averaged 67·3 lines, and ranged from 60 to 76 lines; that of the left ventricle averaged 51·3 lines, and ranged from 50 to 54 lines.

The mean length of the cavity of the right ventricle was 50 lines, and it ranged from 48 to 51 lines; that of the left ventricle had a medium of 40·6 lines, and extremes of 36 and 45.

The walls of the right ventricle were on the average 3 lines in width, and ranged from 2 to 4·5 lines, and in one case not included in the tables, of chronic bronchitis,—with curvation of the spine,—in a young man 17 years old, the parieties of the ventricle were fully 5 lines thick. The walls of the left ventricle had an average thickness of 5·8 lines, and ranged from 5·5 to 6 lines at the broadest part. The septum had a medium thickness of 5·08 lines, and a range of from 4 to 6 lines.

The right auriculo-ventricular aperture had a medium circumfer-

ence of 58·6 lines, and extremes of 54 to 62 lines. The pulmonic orifice a medium of 46 lines, and extremes of 45 and 48. The left auriculo-ventricular aperture, in the three cases measured, was 54 lines, and the aortic had a medium of 39 lines, and extremes of 36 and 42 lines. It will thus be seen, that the girth of the right ventricle was $\frac{1}{7}$ greater than that of the left, and the length of the cavity of the right ventricle 1-4th greater than that of the left. The capacity of all the orifices was above the average, and the pulmonic exceeded the aortic aperture by 1-6th, and the mitral exceeded the aortic by 1-3d, while the tricuspid orifice was, as usual, one-half larger than the aortic. A comparison of the dimensions of the heart in females in the cases of bronchitis, with those of the healthy organ, show similar results, but in only one case did the walls of the right ventricle attain a thickness of 4 lines, and in this instance there was chronic bronchitis with curvature of the spine, and the subject was 25 years of age.

The fossa of the foramen ovale in cases of chronic bronchitis, becomes considerably enlarged, and the valve is not unfrequently dilated and protruded into the left auricle. In one case, of which I have the preparation, a large sac is thus produced. When the fossa is dilated, the portion of the fold which ordinarily overlaps the isthmus is frequently drawn down, so that it may be found scarcely to reach above the edge of the opening; and when the valve has not become adherent the aperture might so be reopened; but, though I have seen several cases of very enlarged fossa with an unadherent valve, I have never met with one in which the fœtal aperture was restored.

Though the disproportion between the size of the right and left ventricles of the heart, and especially the greater size of the pulmonic than of the aortic aperture, exists at all ages, except in very early life, or before birth, and the experiments of Legallois have shown that is not dependent on the mode in which death takes place, it is certainly much aggravated in all cases in which there exists any obstruction to the transmission of the blood through the lungs. This is most marked in cases in which the obstruction has been of long duration, but I have seen the orifice of the pulmonary artery very considerably expanded in persons who have died of acute bronchitis and pneumonia, after short periods of illness.

6. In persons who have died of phthisis, the heart is usually smaller than in those who have sank from other diseases; but the decrease in size is less than obtains in some other forms of disease, and is chiefly due to the diminution in the size of the cavities and in the capacity of the orifices, while both the right and left ventricles are thicker than in other chronic cases where there is no pulmonary disease.

The average circumference of the heart in males who died of phthisis was 94·5 lines, and the extremes 92 and 97.

The girth of the right ventricle averaged 49·5 lines, and its extremes were 49 and 50; that of the left ventricle had a mean of 45 lines, and extremes of 42 and 48.

The length of the right ventricle had an average of 43·3 lines, and extremes of 36 and 48; that of the left ventricle averaged 37·3, and its extremes were 36 and 39.

The walls of the right ventricle had a mean thickness of 2 lines; those of the left ventricle, a mean of 6·3 lines, and extremes of 5 and 7 lines.

The right auriculo-ventricular aperture had a medium circumference of 49·3 lines, and extremes of 43 and 51. The pulmonic orifice a mean of 38·6 lines, and extremes of 29 and 48. The left auriculo-ventricular aperture a medium of 45·3, and extremes of 37 and 51; and the aortic aperture a mean of 34·6, and extremes of 26 and 42. It will thus be seen, that while the ventricular walls retain their usual thickness, or exceed it, the length of the cavities, and the capacity of the orifices are much less than usual, and the difference is the more apparent when the dimensions of the heart in phthisis are compared with those of the organ in other chronic affections, unconnected with disease in the lungs. It will also be observed that the proportion between the cavities and orifices on the two sides of the heart is not materially different from that which obtains in the healthy organ; the right side being, however, somewhat less than natural, relatively, to the left. As some of the results here deduced differ from those of M. Bizot, who states the heart in phthisis to be smaller in all its dimensions, it is right to state that the data here analysed are much fewer than those collected by that observer.

7. The observations are too few to warrant definite conclusions as to the different dimensions of the heart in persons who have died of acute and chronic diseases, but they would appear to indicate that the chief difference consists in the diminished thickness of the walls of the left ventricle.

It will be observed, from the annexed statements (Table XII.), that though the series of observations collected by M. Bizot, Dr Reid, Dr Ranking, and myself, correspond to a considerable extent, there are differences between them. M. Bizot's measurements of the width of the walls are generally the least, those of Dr Reid the largest. In the dimensions of the orifices, Dr Ranking's observations show the smallest size, and Dr Reid's the largest. The seat at which M. Bizot took the dimensions of the aortic and pulmonic orifices differed from that selected by Dr Ranking, the former having measured the opening at the level of the free border of the valves, while the latter took the dimensions on the line of their insertions; the precise point measured by Dr Reid is not mentioned. All three observers estimated the circumference of the orifices after they had been laid open, whereas my own measurements were made while the apertures were entire, by graduated balls passed through them, and therefore indicate their absolute capacity. After division the fibrous ring of the arterial orifices,

TABLE XII.—For the purpose of comparison I have prepared the following table, which exhibits the mean dimensions of the *healthy heart* as deduced by M. Bizot, Dr Reid, and Dr Ranking. I have not thought it necessary to include M. Bouillaud's observations, for they are few in number, include persons of different ages and of both sexes, and are very imperfect as to the measurements in each case.

	Bizot.[1]		Dr Reid.[2]		Dr Ranking.[3]		Own Cases.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of Heart,					113·	99·5	102·7	104·9
Length of cavity of right ventricle,	37·2	34·					43·3	44·
,, ,, left ,,	34·39	31·03					37·6	37·1
Thickness of walls of right ,, base,	1·9	1·6					1·85	1·86
,, ,, ,, ,, midpoint,	1·4	1·29	2	1·75	1·3	1·5	1·98	2·0
,, ,, ,, ,, apex,	1·03	·93					1·42	1·3
,, ,, left ,, base,	4·5	4·3					5·15	4·9
,, ,, ,, ,, midpoint,	5·15	4·3	5·6	4·65	6·25	5·5	6·	6·
,, ,, ,, ,, apex,	3·7	3·4					2·4	2·5
Thickness of septum, ,, ,,	4·9	4·4	5·6	4·25	5·2	4·5	5·73	4·7
Circum. of right auriculo-ventricular aperture,	54·19	48·25	59·7	53·	52·75	47·	53·9	51·
,, pulmonic,	32·34	30·1	42·	39·3	30·3	28·1	40·	39·3
,, left auriculo-ventricular aperture,	45·5	41·33	52·	47·3	44·25	38·75	45·2	45·
,, aortic,	31·24	28·8	36·	34·	29·5	27·5	35·6	34·

The measurements of M. Bizot are calculated from the examination of the hearts of 60 males and females, from 16 to 79 years of age. The thickness of the septum refers only to persons between 30 and 50 years of age.—The observations of Dr Reid are based upon the measurement of the hearts of 16 males and 9 females, adults, weighing from 7 to 13½ oz.—Dr Ranking's conclusions are drawn from the weight of the heart in 15 males from 26 to 65 years of age, and 17 females from 18 to 62 years of age.—My own observations include the hearts of 16 males and 13 females, dying of various different forms of disease in persons from 20 to 66 years of age, in whom the hearts weighed from 6 oz. 9½ dr. to 12 oz.—Dr Reid's and Dr Ranking's observations are given in the original papers in fractional parts of an English inch; I have here reduced them to French lines for facility of comparison.

[1] Mem. de la Soc. Med. d'Observat., t. i., p. 262, Paris, 1837. Anatomical, and Pathological Researches, Ed. 1840, p. 573.

[2] L. & E. Monthly Journal, 1842; and Physiological,

[3] London Medical Gazette, vol. xxix.; vol. i. 1841–42, p. 903.

TABLE XII. B.—Showing the mean dimensions of the heart according to the observations of M. Bizot, Dr Reid, Dr Ranking, and myself, in fractional parts of English inches.

	Bizot.		Dr Reid.		Dr Ranking.		Own Cases.	
	Males.	Females.	Male	Females.	Males.	Females.	Males.	Females.
	inch.	inch.	inch.	inch.	inch.	inch.	inch.	inch.
Circumference of the heart,					9¼	8¼	9⅛	9⅛
Girth of the right ventricle,							4⅜	5⅛
,, left ,,							4⅛	4⅛
Length of cavity of right ventricle,	3⅜	3·					3⅜	3⅜
,, ,, left ,,	3⅛	2⅞					3⅛	3⅛
Thickness of walls of right ventricle,	⅛	⅛	⅛	⅛	⅛	⅛	⅛	⅛
,, ,, left ,,	⅛	⅛	⅛	⅛	⅛	⅛	⅛	⅛
,, septum,	⅛	⅛	⅛	⅛	⅛	⅛	⅛	⅛
Circumference of right auriculo-ventricular aperture,	4⅞	4⅜	5⅜	4⅞	4¾	4⅜	4⅞	4⅝
,, pulmonic aperture,	2⅞	2⅝	3⅛	3⅛	2⅝	2⅜	3⅝	3⅝
,, left auriculo-ventricular aperture,	4⅛	3¾	4⅝	4⅛	3¾	3½	4⅛	4·
,, aortic aperture,	2¾	2⅝	3⅛	3·	2⅝	2⅜	3⅛	3·

According to M. Bouillaud the mean circumference of the healthy heart in 10 males, from 16 to 38 years of age, is 105·37 lines; in 6 cases (4 males and 2 females) from 17½ to 45 years of age, in which he regards the organ as atrophied, 81·5 lines. Thickness of the walls of the left ventricle, 6½ to 7 lines, and 6½ lines; of the right ventricle, 2½ and 2½ths; of the septum, 7 lines; right auricle 1 line; left auricle, 1½ line; right auriculo-ventricular aperture, 46 and 47 lines; pulmonic aperture,[1] 31¾ and 28 lines; left auriculo-ventricular aperture, 42 and 40; aortic, 29½ and 28 lines.

Lobstein states the weight of the heart to be from 9 to 10 oz., and the dimensions as follows:—Length, 5 inches and 6 lines; breadth at base, 3 inches; thickness of the walls of the right ventricle, 2¼ lines; of left ventricle, 7 lines; the right auricle, 1 line; the left auricle, half a line (French measures).

[1] Traité d'Anatomie Pathologique, tome ii. Paris, 1833, pp. 419 and 449.

TABLE XIII.—Showing the dimensions of the heart in males in cases of hypertrophy, unconnected with marked obstruction, in cases of obstruction at the aortic orifice or in the aorta, and in cases of incompetency of the aortic valves.

	Hypertrophy.			Obstruction at Aortic Orifice or in Aorta.			Incompetency of Aortic Valves.		
	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of heart,	129·	104·	182	119·6	102·	138	124·3	104·	171
Girth of right ventricle,	66·5	54	96	61·3	54	72	60·8	44	90
„ left ventricle,	62·5	50	86	58·3	48	66	63·5	60	81
Length of cavity of right ventricle,	51·2	48	62	43·	42	48	55·7	38	72
„ „ left ventricle,	45·7	40	51	38·8	36	42	45·4	38	60
Thickness of walls of right ventricle base,	2·2	2	3	2·3	3	2	2·32	2	2·5
„ „ „ „ midpoint	2·0	1·5	2·5	2·7	3	3	3·25	3	5·
„ „ „ „ apex,	1·7	1·	2·	1·5	2	1·5	1·82	1	2·
„ „ left ventricle base,	5·7	5·	8	8·1	6	10	6·6	7	10
„ „ „ „ midpoint	8·5	7·	11	8·06	8	11	6·7	7	5·0
„ „ „ „ apex,	2·8	2·	3·5	2·47	3	2·5	2·35	2·5	1
Thickness of septum,	8·2	7·	10	6·6		8	7·5	9	
Circumference of right auriculo-ventricular aperture,	55·7	42	63	57·7	60	60	57·4	48	60
Circumference of pulmonic aperture,	45·	35	54	44·2	42	42	45·4	39	51
„ of left auriculo-ventricular aperture,	51·2	39	60	49·8	48	51	53·5	42	60
Circumference of aortic aperture,	39·2	34	42	37·7	39	36	39·	36	45

The cases of hypertrophy analysed in this table are four, in persons from 44 to 65 years, and with a mean age of 51 years. The hearts in the cases, weighed from 12 oz. 12 dr. to 40 oz. 12 dr., the mean weight being 21 oz. 4 dr.

The cases of obstruction are eight in number, in persons from 24 to 78 years of age, and with an average of 53·7. The weights of the heart range from 10 oz. 12 dr. to 21 oz., the mean being 15 oz. 2 dr.

The cases of incompetency are seven, the ages of the subjects from 32 to 57, the weights of the heart from 11 oz. 8 dr. to 34 oz., and the mean 19 oz. 3½ dr.

TABLE XIV.—Showing the dimensions of the heart in females, in cases of obstruction at the aortic orifice, of incompetency of the aortic valves, of mitral valvular disease, and of combined mitral and aortic valvular disease.

	Obstruction at Aortic Orifice.			Incompetency of Aortic Valves.	Mitral Valvular Disease.			Combined Mitral and Aortic Disease.		
	Mean.	Lightest.	Heaviest.		Mean.	Lightest.	Heaviest.	Mean.	Lightest.	Heaviest.
	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
Circumference of heart,	105·6	104·	120·		111·8	108·	127·	118·	126·	110·
Girth of right ventricle,	56·	60·	60·		62·8	60·	75·	63·5	72·	55·
„ left ventricle,	49·6	44·	60·		49·	48·	52·	54·5	54·	55·
Length of cavity of right ventricle,	42·3	39·	48·	52·	46·	42·	51·			43·
„ „ left ventricle,	37·	39·	42·	52·	39·	36·	42·			42·
Thick. of the walls of the right ventricle base,	2·16	25·	2·	2·	2·2	2·	2·	2·6	3·	2·
„ „ „ midpoint,	2·3	3·	2·	2·	2·2	2·	3·	2·6	2·	2·
„ „ „ apex,	2·	2·	2	1·5	1·3	1·	1·5	2·1	1·	1·5
„ „ left ventricle base,	6·	5·	5·	6·	5·	4·	5·	7·3	6·	7·
„ „ „ midpoint,	7·6	8·	7·	7·	5·4	5·	5·	9·	6·	10·
„ „ „ apex,	2·3	3·	2·5	3·	2·2	2·	2·	2·6	3·	2·
Thickness of septum,	5·5	6·	5·		5·	4·	5·		5·	
Circum. of right auriculo-ventricular aperture,	57·	54·	60·		51·2	57·	54·	48·	42·	51·
„ of pulmonic aperture,	36·5	34·	39·	39·	34·6	39·	39·	35·6	36·	32·
„ of left auriculo-ventricular aperture,	43·5	42·	45·		23·	39·	12·	34·3	42·	48·
„ of aortic aperture,	32 5	32·	33·	29·	29·	30·	33·	30·6	29·	35·

The cases of aortic valvular obstruction are only three in number, the subjects were 75, 75, and 60 years of age, and the hearts weighed 10 oz. 2 dr., 10 oz. 12 dr., and 14 oz. respectively.

In the single case of incompetency of the aortic valves, the subject was 46 years of age, and the heart weighed 16 oz. The cases of mitral valvular disease are five in number, the ages of the subjects ranged from 23 to 63, and the mean age was 39·4, the hearts weighed from 8 oz., 8 dr. to 15 oz. 12 dr., the mean weight being 11 oz. 13½ dr. The cases of combined mitral and aortic valvular disease are three in number, the ages from 18 to 63, the mean being 40 years, the weights of the heart ranged from 13 oz. 8 dr. to 22 oz., and the mean weight was 19 oz. 2¾ dr. The dimensions of two cases of combined disease in females, 11 and 12 years of age, included in the general table, are excluded from this table.

especially that of the pulmonary artery, contracts, and it becomes extremely difficult, or impossible, to judge of the size, which should be assigned to the opening. The mode in which the length of the cavity of the right ventricle is estimated by M. Bizot and myself, also differs; his measurements refer to the length of a line from the base to the apex; mine, to that of a line following the course of the ventricle from the orifice of the pulmonary artery to the apex. It will be seen that the relative size of the heart in males and females in my own table differs but little, much less than is the case in the other observations. This is explained, as before mentioned, by the female hearts measured being, almost all of them, those of persons who had died of acute diseases, while the majority of the male hearts were from cases of chronic disease.

Part IV.—*Dimensions of the Diseased Heart.*

INFERENCES.

1st. The heart attained the greatest increase of size in the cases in which the hypertrophy and dilatation were unconnected with any marked disease of the valves or aorta, and the enlargement in these instances was not confined to the left cavities, but involved, though to a less degree, those of the right side also.

2d. In the cases of obstructive disease of the aortic valves, or of the aorta, the heart was also very greatly enlarged, and the enlargement involved both the left and right cavities of the organ, but was not so great as in the cases of hypertrophy unconnected with valvular or aortic disease. The increased size was also by no means proportionate to the amount of impediment to the circulation, being in some cases very great where the obstruction was only trivial, and in others slight where very great obstruction existed.

3d. In the cases of incompetency of the aortic valves, the capacity of the left ventricle was greater than either in the cases of simple hypertrophy or of obstructive disease, but the walls of the ventricle were usually less increased in thickness than in those diseases. The hypertrophy involved both sides of the heart, but the right ventricle attained a greater thickness in this, than in either of the other forms of disease.

It will be seen from the table of dimensions of the diseased heart in males, that the circumference of the organ averaged in the cases of hypertrophy, obstructive disease, and incompetency, respectively, 129, 119, and 124 lines, and attained the extremes of 182 lines, or 15 French inches, or 16 English; 138 lines, or 11½ French inches, or about 12 inches English; and 171 lines, or 14¼ French inches, or about 15 English. The girth of the right ventricle measured externally averaged, in cases of hypertrophy, 66 lines, of obstructive disease 61 lines, and of incompetency 60 lines, and attained the extremes of 96, 72 and 90 lines. The girth of the left ventricle averaged in

three several forms of disease 62, 58 and 63 lines, and attained the extremes of 86, 66 and 81 lines.

The length of the cavity of the right ventricle averaged in the three several forms of disease 51, 43 and 55 lines, and attained extremes of 62, 48 and 72 lines; the length of the left ventricle averaged 45, 38 and 45 lines, and the extreme lengths were 51, 42 and 60 lines. The walls of the right ventricle, measured at the thickest part, were 2·2 lines in width, in cases of hypertrophy, 2·7 lines in cases of obstructive disease, and 3·25 in cases of incompetency; and their minimum and maximum thickness in these several forms of disease were 2 and 3 lines, 3 lines, and 3 and 5 lines. The walls of the left ventricle averaged in width in cases of hypertrophy 8·7 lines, in obstructive disease 8·1 lines, and in incompetency 6·7 lines, and the extremes were respectively 7 and 11 lines, 6 and 11 lines, and 6 and 10 lines.

The septum had an average thickness in the cases of hypertrophy of 8 lines, and its extremes were 5 and 10 lines: in the cases of obstruction of 6·6 lines, and its extremes 6 and 8; and in the cases of incompetency, in the only two cases in which it was measured, it was 6 and 9 lines thick.

The capacity of the orifices in these diseases varied according to the form of the affection. In the cases of hypertrophy they were all above the healthy standard. The aortic orifice averaged 39 lines in circumference, and its extreme dimensions were 34 and 42 lines. The left auriculo-ventricular aperture averaged 51 lines, and its extremes were 39 and 60 lines. The pulmonic aperture averaged 45 lines, and ranged from 35 to 54 lines, and the right auriculo-ventricular aperture averaged 55 lines, and ranged from 42 to 63 lines.

In the instances of obstructive disease the aortic orifice was in some cases greater, in others less, in circumference than natural. Its average capacity was 37 lines, and its extreme dimensions 33 and 42 lines, and all the other apertures exceeded the natural size. The mitral orifice averaged 49, and ranged from 48 to 54 lines, the pulmonic averaged 44 lines, and ranged from 42 to 48, and the tricuspid averaged 57, and ranged from 54 to 60 lines.

In cases of incompetency of the valves the capacity of the aortic orifice also varied with the nature of the disease, in some instances the valves being healthy, but incapable of closing the aperture from the amount of dilatation; while in others the capacity of the aperture was not greater than natural, but the valves being diseased, were incapable of closing it. The mean circumference of the orifice in these cases was 39 lines, and it ranged from 30 to 45 lines; but in the case in which the capacity of the orifice was only 30 lines, the incompetency was the result of laceration of the valves, from violent muscular exertion, and as there was no reason to suspect the existence of any disease of the heart before the occurrence of the accident, it is probable that the aperture had contracted, so as

to afford some compensation for the imperfection of the valves. In these cases the capacity of all the other orifices was above the healthy standard, the mitral had a mean circumference of 53 lines, and its extreme dimensions were 42 and 60 lines, the pulmonic a mean of 45, and extremes of 59 and 51 lines, and the tricuspid a mean of 57, and extremes of 48 and 60 lines.

The auricles were greatly enlarged in all three forms of disease, and their walls generally increased in thickness; the right auricle being from $1\frac{1}{2}$ and to 2 lines thick, the left from $1\frac{1}{2}$ to 2 or $2\frac{1}{2}$ lines. The coronary arteries, when measured, were also found above their natural size.

Though the walls of the left ventricle did not exceed 11 lines or about one English inch in thickness, in any of the cases included in the table; in another instance, in a man 74 years, in which there was some valvular thickening with dilatation of the aorta, the organ attained the weight of 19 oz. 8 dr., and the left ventricle near the base measured 14 lines, or nearly an inch and a quarter English. The cases included in the table, also, do not afford evidences of the extent to which the aortic office may be contracted; in one case, that of a female 75 years of age, which has before been several times alluded to, the aortic aperture was a mere slit 10 lines long, and the thickened and ossified valves did not admit of being separated for more than 3 or 4 lines; yet, though the disease probably originated in a congenital malformation of the valves, and had therefore very slowly advanced, the parieties of the left ventricle were only 8 lines in thickness, and those of the right 2 lines. In a case of obstructive and regurgitant disease of the aortic valves, not included in the tables, the septum had a width of 8·7 lines or $\frac{3}{4}$ of an English inch.

The shape of the heart varies considerably in these different forms of disease, according to the seat and extent of the hypertrophy and dilatation. In cases of obstructive disease, whether the impediment be seated in the aortic valves or in the ascending portion of the aorta, the cavity of the left ventricle becomes peculiarly elongated, and the walls are ordinarily thicker near the base than elsewhere, and hence the whole organ has an acutely triangular form, and is much longer in its longitudinal, than in its transverse diameter. On the other hand, in cases of regurgitation through the aortic valves, while the left ventricle is elongated, the thickening of the parieties is more equably distributed throughout the cavity, and the apex, instead of being pointed, is obtuse, and thus the heart assumes a more oblong form, of which, however, the longest diameter is the longitudinal. In cases of regurgitation through the mitral aperture, and where there is considerable disease both of the mitral and aortic valves, the left ventricle, instead of being elongated, is expanded laterally, and especially at the apex, so that the organ has a more obtusely triangular form, and is generally broader from side to side than longitudinally. In these forms of disease, the right ventricle though implicated to a greater or less degree, is not so much so as

to affect the shape of the heart generally, but in cases of obstructive disease of the mitral valve, the left ventricle, as will be shown more particularly below, is little, if at all, altered in dimensions, while the right ventricle is hypertrophied and very greatly dilated; and hence the organ, though not very greatly larger than natural, is much broader, and is very obtuse at the apex. These differences of form are so striking and peculiar, that in many cases of diseased heart, it is easy to predicate before the cavities are laid open, the nature of the affection which will be found.

4. In cases of contraction of the left auriculo-ventricular aperture, uncomplicated with disease of the other orifices, the heart does not attain so great an increase of size as in the forms of disease before mentioned, for the enlargement is chiefly limited to the right cavities. Indeed, in some cases, more especially in young subjects, the capacity of the left ventricle, the thickness of its walls, and the circumference of the aortic orifice, are found not at all to exceed the natural dimensions, or even to fall below the healthy standard.

It will be observed, that the comparison between the cases of mitral valvular disease and of obstructive disease, in females, would lead to the conclusion, that the heart in the latter disease is smaller than in the former; but this is doubtless an erroneous inference, founded upon the smallness of the number of cases of obstructive disease in females. A more correct impression is most probably to be gained by comparing the dimensions of the heart in cases of mitral valvular disease in females, with its size in the other form of disease in males;—for though the heart does not in any disease attain so great a size in females as in males, the general effect of different forms of disease on its nutrition, may be inferred to be similar in the two sexes. The average circumference of the heart in the cases of mitral valvular disease, given in the table, will be seen to have been only 111 lines, and in no case did the circumference exceed 127 lines or $10\frac{1}{2}$ French, or upwards of 11 English inches. The girth of the right ventricle averaged 62 lines, and in one case amounted to 75 lines. The girth of the left ventricle was very much less than that of the right, or only 49 lines on the average, and in no case more than 52 lines. The length of the right ventricle was on the average 46 lines, and its greatest length was 51 lines. The average length of the left ventricle was 39, and the extreme length 42 lines. The walls of the right ventricle averaged 2·2 lines in thickness, and ranged from 2 to 3 lines; those of the left ventricle had an average thickness of 5 lines, and ranged from 4 to 6 lines.

The mitral aperture varied considerably in the amount of contraction. In two cases, one of which is not included in the tables, it only admitted a cylinder 11 lines in circumference; in a third instance its circumference was only 18 lines, and in three others it was 22, 24, and 39 lines, the mean being 23 lines. The aortic orifice was

also below the natural size, or on the average only 29 lines in circumference, and its dimensions ranged from 25 to 33 lines.

The orifices on the right side of the heart were, on the contrary, ordinarily larger in comparison. The pulmonic aperture averaged 34½ lines in circumference, and ranged from 29 to 39 lines; the tricuspid averaged 51 lines, and ranged from 40 to 57 lines. The left auricle was in all the cases greatly dilated, and its walls varied in thickness from 1½ to 2 lines; the right auricle was generally still more enlarged, and its walls had a width of from 1 to 1½ lines.

It will thus be seen that, while the left ventricle and aortic orifice were below the healthy standard, the dimensions of the other parts of the organ exceeded the usual size, so that it may be inferred that the operation of this form of disease is either to cause atrophy of the left ventricle, or, when the disease commences in early life, to prevent the full development of that cavity. It will also be observed that the hypertrophy of the right ventricle did not, in any cases of mitral valvular disease, equal that which was found in the two cases of chronic bronchitis with curvature of the spine.

5. The tables contain only one case of regurgitation through the left auriculo-ventricular aperture, from dilatation of the orifice without disease of the valves; and in this the subject was a child eight years of age, so that it cannot be compared with the other cases; allowance, however, being made for the age of the patient, the heart was much more enlarged than in the cases of mitral valvular contraction, and the left ventricle also was increased in size.

6. In the cases in which both the aortic and mitral valves were diseased, the dimensions of the heart were intermediate between those in cases of uncomplicated mitral and aortic disease. The enlargement, instead of being chiefly limited to the right ventricle, involved the left also; and the length of the cavities and the thickness of the walls, were greater than in cases of mitral disease alone, but less than in cases of aortic disease.

The girth of the right ventricle in two of the cases in the table was 72 and 55 lines; of the left ventricle 54 and 55 lines. The mean thickness of the parieties of the right ventricle was 2·6 lines, and the extremes 2 lines and 4 lines; the mean width of the walls of the left ventricle was 9 lines, and the extremes 6 and 11 lines. In the only case in which the width of the septum was measured, it was 5 lines. In these instances the subjects were females, but I have notes of other cases of combined aortic and mitral disease in males. The subject of one of these, 36 years of age, and the heart weighed 17 oz. 8 dr., the dimensions are not given in the tables. The aortic valves were incompetent, and the mitral aperture so contracted as only to admit the handle of a scalpel, indicating a circumference of about 12 lines. The cavity of the right ventricle was 42 lines long, the left 37 lines. The walls of the right ventricle had a width of from 4½ to 5¾ lines, and the left ventricle of 5¾ at the base

and midpoint, and 3½ near the apex. The longitudinal diameter of the heart was 42 lines, the transverse 54 lines. The walls of the right auricle averaged 1½ lines in thickness, those of the left auricle 2 lines. In a second case the subject was a boy 18 years of age, and the disease was the sequence of rheumatism four years before. The organ weighed 16 oz. The pericardium was adherent, and both the aortic and mitral valves were thickened and incompetent. The circumference of the heart measured 122 lines. The cavity of the right ventricle was 27 lines in length, the left 48 lines. The walls of the right ventricle measured 3 lines in thickness, those of the left 8 lines. The aortic orifice had a capacity of 42 lines, the mitral of 45, the pulmonic of 42, and the tricuspid of 54. The longitudinal diameter of the heart was 42 lines, the transverse 56 lines.

7. Only one case of combined obstruction of the right and left auriculo-ventricular apertures is included in the tables; and in this instance the most remarkable feature was the great degree of contraction which existed in both openings, the left auriculo-ventricular aperture having a circumference of only 18 lines, and the right of only 21 lines; yet with this very great amount of obstruction, which had probably commenced before birth, the walls of the right ventricle was only 1·5 lines in width, and those of the left ventricle 4 lines. The right auricle was very greatly dilated, and its walls were from ¼ a line to 2 lines in thickness; the right ventricle was somewhat dilated. The left auricle was also dilated, and the left ventricle, though to a less degree. The aortic orifice measured 30 lines in circumference, the pulmonic 35 lines. The aortic valves were also thickened and adherent. The subject of the case was a female 37 years of age.

8. The dimensions of the heart in cases of malformation vary with the kind of the deviation from the natural structure. The most frequent form of irregular development is that in which the pulmonary orifice is contracted, and the septum ventriculorum imperfect, or the foramen ovale open. Three cases of this description are included in the tables. The subjects were males, 15 and 20 years of age, and a female, 19 years old. In the first case the pulmonary artery was so contracted, as only to give passage to a ball 13 lines in circumference, in the second of 12 lines, and in the last of 8 lines. In the first and third cases the aorta arose from both ventricles, in the second the foramen ovale was open. In each of these cases the right ventricle was very greatly hypertrophied, measuring 54, 66, and 84 lines in circumference externally, while the left ventricle had only a girth of 42, 42, and 48 lines. The walls of the right ventricle measured 5½, 7, and 4 lines, those of the left ventricle 4½, 6, and 6 lines. It will thus be seen that, though the enlargement was chiefly on the right side, allowance being made for the age and sex of the subjects, the left ventricle must also have partaken of the hypertrophy; and this is still more obvious in cases of malformation in younger persons. The auricles were also

greatly dilated and hypertrophied, measuring from 1½ to 2 or 2½ lines.

In another instance in which malformation of a similar description was found in a child 6½ years old, the pulmonary orifice only admitted a cylinder 6½ lines in circumference; and in a fifth, in an infant 2 years and 5 months old, it was a mere slit 2 lines long. In these cases the walls of the right ventricle were much hypertrophied, measuring in each case, at the thickest point, 4 lines; while the left ventricle measured in the former case 6 lines, in the latter 3; and the density and resistance of the walls of the right ventricle, as in all the other cases of this kind of malformation, contrasted remarkably with the flaccidity of those of the left ventricle.

The septum is not, in these cases, hypertrophied in proportion to the walls of the right ventricle; indeed, that portion of the heart is more implicated in diseases of the left ventricle than in those of the right. Thus, while in cases of hypertrophy without valvular disease and in others of obstructive disease it had a thickness nearly equal to that of the outer walls of the left ventricle, or of from 9 to 10 and 11 lines;—in the cases of malformation in which the parieties of the right ventricle were from 6 to 7 lines thick, or fully three times the natural width, the septum measured only 6 lines in thickness, or was not materially wider than usual.

Different opinions have prevailed as to the cause of the hypertrophy of the right ventricle in cases of malformation. It has been supposed to be due to the entrance of aerated blood into the right cavities of the heart; but this cannot be the true explanation, for, in the most remarkable cases of hypertrophy, the course of the blood must necessarily be from the right ventricle or auricle into the left. The more correct theory is, doubtless, that which ascribes the hypertrophy to the effort to overcome the obstacle to the flow of blood from the ventricle, through the contracted pulmonary orifice. Another cause, which would have a similar effect, obtains in many cases, and appears to have been overlooked. I allude to the increased action of the right ventricle consequent upon the aorta arising in part from that cavity, and the share which it consequently takes in the maintenance of the systemic circulation. In one of the cases of malformation which I have mentioned, while the parieties of the infundibular portion of the right ventricle, upon which the stress from the contracted pulmonary orifice would chiefly fall, had only an extreme width of 4 lines; those of the sinus, which were chiefly concerned in propelling the blood from the right ventricle into the aorta, were 7 lines in thickness. It is true that the walls of the sinus are ordinarily thicker than those of the infundibular portion of the right ventricle, but the difference is ordinarily much less considerable.

It has been thought that the condition of the right ventricle, in some cases of malformation, affords an exception to the general rule of the non-occurrence of true concentric hypertrophy; and specimens, when first removed from the body, and preparations preserved in museums, not unfrequently present the appearance of increased thickening of the walls of the right ventricle, with diminution of its cavity. These appearances are, however, I believe, deceptive, and depend upon specimens being examined, or the preparations having been immersed in spirit, before the tonic contraction of the muscular fibres had subsided. I have seen cases of malformation in which, when the right ventricle was first laid open, there seemed to be absolutely no cavity, but in which, after maceration, the ventricle proved to be unusually large.

In cases in which the maintainence of the circulation is thrown upon the left ventricle, as when the right auriculo-ventricular aperture is obliterated, or when that opening or the pulmonary orifice is greatly contracted after the complete development of the septum of the ventricles, the left ventricle becomes much hypertrophied and dilated, while the right ventricle undergoes a proportionate decrease in size. In some of these cases, indeed, the right ventricle becomes reduced to a small hollow, about the size of a pea, which is surrounded by thick ventricular walls; but this condition is clearly one of atrophy, not of hypertrophy, and the defective nutrition of the muscular substance of the right ventricle from disuse, is shown by its unusual paleness and flaccidity, and by the looseness of its texture.

General Remarks on the Dimensions of the Diseased Heart.—The changes which the heart undergoes in disease have attracted the attention of most systematic writers, and especially of Laennec, Bertin, Lobstein, Cruveilhier, and Hope; and I may particularly refer to the series of observations published by Bouillaud, and to the incidental allusions to the measurements of the organ in cases of disease, contained in Dr Ranking's valuable paper. It may not be without interest to compare the observations of these writers with the results obtained from the analysis of the cases now published.

The circumference of the largest heart measured by M. Bouillaud was 12 French inches, and of that mentioned by Dr Ranking 12¾ English inches—dimensions which very nearly correspond. In my own tables the dimensions are given of the heart of a male in a case of hypertrophy without valvular disease, which measured 15 inches and 2 lines French, or somewhat above 16 English inches in circumference, and one of incompetency of the aortic valves, in which the circumference of the heart was 14 inches and 3 lines, or about 15 English inches. In females the extreme circumference of the heart was 10 inches and 7 lines, or about 11 English inches, in a case of mitral valvular disease, and 10 inches and 6 lines in one of combined aortic and mitral disease, and 10 inches in one of obstructive disease at the aortic orifice.[1]

[1] M. Bouillaud, for purposes of comparison, gives the weight and dimensions

The thickness of the walls of the right ventricle is stated by Laennec[1] rarely to exceed 4 or 5 lines; and in the observations of M. Bouillaud, no case is given in which it measured more than 4½ lines. Bertin,[2] however, describes a case in which there existed a congenital contraction of the orifice of the pulmonary artery from adhesion of the valves, with patency of the foramen ovale, in a female 57 years of age, and, in this instance, the cavity of the right ventricle was less than natural, and its walls from 16 to 11 lines thick. M. Burnet[3] relates a somewhat similar case of congenital disease, in which, however, the foramen ovale was closed, in a girl 7 years of age, in whom the walls of the right ventricle were nearly an inch in width, and the cavity almost obliterated; and Louis[4] has described a case of contraction of the pulmonary orifice with imperfection of the septum ventriculorum in a man 25 years of age, in whom the parieties of the right ventricle had a width of 8 to 10 lines. Hope, in cases of congenital obstruction of the pulmonic aperture with an open foramen ovale, in a girl 8 years of age, and of aneurism of the aorta with regurgitation through the aortic valves, in a man 25 years of age, found the walls of the right ventricle half an English inch, or 5¾ lines thick. Dr Ranking refers to a case in which the pulmonary artery was contracted and the foramen ovale unclosed; and to another, in which the aorta arose from both ventricles, with pulmonic valvular obstruction, in which the parieties of the right ventricle measured 17-48ths (about 4 lines), and 44-48ths (10 lines) of an English inch in thickness. In none of the observations now published were the walls of the right ventricle so extremely hypertrophied as in the cases last named. The greatest thickness of the parieties being 5 lines in a case of chronic bronchitis, with deformed spine, 5 lines in cases of obstructive disease, and of incompetency of the aortic valves, 5¾ lines in a case of combined mitral and aortic valvular disease, and 7 lines in cases of congenital obstruction at the pulmonic orifice, with a patent foramen ovale in one, and deficiency of the septum ventriculorum in the other. In the latter case the subject was a female, while in all the others they were males; with this exception, the walls of the right ventricle did not exceed 4 lines in thickness in females, and the instances in which they attained this width were cases of combined aortic and mitral valvular disease, and of chronic bronchitis.

The parieties of the left ventricle are stated by Laennec to have been seen by him an inch, or even 18 lines thick, or double or triple the size in the sound state. This statement is repeated by Elliotson,[5]

of the heart of an ox; the weight was 66 oz. 7 dr. avoird., and the circumference 18 French inches.

[1] Diseases of the Chest, 4th Ed. Forbes' Translation, 1834, p. 547.

[2] Maladies du Cœur. Paris, 1824, p. 326. Obs. 87. See also Bouillaud, Traité Clinique, 2me Ed., 1841, t. ii., p. 273. Obs. 126.

[3] Bouillaud, p. 281. Obs. 126; and Journal Hebd. de Med., 1831.

[4] Mém. on Recherches Anatomico-Pathologiques. Paris 1826, p. 313. Obs. 10. See also Arch. Gen. de Med., 2me serie, t. iii., 1823.

[5] Lumleyan Lectures.

and Hope mentions, that the walls of the left ventricle may attain a thickness of one, one and a half, or, according to some, of two inches; and he mentions a case in which they were 1½ inch thick in a case of regurgitation through the aortic orifice. The extreme thickness of the parieties of the left ventricle in the cases mentioned by Bouillaud, is 13 lines. Of the hearts examined by Ranking, in one the walls of the left ventricle attained a width of one inch, or about 11½ lines, and Bertin mentions a case in which they were upwards of one inch in thickness.

The greatest width of the parieties of the left ventricle in the cases given in the present memoir, was in males 11 lines, in cases of hypertrophy without valvular disease, and of aortic disease, and of dilatation of the aorta with chronic bronchitis; but another case is referred to, in which there was slight valvular disease with dilatation of the aorta, and the parieties of the left ventricle measured 14 lines in width. In females the greatest thickness of the walls of the left ventricle was 10 and 11 lines, in cases of combined aortic and mitral valvular disease.

The septum of the ventricles is mentioned by M. Bertin to have been found by him one inch in thickness; the greatest width in M. Bouillaud's observations is 10 lines, and Dr Ranking found it 38-48ths of an English inch (8·75 lines) in one case. In the present observations the greatest width in males is 10 and 11 lines in cases of hypertrophy without valvular disease, and 9 lines in cases of incompetency. In females the greatest width is 6 lines in a case of mitral valvular disease. I regret, that in the observations, the dimensions of the septum were less frequently obtained than would have been desirable.

The parieties of the right auricle were found by M. Bouillaud, in two cases, to measure 3 and 3½ lines in width, but his measurements are taken near the appendix, where ordinarily the walls are thicker than across the middle of the sinus. Bertin also examined a heart in which the walls of the right ventricle had a width of 3 lines; and Dr Hope speaks of a thickness of a quarter of an inch as occasionally seen. In my own observations, the walls of the right auricle attained a width of 3 lines in only one case, that of the female, 19 years of age, in whom congenital contraction of the pulmonary aperture and deficiency of the septum of the ventricles, was combined with some disease of the tricuspid valves. The parieties of the right auricle measured 2½ lines in a case of combined mitral and aortic disease, in a boy of 18 years of age, and attained the same thickness in a case of aneurism of the apex of the left ventricle, with open foramen ovale. They were also 2 lines thick in the case of great contraction of the mitral and tricuspid valves, and in one of combined aortic and mitral valvular disease.

The maximum thickness of the parieties of the left auricle, mentioned by M. Bouillaud, is 2½ lines. In my own observations its greatest width was two lines in cases of regurgitation through the

left auriculo-ventricular aperture, obstructive and regurgitant disease of the mitral valves, combined aortic and mitral valvular disease, and regurgitation through the aortic aperture.

In the data collected by M. Bouillaud, the aortic aperture had a maximum capacity of 41 lines, and a minimum of 11 lines. Dr Hope mentions a case in which it was contracted to the size of a small pea. Its circumference in one case, measured by Dr Ranking, in a boy 18 years of age, was only one inch and 3-48ths (12 lines). In my own observations, its largest size in males is 45 lines, and its least 30 lines in cases of regurgitation through the aperture. In females its greatest circumference was in a case of combined aortic and mitral valvular disease, in which it measured 35 lines, while in another case before referred to, it was reduced to a mere slit 10 lines in length, and the thickened and ossified valves did not admit of being separated for more than three or four lines.

The pulmonic aperture in the hearts measured by M. Bouillaud, had a maximum circumference of 42 lines, and a minimum of 34 lines. The extreme size of the orifice is not mentioned by Dr Ranking in his observations; but he gives two instances in which the aperture was contracted, as is almost always the case, from congenital malformation, and in these the aperture measured one inch, and one inch and 40-48ths (rather more than 11 lines and 20·5 lines) in circumference. In the cases related by Bertin, Louis, and Burnet, the aperture had a diameter of only $2\frac{1}{2}$, $2\frac{1}{2}$, and $1\frac{1}{2}$ lines. In the case quoted by Hope, the pulmonary orifice would only admit a goose quill, and in one related by Dr Hunter, the pulmonary artery would only admit a small probe, though the patient was 13 years old. In the observations published in the paper, the largest size of the pulmonic orifice in males is 54 lines in a case of hypertrophy, unconnected with valvular disease; and in females 39 lines, in a case of mitral valvular disease. In the cases of malformation before mentioned, in males 15 and 20 years of age, and in the female 19 years of age, the aperture had a circumference of only 13, 12, and 8 lines; and in children $6\frac{1}{2}$ years, and 2 years and 5 months old, it was only $6\frac{1}{2}$ lines, and 5 lines in circumference.

The left auriculo-venticular orifice in the cases of M. Bouillaud had an extreme capacity of 51 lines, and of 24 lines; but he mentions another case in which the aperture was so contracted as only to be 6 or 7 lines long and 5 lines wide, and two in which it was only 3 lines in its largest diameter, or 8 or 9 lines in circumference.[1] Dr Ranking measured two hearts, in which the mitral orifice had a circumference of $5\frac{1}{2}$ inches ($61\frac{3}{4}$ lines), and 5 inches and 2-48ths (54 lines), and he found none in which it was less than 2 inches and 19-48ths (27 lines). In the present series of measurements, the extreme capacity of the left auriculo-ventricular aperture is in males 60 lines, in cases of hypertrophy without valvular disease, and incompetency

[1] Observations 112, 116, and 117.

of the aortic valves; and in females 45 lines in a case of obstruction at the aortic orifice. Its smallest size is in males, in a case of combined disease of the aortic and mitral valves, in which it would only admit the handle of a small scalpel, and had a circumference of not more than 12 lines. In females its smallest circumference was 12 and 18 lines, in cases of uncomplicated mitral valvular disease.

The right auriculo-ventricular aperture in the observations of M. Bouillaud had an extreme capacity of 69 lines, and the only case of contraction of this aperture to which he refers, is that of General Whiple,[1] in which there was an opening one inch long and one line wide. Laennec does not refer to any case of extreme contraction of the tricuspid aperture; but Dr Forbes, in a note to his translation,[2] mentions one in which the aperture would only admit the thumb, and in which, like one I have before referred to, the mitral and the aortic apertures were also contracted. In the case related by M. Louis, contraction of the tricuspid aperture was also, as frequently happens, combined with the disease of the pulmonic valves. Dr Ranking measured a heart in which the right auriculo-ventricular aperture had a circumference of $6\frac{1}{2}$ English inches, or somewhat more than 73 lines. He does not refer to any cases of contraction. In the present series, the largest circumference of the tricuspid aperture in males is 63 lines and 60 lines, in cases of hypertrophy without valvular disease, and 60 lines in cases of obstructive disease, and incompetency of the aortic valves. In females the greatest circumference is 60 lines in a case of obstructive disease, and 57 lines in one of uncomplicated mitral valvular disease. The most extreme degree of contraction of this aperture with which I have met, was in a case of combined aortic, mitral, and tricuspid disease, not mentioned in the tables, in a female 32 years of age, in whom the tricuspid valves were adherent, so that the aperture would only admit the point of the forefinger, indicating a circumference of about 16 lines. The case of a similar kind mentioned in the paper, the aperture was 21 lines in circumference. Elsewhere I have expressed the opinion, that the disease of the valves in these cases, like the similar fusion of the valves of the pulmonary artery, is due to intra-uterine disease, and that they are, therefore, to be regarded as malformations.[3]

In the following table are included the weights and dimensions of hearts examined since the first and second parts of the paper were written. They are not, therefore, included in the calculations as to the weight of the heart, but they enter into those of the dimensions of the organ, both healthy and diseased.

[1] Journal de Médicine et de Chir. Par MM. Corvisant, Leroux, etc. Vol. xix, p. 468.
[2] Page 590, 4th Ed. 1834.
[3] See Lectures on Malformations.—London Medical Times and Gazette, 1834.

APPENDIX TO

Sex.	Age.	Weight.	Girth of Right Ventricle.	Girth of Left Ventricle.	Length of Cavity of Right Ventricle.	Length of Cavity of Left Ventricle.	Thickness of Walls of Right Ventricle.			Thickness of Walls of Left Ventricle.		
							Base.	Mid Point.	Apex.	Base.	Mid Point.	Apex.
	Years.	oz. dr.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.	lines.
M.	14	4 10	41	37	33	34	1½	2	¾	4	5	3
M.	23	6 9½	54	44	42	36	2	2¼	2	5	6	3½
M.	25	11 0				50	44	2	2	2	5	6	3
M.	35	3 0	58	48	44	36	1½	2	1½	4½	5	3
M.	38	11 0	66	50	51	45	2	2½	1½	5	5½	2
F.	19	8 10	54	48	52	42	2	2½	1	4	4	3
F.	21	9 8	60	60	48	44	1½	1½	1½	4½	6	3
F.	21	7 13	58	48	42	36	2	1½	1	6	5	3
F.	20	9 5	60	50	46	41	2	2½	1	5	5	2½
F.	22	9 10	72	48	48	39	2	1½	1	5	6	2½
F.	28	8 8	54	48	48	36	1½	2½	1½	5	6	3
F.	37	7 1	60	48	42	36	1	1½	¾	4	5	2½
F.	50	9 4	60	52	48	41	2½	2½	1	4	6	3
F.	56	10 8					2½	3	2½	6	8	2
F.	64	7 0			34	36	2	2	2	5	5	3
M.	18	16 0	66	56	57	48	3	2½	1½	7	8	4
F.	3 mos.	1 0	27	24	17	18	1⅛	1⅛	1⅛	2⅛	3⅛	1¼
F.	19 yrs.	17 8	84	48	53	46	4	4	3	5	6	2
F.	75	10 2	60	44	39	39	2½	3	2	5	8	3

GENERAL TABLES.

Thickness of Septum.	Circumference of Orifices.				Cause of Death, etc.
	Tricuspid.	Pulmonic.	Mitral.	Aortic.	
lines.	lines.	lines.	lines.	lines.	
4	42	36	39	30	Fever.
6	57	39	42	33	Peritonitis; waxy liver; morbus renum; weight of body 7 st. 7 lbs.
6	54	45	45	39	Cholera.
		36		32	Hemiplegia; weight of body 88 lbs.
5	62	45	54	39	Secondary abscesses in lungs and liver.
5½	60	39	48	33	Fever; weight of body 9 st. 7 lbs.
	57	39	51	33	Cholera.
5	45	39	42	36	Chronic peritonitis; meningitis.
3½	51	39	39	33	Phthisis; weight of body 6 st. 11 lbs.
6	51	42	45	36	Cholera; weight of body 8 st. 10 lbs.
5	57	42	54	36	Cholera.
4	54	39	45	33	Cholera; weight of body 6 st. 2 lbs.
5½	57	36	45	33	Erysipelas.
	45	39	45	30	Chronic bronchitis.
	54	42	45	36	Morbus renum.
7	54	42	45	42	Aortic valves much thickened and incompetent; mitral valves thickened, adherent and permanently open; pericardium universally adherent, sequence of rheumatism 4 years before.
3	24	18	21	16	Atelektasis pulmonum.
6	51	8	45		Malformation; contraction of pulmonic aperture, aorta arising from both ventricles; posterior wall of right ventricle 7 lines thick.
6	54	34	42	32	Obstruction at aortic orifice, with dilatation of aorta; valves with vegetations, some ath. of mitral and in aorta; 5 valves to Pulmonary artery.

In reviewing the communication, it will be seen that the conclusions drawn from the data collected, correspond generally with those of other observers, so far as relates to the weight and dimensions of the healthy heart; but in reference to the diseased organ, they present several differences which are not without interest. They show that the heart may attain a weight and size much larger than was previously supposed, and that the greatest amount of enlargement may be found when there is no material valvular disease, or any other obvious source of obstruction to which it can be referred. They indicate also, a general relation between the dimensions and weight of the heart, and the different forms of disease of which it may be the seat, or in which it may be indirectly implicated. This connection, so far as I am aware, has not been previously clearly pointed out; but if, as I feel much confidence in believing, the conclusions here arrived at, should prove, on more extended investigation, to be of general application, they must be admitted to be not only of much pathological interest, but also of practical importance.

It was my intention, before concluding this memoir, to have alluded to some of the preparations which illustrate the size and form of the heart in disease, contained in the museums of the metropolis; but the length to which the paper has already extended precludes my doing so.

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ON THE

EPIDEMIC MEASLES OF 1854,

IN

LEITH.

By JOHN BROWN, L.R.C.S. Edin., H.E.I.C.S.,

HON. PRES. HUNTERIAN MED. SOC., EDIN., LATE HOUSE-SURGEON, LEITH HOSPITAL.

[FROM THE MONTHLY JOURNAL OF MEDICINE, APRIL 1855.]

In the following communication it is my intention to describe the principal features of interest, which have occurred in the cases of measles, attended by me during 1853–4, while that disease was epidemic in the town of Leith.

It is necessary to remark, that these cases can only be held to represent the epidemic as occurring among the poorer classes, and that in consequence they may furnish in their complications and mortality some variations from the cases observed among those in more affluent circumstances; still, this will be found insufficient to explain the variable nature of the results at different periods, while the appreciable modifying causes, particularly at work among the poorer classes, were to a great extent stationary.

The town of Leith is favourably situated for the extension of epidemic influences. Lying low, and ineffectively cleansed and drained, in its centre, an overcrowded churchyard, and bounded on one side by a harbour which receives the vegetable and animal decay of most part of the towns of Edinburgh and Leith, and which, twice in the twenty-four hours, at low tide, gives off, by evaporation, the most offensive emanations. The town, in addition, is very densely inhabited, containing a population of 30,919, the greater majority of whom reside within an area of a few acres, in narrow streets and closes. If we look to the internal appearance of the houses of the poorer classes, we find that they are generally small and ill-ventilated, over-crowded, and having free communication with each other, and in many instances *several* families are found living in the same room.

This epidemic may be said to have commenced in December

1853, and to have terminated at the end of June 1854. The cases attended during that period have been 170, of whom 16 died, being 9·7 per cent., or nearly 1 in 10.

The patients affected were generally below the age of five years, —a fact similar to what has been observed in former epidemics of this disease, and which is probably not so much dependent upon the predilection of measles for children, as *upon* its frequent occurrence as an epidemic, so that few attain that age without being under, and affected by, such an influence.

The following table shows the proportion attacked to the age:—

Under 1 year, ...	12	Between 7 and 8 years, . .	6
Between 1 and 2 years, . .	24	„ 8 and 9 „ . . .	3
„ 2 and 3 „ . . .	49	„ 9 and 10 „ . . .	4
„ 3 and 4 „ . . .	22	„ 10 and 11 „ . . .	1
„ 4 and 5 „ . . .	22	„ 12 and 13 „ . . .	1
„ 5 and 6 „ . . .	18	„ 13 and 14 „ . . .	2
„ 6 and 7 „ . . .	6	„ 23 and 24 „ . . .	1

This table shows that of these 170 cases, 129 occurred below the age of 5; that the number affected was greatest between the second and third years, and that from this period a gradual decline in the number of attacks is noticeable.

General description of the disease in these 170 cases.—The premonitory symptoms (or period of febrile commotion) were generally mild febrile disturbance, rough cough, with slight dyspnœa, watery eyes and sneezing followed on the *5th day*[1] by the appearance of the characteristic eruption, at first on the face, and gradually extending to the extremities. In three cases the eruption was delayed; in one appearing on the 6th day, in the other two on the 7th. In 3 cases symptoms of cynanche laryngea occurred during this stage; in 2 subsiding on the appearance of the rash; in 1 becoming less severe during its continuance, but again becoming aggravated on its recession. In no instance was the eruption ushered in by convulsions or cerebral disturbance.

The eruption was generally fully developed for 3 days, on the evening of the third day becoming faint and scarcely perceptible on the 6th; in none but severe cases was there any cuticular desquamation, and when this occurred the face was the only part distinctly affected.

Irregularity in the persistence of the eruption was observed in 9 cases; in 3 the eruption was ill coloured, and suddenly disappeared on the morning of the third day; in 2 cases, proving fatal in 24 hours by coma, in 1 by typhoid fever (7 days); in the 4th the eruption was not previously seen by me, but was found absent on the 4th day; coma supervened, terminating in convulsions, and death on the 4th day from commencement of eruptive stage; in the 5th, 6th, and 7th

[1] The appearance of eruption on fifth day was also observed by others in Leith.

cases the eruption remained vivid for five days; in the 8th case appearing scantily for 24 hours, suddenly receding, appearing again three days after, and permanently disappearing in 24 hours; in the 9th case the eruption was persistent for three days, reappearing every third day, and on the ninth day after its first appearance it became again vivid, and finally disappeared in 24 hours thereafter.

An imperfectly established but normally persistent eruption was present in eleven cases.

With one exception the period of the eruption in these 170 cases was unattended with any special complication requiring treatment; the exceptional case being one of croup, where that disease had manifested itself in the premonitory stage. In three cases the eruption supervened while the patients were convalescent from pertussis, a number too small to corroborate the opinion of Copland, West, and others, as to the connection between these two diseases.

In one case a second appearance of the disease was observed—the first attack having occurred two years before, and having been attended by catarrhal symptoms.[1]

In no instance was the rubeola sine catarrho or sine exanthemate observed.

And now having discussed the general features of the disease in its premonitory and eruptive stages, we proceed to consider the general features of the disease in its decline and disappearance as exhibited in the sequelæ, and the complications arising therefrom.

1*st. Of the Nervous System.*—A remarkable immunity from sequelæ of this class was observed. In one case only, was there complete coma and convulsions; in other two cases, slight coma followed the sudden recession of the rash; these cases, however, are not so much specimens of sequelæ as of accidents arising suddenly, and referable not to a local lesion, but to a general one, namely, blood poisoning.

2*d. Of the Respiratory System.*—In fifty-four cases symptoms of acute bronchitis supervened, and in three cases proved fatal, the disease arising during the recession of the rash, and in all the cases observed the eruption being vivid, and the accompanying fever of the inflammatory character. In one case croup supervened and proved fatal, complicated with bronchitis and extreme collapse of lung. In two cases *lobar* pneumonia supervened—these cases recovered.

3*d. Of the Digestive System.*—In eight cases symptoms of dysentery supervened upon the subsidence of the eruption, and in one case proved fatal; latterly complicated with cancrum oris. In all the eruption was vivid, and the accompanying fever of the inflammatory form. In five cases apthæ of the mouth supervened. In two cases only was enlargement of the submaxillary glands observed.

[1] Upwards of 20 cases of rubeola have been twice attended by the medical men of Leith; in all, catarrhal symptoms were present. The seizures occurred at various periods of this epidemic, with four exceptions; two with an interval of two years, two with an interval of one year.

4th. Of Organs of Sense. No Sequela connected with the Ears was observed.—In two cases only did an ophthalmia occur requiring treatment; these were instances of the more severe form of catarrhal conjunctivitis, attended with some degree of palpebral swelling, and a muco-purulent discharge, but leaving no corneal ulcer. In no instance was the cornea primarily affected; in two cases to be noticed under the complications of typhoid fever, it was implicated secondarily.

5th. Sequelæ of a General Nature and probably connected with Blood Poisoning.—Under this head at least 2 of the cases of sequelæ of the nervous system might be put, probably all of the cases, but as this might be objected to, we have put them under the aforesaid head.

Excluding these accidental cases, we find that the sequelæ under this designation were consequent upon an imperfectly established, ill-coloured, and sometimes quickly receding eruption, and consisted of a typhoid form of fever, which supervened in nine cases, and in eight proved fatal. In two cases the fatal termination was ushered in by diptherite, in four by pneumonia, in one by tuberculosis and sloughing of both corneæ, in one by collapse of the lung; the case which recovered was not attended by any special complication. The arrangement of several apparently distinct diseases, under the head of complications, may be considered to be rather arbitrary, but as they seemed to be the direct effects of the sequelæ, it was thought advisable so to arrange them. The following table presents at a glance these various sequelæ, and the complications arising therefrom:—

SEQUELÆ.		COMPLICATIONS.	
Of respiratory organs, .	57	Collapse of lung, . . .	4
Of digestive organs, .	15	Cancrum oris, and collapse of lung, ...	1
Organs of sense, . .	2		
Typhoid fever, . .	9	Diptherite, 2; pneumonia, 4; collapse of lung, 1; tuberculosis and sloughing of both corneæ, 1 . .	8

Having thus briefly sketched the leading features of this epidemic, we now proceed to describe the various forms which the disease assumed, and the tendency to sequelæ which characterised each variety. Dismissing from our consideration those cases which proved suddenly fatal from toxæmia, we may consider the disease to have exhibited itself in three forms, which were generally well defined.

1. The type of the disease, accompanied by a well-marked copious eruption, which appeared on the 5th day, with alleviation of the symptoms which preceded its appearance, a pulse but little accelerated, slight catarrhal symptoms; a critical discharge of urine on the 3d day, containing urates of soda; albumen in quantity sufficient to be appreciable, frequently by chemical tests, if not by chemical tests, almost always disclosing the presence of blood corpuscles by the microscope in the cases examined, and a speedy return to health thereafter.

2. An inflammatory form, characterised by a vivid, copious eruption on the 5th day, the accession of which was attended by *aggravation* of the previous febrile symptoms, along with considerable hoarseness and dyspnœa, and on the 3d day, coincident with the fading of the rash, the signs of acute bronchitis or dysentery arising; these diseases being cut short if seen early, but if fully formed before treatment was adopted, proving frequently fatal.

3. A typhoid or asthenic form, with scanty, livid, and sometimes quickly receding eruption; in severe cases proving fatal on 3d or 4th day of eruption; in milder cases a typhoid form of fever occurring, attended with great prostration, dyspnœa, scanty or suppressed secretion of urine, with progressive emaciation, terminating generally in death, from the 8th to the 18th day; in all but one the chest being unaffected primarily, but in many the fatal termination being ushered in by inflammatory affection of the lungs.

We now proceed to inquire into the mortality of this epidemic, and the various cognate causes which may be supposed to have had an influence upon it, as well as the morbid anatomy in those cases in which a *post-mortem* examination was permitted.

In connection with this point we proceed to consider,

1*st. The Influence of Season, Temperature, etc., upon the Mortality of this Epidemic.*

Under this head we purpose not merely to inquire into the mortality of this epidemic as influenced by these combined causes, but also the relative proportion of sequelæ at different periods, and *their* mortality at such times. In the following rather complex table we have arranged the mortality in different months, as well as the total number of sequelæ from which that mortality was derived.

Months.	Cases.	Sequelæ and Relative Mortality.								Total Mortality.	Per Centage.
		Toxæmia.	Mortality.	Respiratory System.	Mortality.	Digestive Syst.	Mortality.	Typhoid Fever.	Mortality.		
December, .	5	...	...	1	...	2	...	...	...	...	...
January, .	5	...	...	2	...	1	...	...	...	...	...
February, .	34	1	1	14	...	1	...	...	...	1	2·94
March, . .	49	1	1	20	2	1	...	6	6	9	18·03
April, . .	35	1	1	11	2	2	1	2	1	5	14·28
May, . .	23	...	...	4	...	4	...	...	...	...	...
June, . .	19	...	...	5	...	4	...	1	1	1	5·26

We find from the above table that the highest mortality occurred

in March, and arose principally from respiratory sequelæ and typhoid fever, while in the same month the greatest number of cases occurred. In connection with this subject, a table illustrative of the temperature and prevailing winds in these months was prepared, but the evidence was of so negative a character as not to warrant its insertion; the only point worthy of consideration with reference to the high mortality in March, was the frequent occurrence of wet days.

To give the true value to the varying mortality in different months, we must also bear in mind that this epidemic lasted for 7 months, and that probably epidemics, like individual diseases, have a development, a maturity, and a decline. We find, then, that the month of March may be held to represent the maturity of the epidemic, and the period of its greatest virulence. In fine, it may be considered that season influenced mortality, but that this influence was strengthened by the maturity of the epidemic at the period of greatest mortality. We next proceed to consider,

2*d*. *The Influence of Age upon the Mortality.*—I have arranged the results furnished under this head in the following table:—

Ages.	Total Cases.	Mortality.	Per Centage.	Ages.	Total Cases.	Mortality.	Per Centage.
Under 1 year, .	12	...	...	Between 7 and 8,	6	...	...
Between 1 and 2,	24	5	20·83	„ 8 and 9,	3	...	...
„ 2 and 3,	49	7	14·28	„ 9 and 10,	4	...	...
„ 3 and 4,	22	2	9·09	„ 10 and 11,	1	...	...
„ 4 and 5,	22	1	4·54	„ 12 and 13,	...	...	...
„ 5 and 6,	18	...	...	„ 13 and 14,	2	1	50
„ 6 and 7,	6	...	...	„ 23 and 24,	1	...	...

From this table it would appear that the epidemic was most fatal to children between 1 and 2 years of age, a time corresponding to that of dentition; the mortality steadily declines from this period, ceasing between the age of 4 and 5, and not commencing again until at 13, when we have another fatal case. These cases show the fact alluded to by Copland, etc., that measles is most fatal during the period of dentition. The numbers about the age of puberty are not sufficiently great to corroborate the opinion as to the danger of measles at this period, although there is presumptive evidence in its favour, seeing that from the age of 5, up to a period approaching puberty, no fatal cases occurred. In accordance with the established views of infantile pathology, we would, *a priori*, have been inclined to expect the fatal cases which occurred during the period of dentition to have arisen from sequelæ, involving the respiratory or digestive systems, seeing that sympathetic disorders of these systems are

common at such a period; still further, we would have expected the respiratory system to have been solely affected, as in winter and spring (the times in which these cases occurred) sympathetic disorder of the respiratory system is common; while in summer and autumn sympathetic disorders of the digestive system preponderate. The results obtained do not, however, corroborate our expectations, for we find that, of the 5 cases fatal between the age of 1 and 2 years, one arose from sudden recession of an ill-coloured, scanty eruption, two from typhoid fever, one from bronchitis, and one from croup. While of 7 cases between 2 and 3 years, when the sympathetic irritation of dentition is nearly or entirely gone, we find that 2 cases prove fatal from bronchitis, 4 from typhoid fever, and 1 from sudden recession of an ill-coloured eruption. The fatal cases between 3 and 4 being one of typhoid fever and one of dysentery; between 4 and 5 one of typhoid fever; that between 13 and 14 being a case of speedy death from recession of rash.

As it might be objected that a mere recital of the ages at which the fatal cases occurred, can afford no valid information as to the frequency of the sequelæ at different ages, we have in the following table arranged the sequelæ according to the ages at which they occurred.

Ages.	Total Cases.	Respiratory Sequels.	Digestive Sequelæ.	Toxæmia.	Typhoid Fever.
Under 1 year, ...	12	6	1	...	...
Between 1 and 2, ...	24	5	1	1	2
„ 2 and 3, ...	49	16	5	1	4
„ 3 and 4, ...	22	7	2	...	2
„ 4 and 5, ...	22	10	1	...	1
„ 5 and 6, ...	18	5	1	...	...
„ 6 and 7, ...	6	2	1	...	...
„ 7 and 8, ...	6	1	...	...	...
„ 8 and 9, ...	3	2	...	...	...
„ 9 and 10, ...	4	3	1	...	...
„ 10 and 11, ...	1	...	1	...	...
„ 12 and 13, ...	...	...	...	...	...
„ 13 and 14, ...	2	...	1	1	...
„ 23 and 24, ...	1	...	...	...	...

This table, then, shows us that, during the period of active dentition, namely, between 1 and 2 years, the number of cases of respiratory and digestive sequelæ is considerably less than between the age of 2 and 3 years, a time when, in the great majority of instances, the process of dentition is completed. From the facts exhibited by this table, we would be inclined to consider that in this epidemic

age had no influence in predisposing to particular forms of sequelæ; in a word, we conceive that the type of the disease determined the form of sequelæ, while from the high mortality exhibited between 1 and 2 years of age, the coincidence of dentition may have influenced their fatality.

3d. Sex, as Influencing Mortality.—Of these 170 cases, 75 were boys, and 95 girls; of the boys 3 died, or 4 per cent.; of the girls 13 died, or 13·68 per cent.

It would thus appear that the number of females attacked was greater than that of males, a fact which may be owing to the excess of females in the population, seeing that according to the last census there were 110 females to 100 males living.

The mortality of females is enormously greater than of males; a fact at variance with what is observed in epidemics of fever, in which, although the seizures are fewer than in males the mortality is much less. If we look to the births which take place in this country, we find that 105 boys are born to 100 girls, while the whole population living gives a preponderance to females; in what way, then, can we account for the enormous mortality of females in this epidemic on such data? The researches of Professor Simpson have proved that while the *whole* mortality, of children, shows an excess of males, still that it is limited to the first year of life, and arises in consequence principally of nervous diseases induced during labour by the size of the male head.

In this epidemic we find that during the first year of life, while the condition of male and female are unequal, that no deaths arise; yet that when we come to a time when the conditions of the two sexes are equal, the mortality is in excess on the female side, we infer, therefore, that in this epidemic sex had a decided influence on mortality; whether it is always so with measles we cannot say, but it is certainly worth the attention of future observers. If we look to the relative number of sequelæ affecting each sex, as a means of explaining these results, we find that of 57 cases of sequelæ of the respiratory system, only 23 were girls, but the whole mortality belongs to that sex. Of 15 cases of sequelæ of the digestive system, we find that, although the seizures were nearly equal, the only fatal case belonged to the female sex. Of 3 cases fatal from toxæmia, we find 2 to be girls. Of 9 cases of typhoid fever 6 are girls, the only one that recovered being a boy. The conclusion we would be inclined under these circumstances to come to is, that while the total number of sequelæ shows the male sex to be most frequently affected, yet, that the mortality of that sex is notably less in consequence of their superior strength in resisting or overcoming disease when established.

4th. Constitution of Patients' previous Diseases, and State of Health at Invasion of Disease as influencing Mortality.—Upon this subject we will not enter into detail, seeing that their elaboration would prove neither interesting nor instructive; we will, therefore, only

particularly inquire into the constitutional states, etc., of the fatal cases, with such references as may be necessary to understand their relation to the recoveries. Of eight cases in which typhoid fever proved fatal, six were stout and of previous good health; the seventh was a twin of feeble health, but with no constitutional taint; the eight was a feeble child in consequence of labouring under a severe attack of pertussis at the time of seizure; the only successful case was a robust boy, but of no better apparent health than the six fatal cases. Of four cases fatal from sequelæ of the respiratory system, all were previously in good health; in none of these cases was there any obvious constitutional taint, for farther on we will show that in the post-mortem examinations tuberculosis was absent in all the sequelæ of the respiratory system. One child, previously of robust health, and no previous disease, died of dysentery. Three children died suddenly from toxæmia; in two the previous state of health and constitution were good, in the third the state of health was doubtful.

It would appear that the typhoid fever attacked those of a robust constitution generally. The respiratory and digestive sequelæ attacked also the robust and strong, and we accordingly find that the fatal cases belong to that category; the cases of toxæmia are too small to come to any conclusion upon as to the state of health. In predisposing to this form, let me here remark that as the general rule the fatal cases were the most robust, while of the successful cases a great, a very large majority, were feeble strumous creatures; in a word, the epidemic in its mortality bore no obvious relation to a previously debilitated state, but the reverse. Were we to describe the kind of children we most dreaded to take measles, we would specify those in robust health, who either took that form designated as inflammatory, and in consequence bronchial inflammation, of a severe character, or a typhoid fever attended with great depression.

5th. Ventilation—state of dwelling, etc., as influencing Mortality.—The evidence of the influence of these combined causes is also not of a nature which we would have been inclined to expect. In the three cases of sudden recession of rash and blood poisoning, the houses were all ill ventilated, overcrowded, badly lighted, and in one very damp. In the four fatal cases of respiratory sequelæ the dwellings were in three well ventilated, etc., in the fourth ill ventilated and overcrowded. In the only fatal case of sequelæ of the digestive system the dwelling was well ventilated and not overcrowded. In the eight fatal cases of typhoid fever the houses were in six well ventilated, etc., and in two ill ventilated and overcrowded. Generally speaking, no very close connection between the state of the dwelling and its ventilation, as compared with the results or complications of the disease was observed, although in predisposing to the disease, and leading to its extension, their influence was undoubted. In those cases in which sudden recession

of the rash was followed by symptoms of blood poisoning, we would be inclined to consider the state of the dwellings to have borne no unimportant relation to the results observed, but in the other cases the conditions present during the progress of the disease did not seem to cause any particular form of sequelæ, or influence their results. With the above exception we therefore consider that the mortality of this epidemic did not seem due to any hygienic imperfections, but more to the form of the disease taken, with which these imperfections bore no apparent relation.

6th. *Period at which Treatment was commenced, and attention of Parents to orders, as influencing Mortality.*—Generally speaking, a direct influence was observed between the successful issue and the early period at which the patients were seen, the exceptions being the cases of toxæmia and typhoid fever, in which cases remedies seemed of little avail. Although, from the short time intervening between the first visit and the fatal event, in the cases of blood poisoning, no treatment could have time to develope its effects. Of the 3 cases fatal in this way, 1 died 2 hours after first visit, 1 in 4 hours, and 1 in 7 hours. Of the 4 fatal cases of sequelæ of the respiratory system, 2 were not seen until the evidence of organic change in the lung was present (collapse); in 1 conjoined with croup, which had gone on to exudation; the fourth case was seen from the commencement, but went on to collapse of the lung. In the first three cases in which organic changes were present before treatment was commenced, the remedies prescribed were regularly administered. In the fourth case, seen from the commencement, the remedies were at the first administered very irregularly, and latterly not at all, the mother being engaged all day in field work, while the child was left in charge of one not much its senior.

The only fatal case of dysentery was well attended to, and the treatment was early commenced.

In the 8 fatal cases of typhoid fever, treatment was early commenced; and in most cases the orders given were attended to. It thus appears that of these 16 fatal cases, 7 were not seen until either the time before death was too short to permit of the action of remedies, or the time past for their use, when disease had progressed to organic change, or the remedies prescribed not administered. In these days of therapeutical scepticism, it is, we imagine, a conclusive evidence of the effect of remedies promptly administered in acute diseases, to show that of fifty-four cases in which bronchitis supervened, in many severe, the three cases which proved fatal did so from the want of early treatment; for we find that of these fifty-four cases three were nearly similar in all respects but one, and that being the treatment.

We have now concluded our remarks upon the causes which influenced the mortality in this epidemic, a subject which, no doubt, will be thought by many to have been dealt with too minutely, still by the means adopted only could we have given the proper value to

certain circumstances, generally acknowledged to bear in no small degree upon such a result. To recapitulate the results of these observations, we find—1. That as regards *season*, the spring months were those in which most fatal cases occurred. 2. That in this epidemic temperature and prevailing winds had probably no influence upon the mortality, but that coincident with the highest mortality a moist state of the atmosphere was observed. 3. That age had no direct influence upon the mortality, but, that as during dentition the highest mortality occurred, that period is unfavourable from the standard of health being lowered. 4. That sex seems to have exerted a powerful influence upon the mortality—the mortality on the female side being enormously greater than on the male. 5. That constitution, previous diseases, etc., evinced no direct influence over the mortality, but may have indirectly predisposed to certain forms of the disease, attended with complications. 6. That generally the influence of ventilation, state of dwelling, etc., upon the mortality, was not at all conclusive. 7. That generally the time at which treatment was commenced, and the degree of attention given to orders, exercised a powerful influence in diminishing or increasing the mortality. Before entering upon the post-mortem appearances, we would here observe that it is not meant to deny the positive influence of certain causes above enumerated in disease, but to show that in this epidemic the influence of these causes was of a negative kind.

Post-Mortem Appearances.—Of the sixteen fatal cases, post-mortem examinations were only permitted in eight. Of the three cases fatal from sudden recession of the rash, one was examined—

1. A stout girl, æt. 2, of previous good health, was attacked with measles. The eruption was scanty and discoloured, suddenly disappearing on the morning of the third day, attended with dyspnœa, feeble pulse, and a drowsy semi-comatose state, proving fatal in seventeen hours after the recession of the rash. Post-mortem, twenty-four hours after death. Body stout, a good deal of subcutaneous fat. *Thorax*—pericardium moist, heart healthy, right ventricle contains dark fluid blood in moderate quantity, left contracted and empty. Pleuræ non-adherent, moist; some vesicles of air between pulmonary pleuræ and lungs in front; lungs generally congested, most so at back part; right lower lobe non-crepitant, infiltrated with blood, non-granular; bronchi exude a frothy fluid; trachea congested. *Abdomen*—liver large, congested; kidneys and spleen also congested.

Of those in whom bronchitis supervened and proved fatal, three in number, two were examined:—

1. A female child, æt. 15 months, previously healthy, was seized with measles, followed by aggravation of the cough, and febrile symptoms on the third day, simultaneous with the fading of the rash. These symptoms were neglected for about fourteen days; at this period medical advice was had recourse to for the first time; the child was found to be labouring under extreme collapse of the lung and croupy breathing, and died a few minutes after. Post-mortem thirty-six hours after death. Body pale, subcutaneous fat in tolerable quantity. Thorax—pericardium contains about a drachm of straw-coloured serum; heart valves normal; right ventricle distended, with dark coloured fluid blood; left ventricle contracted and empty. Pleuræ non-ad-

herent, moist; lungs both imperfectly crepitant throughout—in the substance of both lungs, numerous lobules of carnified tissue are observed; both lower lobes are condensed by bloody infiltration; bronchial tubes filled with purulent matter, tenacious and in greatest quantity in the smaller tubes; trachea congested; larynx, no deposit on free surface, but greatly thickened from a deposit in the submucous tissue of a serous character. *Abdomen*—liver, spleen, kidneys, normal.

2. A female child, æt. 2, previously healthy, was seized with measles, followed on the third day by an aggravation of the cough and fever; when first seen these symptoms had been allowed to go on, and the child seemed labouring under collapse of the lung, attended with dyspnœa and laryngeal cough; died suddenly, fourteen days from appearance of eruption. Post-mortem thirty hours after death. Body pale, emaciated. Thorax—pericardium moist; heart valves healthy; right ventricle dilated with semi-fluid dark blood; left, contracted, empty. Pleuræ non-adherent, moist; left lung, upper lobe, œdematous; lower, non-crepitant, whole lobe nearly composed of carnified lobules, with here and there a small portion of crepitant lung tissue; right lung, a large emphysematous bulla, size of a marble, is found between lung tissue and pulmonary pleuræ, at base in front; lower lobe condensed, the lobules generally have the usual appearance of carnified lung, but others are lighter coloured and imperfectly granular (lobular pneumonia)? Bronchi—all exude a tenacious purulent fluid, which is most abundant in the smaller bronchi. *Abdomen*—liver, spleen, kidneys, healthy.

The case which proved fatal from the supervention of croup was examined:—

1. A healthy stout female child, æt. 14 months, was seized with measles, which commenced to recede on the third day, attended by increase of the febrile symptoms, cough, and the physical signs of acute general bronchitis, which yielded, but left a laryngeal cough; in three days croup commenced, running on rapidly to death in twenty-four hours after the symptoms attracted notice. Post-mortem forty hours after death. Body pale, not emaciated; blueness of nails of hands and feet; a good deal of subcutaneous fat. Thorax—pericardium moist; heart valves normal; right side gorged with semi-decolorised clots; left empty and contracted. Pleuræ moist, non-adherent; lungs, left upper lobe crepitant, slightly œdematous; lower lobe sparingly crepitant, numerous carnified lobules, with œdematous crepitant tissue between; right upper lobe crepitant, slightly œdematous; middle lobe small, nodulated externally (like as is seen in advanced cirrhosis of liver), hard, non-crepitant. This lobe is made up of these nodules, which seem fibrous on section, and contain no cavity; between them are pieces of non-crepitant lung tissue, which become crepitant by inflation, the fibrous bodies remaining unaffected; they apparently represent an extreme degree of collapse; lower lobe imperfectly crepitant, carnified in patches; *larynx and trachea*, rima plugged by a fibrinous mass extending fully an inch down trachea, non-adherent, of the thickness and shape of an ordinary goose-quill; trachea congested to its bifurcation, no lymph on its surface; at the bifurcation a yellowish and very tenacious fluid is found proceeding downwards to the smaller tubes of both lungs, which are completely filled. *Abdomen*—liver, spleen, and kidneys, healthy.

The diseases of the digestive system include one fatal case of dysentery, which was examined:—

1. A stout healthy female child, æt. 3, seized with measles, followed on the 3d day (synchronous with recession of the rash) by symptoms of acute dysentery. On 15th day of its duration *cancrum* oris supervened, destroying part of the upper lip;—the two upper incisors, and about half of the hard palate, with commencing death of the bone beneath; died exhausted three days after its supervention, or 17th day from commencement of dysentery; no chest complication. Post-mortem thirty hours after death. Body pale, thin, little subcutaneous fat. Thorax—pericardium moist; heart valves normal; right side distended, with semi-fluid blood; left ventricle contracted and empty. Pleuræ non-adherent, moist; lungs, *left*, marked emphysema of edge of upper lobe in front, vesicular and interlobular—the latter preponderating. Upper lobe imperfectly crepitant, with carnified portions in centre about half an inch square; lower lobe sparingly crepitant, congested, at back part, numerous collapsed lobules. *Right*, upper lobe crepitant, with vesicular emphysema at apex in front, and atrophy of edges; middle lobe œdematous; lower lobe condensed, with blood, no granulations. Bronchi generally exude a pale frothy fluid on pressure. *Abdomen*—stomach, tissue soft, contains a little milk and bread; not congested. Whole *intestine*, from stomach to within a foot of rectal end, normal, and stained throughout by bile; about twelve inches from anus two or three ulcers, about the size of a split pea, are found, they had only destroyed the mucous membrane; for the last six inches of its course the whole rectum is covered by a deposit of lymph of honey-comb appearance, which is easily stripped off from the congested mucous membrane beneath. Liver, spleen, and kidneys, normal.

Of the fatal cases of typhoid fever, eight in number, four were examined after death:—

1. A stout female child, æt. 1½, was attacked by an imperfect eruption of measles, which commenced to recede quickly on the third day, attended by a typhoid form of fever and great dyspnœa; this state continued for seven days, no physical signs of chest disease having in the interval occurred, but on the evening of the seventh day a fine moist rale was heard in the lower and back part of both sides, and she died early on the eighth day of the typhoid state. Post-mortem thirty-five hours after death. Body pale, not emaciated; a good deal of subcutaneous fat. Thorax—pericardium contains about a drachm of straw-coloured serum; heart valves normal; right side distended, with semi-fluid dark blood. Pleuræ—A few patches of lymph on diaphragmatic surface of right pulmonary and costal surfaces of pleuræ; *right lung*—all this lung, except a portion of about two square inches at apex, is condensed; on section the colour of the lung is a dirty straw, with granulations: the upper lobe is not wholly in this state, there being patches of one and two square inches, with congested lung tissue between; the other lobes are uniformly granular; *left*, upper lobe crepitant, œdematous; lower, condensed, red, and granular on section. Bronchi, especially the smaller, exude purulent mucus on pressure. *Abdomen*—liver, spleen, and kidneys, congested; otherwise normal.

2. A stout healthy female child, æt. 2½, was seized with an imperfect eruption of measles, and on the third day fell into a low typhoid state, attended with dyspnœa, but no physical signs of chest affection on the 7th day of the typhoid state; both cornea observed to become dim, with no previous inflammatory signs beyond slight injection of the palpebral conjunctiva; this state was succeeded next day by ulceration between cornea and sclerotic; ultimately both eyes sloughed, leaving a small fleshy looking mass at bottom of each orbit; died of exhaustion on tenth day of fever, or thirteenth day from appearance of eruption. Post-mortem forty-six hours after death—Body pale, emaciated; little subcutaneous fat; eyelids sunk; cornea, etc., gone. Head—membranes pale, no deposit. Brain—pale, firm; lateral ventricles contain a little clear serum; optic commisure, and nerves firm, but, like the brain, very anæmic. Thorax—pericardium contains about half an ounce straw-coloured serum; heart valves normal; right side distended, with dark fluid blood; left auricle contains a fibrinous coagulum; ventricle contracted, empty. Pleuræ moist, no adhesion; lungs, left upper lobe imperfectly crepitant, contains at

apex two yellowish cheesy masses, each about half an inch square, with defined borders; lower lobe condensed, with blood and carnified lobules; *right*, upper, and middle lobes crepitant, lower imperfectly crepitant, containing numerous crude miliary tubercles. *Abdomen*—liver, spleen, and kidneys normal.

3. A stout female child, æt. 2, had been labouring under well marked pertussis for several weeks, with well-formed hoop and accompanying bronchitis; the paroxysms were becoming less frequent and severe, when it was seized with measles, which came out imperfectly, receding suddenly in twenty-four hours, again appearing imperfectly three days after, and permanently disappearing in twenty-four hours. She fell into a low typhoid state, with slight dyspnœa, and died exhausted on nineteenth day of typhoid state, the chest signs being, during that period, slight inspiratory roughness, with no marked rale. Post-mortem forty-eight hours after death—Body pale, emaciated; little subcutaneous fat. Thorax—pericardium moist; heart valves normal, right side filled with semi-decolorised clots; left contracted, empty; pleuræ, no adhesions, moist; *lungs*, left upper lobe crepitant; lower lobe condensed, tough, carnified; *right*, upper lobe, superior half, crepitant, inferior half, non-crepitant, externally presenting a nodulated appearance, exactly similar to that observed in cirrhosis of the liver in an advanced stage; the middle lobe is atrophied, and seems made up of these nodules, which, on being cut into, are firm, fibrous-looking, and with no apparent cavities; they are about the size of a small marble, and have an imperfectly crepitant tissue between; lower lobe non-crepitant, carnified; smaller bronchi exude a tenacious yellow fluid. *Abdomen*—liver, and spleen, normal; kidneys congested, lobulated.

4. A stout healthy female child, æt. 2, was seized with an imperfect eruption of measles, which receded quickly on the third day, followed by a typhoid state, accompanied with dyspnœa, slight croupal symptoms, and no chest signs beyond inspiratory roughness, and for a few days before death dorsal impairment of percussion, with resistance. Died exhausted, on nineteenth day, of fever, a small abscess of cornea commencing. Post-mortem forty-eight hours after death—Body pale, emaciated, little subcutaneous fat. Thorax—pericardium moist; heart, valves normal; right side gorged with semi decolorised clots; left, contracted, empty. Pleuræ—recent adhesions of pleuræ in front of left lung, soft, white, and easily detached from opposed surfaces; *lungs*, left upper lobe crepitant, normal; lower, condensed, carnified; *right*, upper lobe, crepitant; middle, condensed, carnified; lower lobe, imperfectly crepitant, containing some lobules of fawn-colour and granular, with healthy crepitant tissue between; larynx and trachea, pale, no exudation. *Abdomen*—liver pale, spleen and kidneys normal.

To recapitulate the results of these post-mortem examinations, we find,—1*st*. That in the case of toxæmia, from sudden recession of the rash, the only changes observed were extreme congestion of the viscera, and a fluid state of the blood.

2*d*. That in the cases of respiratory sequelæ examined, the evidences of marked collapse were present in all, and coexistent with a state favourable for its development; but that emphysema was generally absent, contrary to what is found in the adult, and probably so in consequence of the conditions necessary for its production, being absent owing to the enfeebled state the patients were in, and the yielding state of the parieties of the chest.

3*d*. That in the fatal case of digestive sequelæ—a case of dysentery—we have the unusual circumstance of the formation of a false membrane upon the interior of the intestine, and also evidence of the occurrence of marked collapse of the lung in a child who never

had bronchial disease, but who had been lying in a semi-lifeless state for several days, thus proving the accuracy of the opinion of Dr West, that collapse may occur in such circumstances without bronchitis.

4*th*. That in the four cases fatal from typhoid fever which were examined, the parenchyma of the lungs was primarily affected in two; and in two secondarily, in the one case labouring under pertussis, the lesions found seemed more the result of it than of measles.

And now having detailed the leading features of this epidemic, we will briefly advert to the state of the urinary secretion in these cases. It has been already incidentally stated that the urine was albuminous, as proved either by chemical tests or by the presence of blood globules under the microscope. On the difficulties connected with the obtaining of urine for continuous examinations in children we need not dwell; suffice it to say, that in only eighteen cases could this secretion be obtained regularly at various intervals in the progress of the disease, but in those cases in which the observations were interrupted, the results thus imperfectly obtained were not at variance with those which were more precise. In accordance with the arrangement followed in a previous part of this paper, we will consider:—1*st*, The secretion as occurring in cases of uncomplicated measles. At the outset of the eruption in those cases in which the body was quickly covered, an appreciable quantity of albumen was discovered by chemical tests, and under the microscope blood globules were also found, from this period, till the fading of the rash, the urine continued free of albumen, but attended by a slight deposit of urate of soda, and a density varying from 1015 to 1030, the varying nature of the specific gravity being apparently caused by the comparative dilution of equal quantities of solids. We use the term apparently because upon such a point in children exactitude is next to impossible. On the third day, and in proportion to the disappearance of the rash, the quantity of solids was increased by urate of soda, and albumen found either by chemical tests, or the presence of blood globules under the microscope, in no instance accompanied by an increase of epithelium, and in only one case by fibrinous tube-casts in small quantity. In two cases both brothers, and convalescent from pertussis, no albumen was found either on the third or subsequent days, but in them the eruption was remarkably persistent, and showed no signs of fading till the fifth day, when it commenced to disappear slowly. On the fourth day, in those cases in which albumen was appreciable on the third day by chemical tests, a small deposit was still found. In those cases in which the quantity was so small on the third day as only to be diagnosed from the presence of blood globules under the microscope, it was absent on the fourth day. In only one case was the deposit of albumen visible after fourth day, and in this case the deposit was copious on the third day, absent on the fourth, and again present on

the fifth. In all the cases throughout, the chlorides did not seem to be diminished as compared with normal urine. It was also noticed that in those cases in which the albuminous deposit was best marked, the recoveries were most speedy.

The urine could not be obtained for examination in any of the cases of toxæmia from sudden recession of the rash.

In the class of cases in which bronchitis supervened, 4 were examined for changes in the urine; in two cases on the 3d day, while the bronchitis was forming, so to speak, one or two blood globules were observed under the microscope, but no other sign of albumen was again detected. In one case the urine was slightly albuminous on the 3d day; on the evening of the 4th day, with amendment of the bronchial symptoms, an increase of albumen was observed in the urine, with numerous flakes of fibrin under the microscope; this appearance was diminished on the 5th day, and was not subsequently found present. In the 4th case an opalescence of the urine was produced by heat and nitric acid on the 3d day, with a few blood globules under the microscope; this appearance was not again found until on the evening of the 5th day, coincident with amendment of the bronchial symptoms, a deposit of albumen, with blood globules under the microscope was observed. In all these cases throughout an abundant deposit of urates was observed, with scanty high-coloured urine, and a diminution of the relative amount of the chlorides.

In those cases in which dysentery supervened, the urine was examined in one case on the 3d day, before the symptoms were well formed, a few blood globules were observed under the microscope; the after examination was interrupted.

In those cases in which typhoid fever supervened, no continuous observations could be made. In one case the urine was examined on the 3d day, and found to contain a few blood globules under the microscope; the meagre results in such an important inquiry were caused by the difficulty of obtaining the secretion—for in all these cases an almost total suppression of urine was observed, and that which was secreted was passed involuntarily in bed.

These observations are not so numerous as to warrant us in inferring that the same results would have been obtained had *all* the cases been examined; yet we would, from these examinations, infer as highly probable:—1*st*, That in cases of quickly spreading eruption albuminous urine results, and that this is probably owing to the sudden suspension of the functions of the skin. 2*d*, That the system accommodating itself to this change, or the skin in part resuming its functions, no albumen is found during the onward progress of the eruption. 3*d*, That in uncomplicated measles a critical deposit of albumen occurs in the urine on the 3d day, simultaneous with the fading of the rash, which gradually disappears; and that in proportion to the presence of this deposit, so is the recovery quickened. 4*th*, That when the eruption is very tardy in its disappearance, the urine

was not found albuminous; and that probably the albumen is present in proportion to the quick recedence of the rash. 5*th*, That in sequelæ, involving the respiratory mucous membrane, the character of the secretion on the 3d day is not so decided, but that, simultaneous with the decline of the sequelæ, the albumen becomes better marked, and large quantities of fibrin are excreted. 6*th*, That in sequelæ involving the alimentary mucous membrane, the same characters as found in respiratory sequelæ are probably present; but that the observations made are insufficient to determine this point with precision. 7*th*, That in the typhoid form of sequelæ the observations are insufficient to determine the quality of the urine, but that its quantity was invariably very much diminished.

We now proceed to consider the treatment adopted in these cases. Treatment—In our description we will follow the arrangement adopted in a previous part of this paper, and consider the treatment followed in the various stages of the disease, as well as in the sequelæ, which were developed during its abatement.

1*st*. Treatment of the disease.

a. *Previous to Eruption.*—In all cases the patients were confined to the house; and on the evening of the 4th day a warm bath was given, and in some cases an emetic administered. Of the 170 cases only two were treated otherwise, in consequence of the occurrence of croup.

b. *During progress of Eruption.*—With one exception, a case of croup, this stage required no special treatment. In one or two cases with rough cough an emetic was given; in all the use of cold water as a drink was permitted, and the ventilation of the apartment enjoined.

c. *During subsidence of Rash.*—In cases unattended by sequelæ, nothing was given beyond, in some cases, a mild purgative, and then only when constipation existed; a practice with difficulty followed, as the old pathological notions of "peccant humours" is still fashionable among the lower orders; and the sequelæ which follow the disease are attributed by them to a want of purgatives. It may not be out of place to mention here another pernicious practice adopted by the lower orders during the incubative stage of the exanthemata generally, namely, the giving of sulphur and whisky with exemplary regularity, to children of whatever age, with the object of bringing out the eruption.

2*d*. Treatment in cases of toxæmia from sudden secession of rash.—In 2 cases where the pulse was perceptible, warm baths and sinapisms to the chest and abdomen were had recourse to, along with the exhibition of carbonate of ammonia and chloric ether internally. In one case (æt. 13) convulsions ensuing, with signs of determination of blood to head, as flushed face, increased temperature, strong pulsation of carotids, and a quick, strong, hard pulse; the temporal artery was opened and about 6 oz. of blood abstracted without benefit.

3*d*. Treatment of sequelæ of respiratory system.

a. *Of Bronchitis.*—In the inflammatory form of the disease it was observed that, simultaneous with the recession of the rash, the physical signs of bronchitis were established in most. In none of these cases was blood-letting had recourse to; indeed, the early tendency of the disease to collapse and exhaustion, along with the satisfactory effect of a less exhausting treatment, rendered it unnecessary. The treatment adopted was to administer emetic doses of tartrate of antimony every two hours until an impression was made upon the system and the disease; in those below one year ipecacuan was given in preference to antimony. If this treatment was promptly adopted, the progress of the disease in most cases was broken, and the results speedily disappeared under the use of small doses of ipecacuan and antimony. If, on the contrary, this treatment was not attended to, or if aid was not called in early, the disease progressed and terminated with great rapidity in marked collapse of the lung, with extreme exhaustion, requiring the use of stimulants, the most serviceable being a mixture containing carbonate of ammonia, chloric ether, and infusion of senega; and in all the liberal administration of wine was had recourse to upon the supervention of these symptoms. When the disease became chronic, and was attended with profuse secretion from the bronchi, marked benefit was derived from the use of blisters, applied so as to produce redness—not vesication—and applied successively to various portions of the chest.

b. *Of Croup.*—In three cases in which croup appeared before the eruption; local blood-letting, with frequent emetic doses of antimony, were used with success, along with small doses of ipecacuan and calomel in the interval; but in all but one the symptoms disappeared on the occurrence of the eruption. The case which supervened after the recession of the rash was treated first by emetic doses of tartrate of antimony, and then of sulphate of copper; but owing to the exhaustion which ensued, recourse was speedily had to stimulants similar to those specified, when collapse of the lung was established in bronchitis.

c. *Of Pneumonia.*—In the two cases in which this supervened, consolidation of the lung had occurred before they were seen. The general symptoms were of a kind demanding stimulants, which were accordingly given; the most serviceable being the senega infusion, with chloric ether and carbonate of ammonia; blisters were also had recourse to with apparent benefit.

4th. Treatment of sequelæ of digestive system.

a. *Of Dysentery.*—In no case was blood-letting used, or apparently required—the symptoms in all but one yielding to the use of the warm bath, Dover's powder in small doses at first, and then united with gallic acid after the acuteness of the disease had abated; in one case, which was less acute than ordinary, no remedies seemed to have much effect, and the patient sunk, the disease being latterly complicated with cancrum oris.

b. *Aphthæ.*—This disease speedily yielded to the influence of chlorate of potash, combined with borax locally.

5th. Treatment in those cases in which typhoid fever supervened.—The treatment was in all cases of a tonic and stimulating nature; at the outset when warrantable, warm baths were given with sinapisms to the chest, thereafter quinine, wine, and chlorate of potash frequently, but without benefit of a permanent kind, except in one case, which recovered without any complication. The cases which proved fatal from pneumonia were so rapid, that in no case could any treatment be adopted for this disease. In two cases in which diptherite supervened, the fauces were touched at intervals with Green's nitrate of silver solution, without the slightest appreciable benefit.

It would be foreign to the scope of this paper to enter into any comparison between this epidemic and previous ones; we leave that for abler hands, contenting ourselves with taking in conclusion a hurried glance at the various points elucidated in our investigation, as well as their bearing upon the pathology of measles. To recapitulate these we find that the greatest proportion of cases occurred in those under five years of age, after which period the seizures gradually diminished.[1] We have seen that during the premonitory stage, with three exceptions, this stage was free from any complications, that in none was there any disturbance of the nervous system, and that in the majority of cases the eruption appeared upon the fifth day; but that in some it was delayed, and in others irregular, in the period of its appearance. In the stage of eruption it was found that with one exception the disease went on uncomplicated, and was principally noted for its characters, as affording the means of guarding against or being prepared for certain sequelæ.

In the stage of recession of the rash, we found that excluding those cases fatal from blood poisoning, the sequelæ which followed showed a marked tendency to divide into three classes—two of these, affections of the respiratory and digestive system being observed to follow a vivid eruption with accompanying inflammatory fever—while the third class consisted of a low form of fever, following an imperfectly established or quickly receding eruption. This stage was also remarkable from the almost total immunity observed in diseases of the nervous system, and of the organs of sense, while in it the whole mortality of this epidemic is to be found.

The effects of various causes of disease upon the mortality of this epidemic, have shown us some curious facts, the most notable being the varying nature of the mortality at different seasons, the influence

[1] In a previous part of this paper this fact was considered to be owing not to the susceptibility of children in particular to the disease, but to the frequent occurrence of measles as an epidemic, so that few escape its influence. The recent observations of Dr Panum of Copenhagen upon an epidemic of measles in the Faroe Islands prove this. From his observations, he concludes that age, however advanced, in no measure diminishes the susceptibility. His conclusions are decisive, for *all* were ready to receive the disease, no cases having occurred there since 1781.—See *Monthly Journal*, June 1851, p. 589.

of age and sex upon it, the negative evidence afforded by other causes, and, lastly, the effects of early treatment in diminishing the mortality.

The post-mortem examinations have principally shown us the effects of various other diseases in an extreme degree, while the results obtained in an examination of the disease itself have been of a negative kind.

It has been also shown that the urine in uncomplicated measles undergoes various changes, but that these changes are interrupted or suspended when sequelæ arise.

From a consideration of the facts which have been advanced in a previous part of this paper, it will be obvious to the reader that the leading characters of this epidemic are not to be found in the disease itself, but in the pathological states which became developed during its abatement. The mortality from this cause has been high when we consider that the mortality of measles, according to Dr West, is 3 per cent., while in these cases it was 9·7 per cent.; but we find an approximation to a similar mortality in this visitation as seen in other places, leading us to believe that this epidemic has generally been attended with a high mortality.[1]

Although the disease thus proved fatal upon its decline from the occurrence of sequelæ, the characters assumed by the disease itself were so distinctive as to warn us of sequelæ of a certain kind, and thus enabled us to act upon slighter grounds than a want of knowledge upon this point would have warranted. But the experience had to be gained, observations had to be made, comparisons instituted, and treatment tried, modified, or altered, so that during its progress and decline, the remedial measures adapted to the peculiar nature of the epidemic were better understood and more successfully practised.

On a careful consideration of the sequelæ which followed, we find them divided into two distinct forms of diseased action, and answering to the description of a sthenic and asthenic disease. In the first great class we found preceding it a vivid bright eruption, with concomitant fever of a sthenic type, and the evidence of inflammatory disease of the respiratory or digestive system springing up during its decline.

In the respiratory form there was observed a tendency to the rapid formation of a permanent pathological change, collapse of the lung, to an extreme degree. In these cases the remarkable reversal of the breathing movements pointed out by Dr Rees were well marked, the lower part of the chest being retracted and drawn inwards during inspiration;[2] another occurrence was also observed in these cases,

[1] See Abstract of Registrar General's Report for 1853. Ranking's Abstract. January—June 1854, p. 277, *et seq.*

[2] The discovery of collapse of the lung, and its relations to respiratory diseases, which has almost revolutionised infantile pathology, like auscultation and Bright's disease, was nearly made years ago. Reil, in Memor. Clin. 1792, seems to have first separated bronchitis from other lesions of the lung, and describes as occurring in 1791 a form of variola, complicated with severe bronchitis, in which he found the most formidable symptoms to be retraction of the sternum. At each inspiration he found that the sternum, especially its lower end, was, with the cartilages of the ribs, drawn so much inwards that it appeared to touch the dorsal vertebræ, and formed a depression in which the fist could be placed. This was, when well marked, always fatal, and occurred chiefly in children below six years of age. The change in the parenchyma of the lung he attributed, like many after him, to inflammation; but the accurate description of symptoms leave no doubt as to the change being collapse.

namely, that upon the supervention of collapse the rales formerly abundant were found to cease, while a post-mortem examination disclosed the presence of a viscid semipurulent secretion in the bronchi; the cause of this seemed to be the want of inspiratory power, so that air was not permitted to penetrate, the respiratory murmur was consequently more harsh, accompanied by slightly impaired percussion note, but with well-marked sense of resistance. Contrary to what is observed in the bronchitis of the adult, emphysema was generally absent in these cases, which was doubtless owing to the feeble inspiratory power which in healthy children is never strong, but in disease becomes notably weak, and likewise to the yielding nature of the parieties, which permits of their accommodating themselves readily to the changes in their interior.

In the second class of sequelæ, which we have termed asthenic, the symptoms were also preceded by a peculiar form of eruption, and consisted of a general typhoid state, which we have previously described as being attended with great dyspnœa, scanty urine, progressive emaciation, and high mortality. Although a general disease, it had local symptoms, of which the principal and most constant was dyspnœa; this symptom at first sight struck you with the idea that you had to deal with some grave pulmonary disease, but on careful and daily examination no rale was audible, the respiratory murmur being normal but accelerated—this state going on for a variable period, the shortest being eight days, the longest nineteen—the child dying quickly after the supervention of pneumonia or diptherite. The remarkable dyspnœa which formed so prominent a symptom in this form of sequela, bore a striking analogy to that observed in the influenza of the adult; in that disease, as in the sequela we are describing, the dyspnœa is out of all proportion to the implication of the lung, and seems at first to exist without any evidence of its implication. To carry the analogy still further, we find that a formidable and sometimes fatal implication of the lung frequently results.

From a consideration of these cases, we would infer that the typhoid state was produced by the rubeolous poison circulating in the blood, and that its principal action was upon the pneumogastric,

as illustrated by the dyspnœa, and subsequent changes in parts supplied by this nerve.[1]

It will doubtless be considered by many that the dyspnœa may be satisfactorily accounted for in a more ordinary way, namely, as resulting from the presence of fluid in the bronchi and air cells; but our observations in these cases tend to prove that effusion into the vesicular structure of the lung was never found till shortly before death, while the dyspnœa was a constant symptom. Whatever, then, may have been the cause of this symptom, whether from a purely nervous origin or associated with a loaded state of the pulmonary capillaries, we cannot assert, but of this much we are certain, that the dyspnœa, however produced, did not result from effusion into the structure of the lung. That the dyspnœa during life, and the pathological changes observed after death, resulted from congestion of the lung, *per se*, we can scarcely think probable. If we consider the terminations of this sequela, we undoubtedly find pneumonia frequently supervening—a result which might be attributable to a long continued congestion. But again, we find that in others diptherite was the fatal complication; so that although all the cases had a dyspnœa, which could be produced by perversion of nervous function in the pneumogastric, yet all, as would be the case, were congestion of the lung the cause, had not the lung implicated, while the implication of parts under the influence of the pneumogastric was always observed.

We now briefly revert to the state of the urine in these cases, for we consider that its relation to the sequelæ just described, is far from uninteresting. The published observations upon the state of the urine in measles, as far as we have been able to find, are very few; indeed, they seem to be comprised in the observations of Becquerel and Simon.[2] On the characters of this urine Simon writes thus:—"In most cases it more or less resembles the inflammatory type; it is red (as in inflammatory measles), acid, and sometimes jumentous (turbid), as in gastric measles, or deposits a mucous sediment during the course of the morning (as in catarrhal measles)." Becquerel states, as the result of his observations, that the urine is generally inflammatory at the commencement of the febrile period. It becomes very dark, and of high specific gravity, and frequently deposits a sediment of uric acid; a small quantity of albumen was found in a few of these cases. During the eruptive period the character of the urine changes; if the eruption is slight, and there is not much fever, it resumes the normal type; if the contrary is the case, the urine

[1] The recent observations of Bernard upon animals, after division of the pneumogastric in the neck, present many analogies to this form of sequelæ. The inspirations were exaggerated, and the animals died of pneumonia. The older experiments of John Reid are similar with regard to the termination of such experiments, but different concerning the respiration, which he found to be, with a solitary exception, diminished.

[2] Simon's Animal Chemistry, vol. ii. p. 269. Sydenham Soc. Translation.

retains the inflammatory appearance. Becquerel did not meet with any case in which the urine was turbid or sedimentary towards the close of the eruptive stage. "During the period of desquamation and of convalescence, the urine either returns at once to the normal state, or continues turbid and sedimentary for some time, or becomes pale, clear, and anæmic." These observations, however correct as to measles generally, were not found corroborated in some respects in this epidemic. The observations made as to the state of the urine during the stage of eruption were similar to our own, with this exception, that in all the urine deposited, urates instead of uric acid. The observations of Becquerel, that the urine did not in any case become sedimentary towards the end of the eruptive stage, was not found in these cases; for in proportion to the subsidence of the eruption in normal measles, was the increase of the deposit, and in all it was well marked. The presence of albumen is only noticed as an occasional occurrence during the premonitory stage; no further mention being made of it. We now in conclusion dwell shortly upon the probable cause of albuminuria in the disease, and its relation to the sequelæ which followed it. We found that in cases where the eruption appeared quickly, albuminuria occurred, a fact which seems easily accounted for, if we consider that the functions are suspended quickly, if a large excretory surface giving off water and various salts, and furnishing in its whole extent an albuminous secretion which forms cuticle, as well as the secretion found in the sebaceous follicles. We have probable evidence in support of the idea that the kidney may take upon it the functions which are thus temporarily suspended, especially when we consider the intimate connection which exists between the two organs: the absence of albuminuria during the progress of the eruption may be accounted for by the system accommodating itself to the change.

We found that albuminuria was again present on the recedence of the rash, and that the quantity was greatest in those cases where the rash faded quickly, and the recoveries most perfect. This occurrence is probably owing to the sudden disappearance of a quantity of blood from the surface to the internal organs generally, but to the kidneys in particular, as superadded to them is the function of blood depuration, which at this period commences. As it appears to us, the presence of albuminuria is only of service as pointing to the perfect elimination of the morbid poison; its absence, either that other organs are taking this office upon them, or that it is not taking place at all.

When we consider the ordinary effects of cold applied to the skin, we think that three organs are most prone to suffer,—the lung, the kidney, and the intestine. In inflammatory measles we find that the depurating function of the kidney is impeded, as seen by the absence of albuminuria, and that in consequence, this function is taken by the lung or intestine, with this important exception, that while in the kidney the depurating process, so to speak, seems the

normal one, in the case of the lung and intestine, the substance presented to them for excretion is one essentially different from that they are usually called upon to dispose of. It is worthy of remark that amendment of the diseases of the respiratory and digestive system was found to follow, or be coincident with the appearance of albuminuria. The facts observed with regard to these cases would lead to the probable inference that the sequelæ arose from the function of the kidney being suspended, and its functions adopted by the lung and intestine.

The facts advanced upon the condition of the urine in the typhoid form of sequela, do not warrant any decided inferences; the small quantity taken in connection with the imperfect eruption, would lead us to believe that the poison was circulating in the blood, and that efforts at elimination were probably not made by any organ. It will doubtless be considered that such views of the state of the urine in this disease warranted the trial of that class of remedies called "renal depurants," of which potash and its salts are the principal; but these observations were collecting during the progress of the disease, and their probable value only determined upon its decline; should it ever fall to my lot to witness another epidemic, their utility would be quickly tested.

In conclusion, let it be remembered that we have not here attempted to give a history of measles, but that of an epidemic, and that the deductions are made with reference to it alone, our aim having been to describe the peculiarities observed, which we feel confident will not be found dissimilar to those perceived by others who have seen this epidemic on a large scale.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

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ACCOMMODATIONSVERMÖGEN

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ACCOMMODATIONSVERMÖGEN

DER

AUGEN.

NACH Dr. A. CRAMER ZU GRONINGEN UND PROF. DONDERS ZU UTRECHT.

VON

Dr. C. H. SCHAUENBURG,
Docenten an der Universität zu Bonn.

Mit Abbildungen.

LAHR, 1854.
VERLAG VON J. H. GEIGER. (M. SCHAUENBURG.)

Die holländische Gesellschaft für Wissenschaften zu Harlem hatte die Preisfrage aufgestellt: **„worin liegt der Grund und die Ursache des Accommodationsvermögens der Augen?“** — Am 21. Mai 1852 wurde die im November 1851 eingesandte Beantwortung des Herrn A. Cramer, med. chir. et art. obstetr. Dr. in Groningen als der goldnen Medaille und der Prämie würdig erkannt und gekrönt. Im Jahre 1853 erschien die Preisschrift in Harlem bei den Erben Loosjes, durch Budge's und Anderer weitere Untersuchungsresultate bereichert. Eine ausführliche Besprechung der Cramer'schen Schrift von Professor Donders in Utrecht hat kürzlich die Presse verlassen und bringt so zahlreiche und vortreffliche Erörterungen und Vervollständigungen des Discussionsgegenstandes, dass eine Bearbeitung desselben für das deutsche ärztliche Publikum hinreichend gerechtfertigt erscheint.

Cramer sagt zuerst in der Einleitung seiner Schrift.

Von jedem Punkte eines vor dem Auge befindlichen leuchtenden oder erleuchteten Gegenstandes geht ein Lichtkegel aus, dessen Strahlen, beim Auffallen auf die Cornea nur zum Theil zurückgeworfen, übrigens durchgehen und nach der Senkrechte zu gebrochen werden. Diese Strahlen verfolgen nun ihren Weg durch den humor aqueus und die Pupille bis an die Linse, wo sie eine noch grössere derartige Brechung erleiden,

treten weiter durch die Linse in das corp. vitreum, das ihren Grad von Convergenz kaum etwas vermehrt, und vereinigen sich auf der Retina zu einem verkleinerten, umgekehrten Bilde der Gegenstände, von denen der Lichtkegel herkommt. Wie bei einer Linse der Focus seine Stelle verändert, wenn der Abstand der Gegenstände, deren Bilder im Focus entstehen, ein anderer wird, so muss auch, wenn man das Auge als aus aneinandergefügten Linsen bestehend betrachtet, die Stelle, an der das Bild im Auge entsteht, je nach dem Abstande der Gegenstände vom Auge verschieden sein. Wenn also im Auge bei einem bestimmten Abstande des Gegenstandes das Bild auf einer Stelle der Retina entsteht, so muss bei einer grössern Entfernung des Gegenstandes vom Auge das Bild vor die Retina, bei stärkerer Annäherung vor das Auge hinter die Retina fallen, wobei dann auf der Retina selbst nur ein diffuses Bild entsteht. Um eine scharfe Anschauung zu erhalten, ist es nöthig, dass die Bilder auf einer bestimmten Stelle der Retina entstehen, was nach Obigem, ohne Veränderung im Auge, nur bei einer gewissen Entfernung der Gegenstände vom Auge möglich sein würde. Das Sehvermögen müsste also, wenn keine Veränderung im Auge möglich wäre, sehr unvollkommen sein, sie ist aber sehr vollkommen durch die Accommodation.

Die Berechnung des Verhältnisses, in welchem der verschiedene Abstand der Objecte von dem Auge das im Auge entstehende Bild bezüglich seiner Stelle verändert, ist bei dem gegenwärtigen Zustande der Wissenschaft kaum möglich, da der Krümmungsgrad der Oberflächen der den optischen Apparat des Auges zusammensetzenden Theile, also auch das Brechungsvermögen dieser Augenmedien und der Abstand der Theile

untereinander noch nicht hinreichend erkannt sind. Petit, Young, Dr. W. Sömmering, Brewster, Chossat, Treviranus, Krause, Engel und Andere haben allerdings Beiträge geliefert, ohne dass indess die Untersuchung zu einem sichern Abschluss gelangt wäre. Im Gegensatz hierzu hat man wiederholt durch Berechnung zu bestimmen versucht, in wiefern der Platz, auf dem Bilder vor dem Auge befindlicher Gegenstände entstehen, nach Massgabe des Abstandes dieser Gegenstände ein verschiedener ist. Es war ziemlich natürlich, dass man auf die Weise zu widersprechenden Resultaten gelangte. So behauptete Treviranus [1]), dass das Auge zufolge der nach Innen immer zunehmenden Dichtigkeit der Linse bereits für das Sehen auf die verschiedensten Abstände accommodirt sei und glaubte, dies durch ein mathematisches Theorema beweisen zu können. Zu übereinstimmenden Resultaten führten Sturm [2]) geometrische Untersuchungen, die er über die brechenden Augenmedien anstellte. Auch er glaubte, dass das Auge bereits für die verschiedenen Abstände accommodirt sei. Durch Kohlrausch [3]) ist Treviranus übrigens auch mathematisch hinreichend widerlegt.

Besser als durch Berechnung kommt man besonders hinsichtlich der Frage, ob und wieweit das Bild seinen Platz verändert, auf experimentalem Wege zu einiger Sicherheit. Magendie [4]), der an Thieren experimen-

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[1]) Beiträge zur Anatomie und Physiologie der Sinneswerkzeuge etc. 1828. I. Heft.

[2]) Sturm, über die Theorie des Sehens, Poggendorff's Annalen.

[3]) Kohlrausch, über Treviranus Ansichten vom deutlichen Sehen in der Nähe und Ferne. Rinteln. 1836. —

[4]) Magendie, Précis élémentaire de Physiologie. I. p. 73.

tirte, glaubte an einem Kaninchenauge wahrzunehmen, dass alle Gegenstände, in welchem Abstande sie sich auch vor dem Auge befänden, sich gleich deutlich auf der Sclerotica abzeichneten. Das Auge wäre also schon für alle Abstände accommodirt. Ebenso behauptet Ritter [2], dass sich auf der Sclerotica eines todten Auges ein ebenso deutliches Bild von fernen als von nahen Gegenständen bilde. Haldat [3] und Adda [4] gingen noch weiter und wollten beobachtet haben, dass durch ein frisches Ochsenauge sowohl divergente als parallele und convergente Strahlen sämmtlich in ein und denselben Focus vereinigt würden. Hueck [5] berichtet dagegen, dass er an den Augen von Vögeln, namentlich von Schnepfen, nur dann deutliche Bilder auf der Sclerotica erhalten habe, wenn er die Gegenstände vor dem Auge in eine bestimmte Entfernung gebracht habe.

Mehr Beweiskraft hat der Versuch Volkmann's [6] mit einem weissen Kaninchen, den er mit Zuhülfenahme des Scheiner'schen Versuches anstellte. Er sah, dass bei einer bestimmten Entfernung eines Lichtes vom Auge nur ein Bild auf der Sclerotica entstand, dass aber Doppelbilder entstanden, sobald sich das Licht näher beim Auge oder ferner von ihm befand. Hielt er das Licht nur einige Zoll vom Auge entfernt, so sah er Doppelbilder, von denen das rechte verschwand, wenn er die rechte Oeffnung im Kartenblatt schloss; ebenso verschwand das linke bei Verschliessung der linken Oeffnung. Bei der Entfernung des Lichtes vom

[2] Graefe und Walther's Journal 1832, Bd. 8. p. 347.

[3] Comptes rendus, 1842.

[4] Annales de Chimie et Physique. III. Sér. T. XII. p. 94.

[5] Hueck: diss. de mutationibus oculi internis. Dorp. 1826. p. 17 und: die Bewegung der Crystalllinse. Leipzig, 1841.

[6] Neue Beiträge zur Physiologie des Gesichtssinnes. 1836, p. 109.

Auge näherten sich die Bilder einander, bis sie zuletzt ineinander fielen. Mayer [1] sah, dass bei einem Ochsenauge, von dem die Choroidea und Sclerotica an der Hinterseite abpräparirt waren, das auf der Retina entstandene Bild um $1\frac{1}{3}$—$1\frac{1}{4}$ Par. Lin. seinen Platz veränderte, wenn ein vorgehaltenes Licht von einem entfernten Abstande auf 7 Par. Zoll vor das Auge gebracht wurde. Aber auch Mayer's Methode führt zu keiner vollen Sicherheit, während die Volkmann's den unumstösslichen Beweis liefert, dass das Kaninchenauge nicht für verschiedene Abstände accommodirt ist.

In Bezug auf das menschliche Auge kommen die meisten Widersprüche vor, die in der Cramer'schen Schrift mit grosser Ausführlichkeit beleuchtet und widerlegt werden.

Cramer berichtet pag. 12 über den von Helmholtz [2] gelieferten unwiderleglichen Beweis, dass wirklich ein Accommodationsvermögen der Augen und zwar in einer Veränderung des Refractionszustandes der brechenden Augenmedien bestehe.

Helmholtz benützte zu dem Beweise seinen Augenspiegel. Wenn man nämlich durch Jemand einen Gegenstand fixiren lässt, der sich in gleichem Abstande vom Auge befindet, als in welchem die als Lichtquelle für den Spiegel dienende Kerze gebracht ist, so sieht man mit Hilfe des Spiegels die Gefässe der Retina und das Bild z. B. eines vor die Flamme gehaltenen Fadens. Bringt man nun den Faden näher an das Auge, oder entfernt man ihn mehr, so wird er undeutlich und verschwindet endlich, während die Ge-

[1] Prag. Vierteljahrschrift. 1850. 4. Bd. Ausserord. Beilage.

[2] Beschreibung eines Augenspiegels etc. 1851.

fässe gleich sichtbar bleiben. Ebenso verschwindet der Faden aus dem Gesichte zugleich mit den Retinagefässen, wenn man nach einem mehr oder weniger entfernten Punkte sehen lässt, als in welchem sich der Faden vor der Flamme befindet.

Das Auge besitzt also das Vermögen, den Focus der Lichtstrahlen sowohl von entfernten als von nahen Gegenständen auf eine bestimmte Stelle der Netzhaut fallen zu lassen. Ueber den Grund und die Ursache dieses Vermögens, also des Accommodationsvermögens, hatte man seit Jahrhunderten vergebens geforscht und gesonnen. Kepler warf in seiner Dioptrice seu demonstratio eorum, quae visui et visibilibus propter conspicilla non ita pridem inventa accident. Aug. Vindelicor. 1611. prop. 26. zuerst die Frage auf, worin dasselbe bestehe. Donders sagt, dass sie jetzt durch Cramer's Arbeit hinreichend beantwortet sei.

In einer kurzen Mittheilung berichtete Cramer schon früher [1]), dass es ihm geglückt wäre, eine Formveränderung der Linse bei der Accommodation für die Nähe wahrzunehmen. Donders, der Gelegenheit hatte, sich von der Richtigkeit dieser Beobachtung zu überzeugen, beeilte sich, über diese Entdeckung zu berichten [2]). In seinem neuesten Berichte, dem wir hier folgen, spricht er ausführlich und vervollständigend über den Gang und die Resultate der Untersuchungen, auch desshalb, weil er mit Recht fürchtet, das Cramer'sche Werk möge nicht in Vieler Hände kommen.

So lange man keine innere Veränderung auffinden, noch auch eine befriedigende Hypothese über einen vielleicht statthabenden Mechanismus entdecken konnte,

[1]) Tijdschrift der Maatschappij tot Bevordering der Geneeskunst. Jaarg II. p. 99.

[2]) Nederl. Lancet. 3. Ser. 1. Jaarg. p. 529.

beschränkte man sich auf den Beweis, dass sich das Auge überhaupt nicht zu verändern brauche, um sich auf Objecte von verschiedenem Abstande zu richten. Cramer beweist zuerst die Nothwendigkeit einer solchen Veränderung im inneren Auge.

Engel hat kürzlich gesagt, dass die isolirte frische Linse von Gegenständen in verschiedenem Abstande ein vollkommen deutliches Bild fast in demselben Punkte ergeben habe. Mayer war schon dagegen aufgetreten und hatte richtig bemerkt, dass die Verschiedenheit hinsichtlich der Stelle des entstehenden Bildes sehr gering sein müsse, wenn die Linse sich in der Luft befinde, dass sie viel grösser sein müsse, wenn die Strahlen aus der Linse in den Glaskörper träten, dessen Lichtbrechungscoefficient nicht so verschieden von dem der Linse sei.

Cramer stellte Proben mit frischen Menschenaugen an, die er, nachdem er an der Hinterseite Sclerotica, Choroidea und Retina entfernt hatte, mit der Cornea nach unten, auf die Oeffnung in der Objecttafel eines Mikroskopes brachte, um wahrzunehmen, auf welcher Stelle die Bilder von Gegenständen von verschiedenem Abstande, von einem Planspiegel zurückgeworfen, entständen. Er bediente sich 80maliger Vergrösserung. Um sich von einer unwillkürlichen Mitwirkung seines eigenen Accommodationsvermögens unabhängig zu machen, legte er ein Haar über den Glaskörper, welches er gleichzeitig mit dem Bilde von verschieden weit (— 200 Schritt) entfernten Gegenständen deutlich wahrnahm. Nun fand er, dass bei einem Abstande von 43—37 Centim. das Bild einer Nadel nicht mehr deutlich gesehen wurde, es sei denn, dass er den Abstand des Objectivs vergrösserte, wobei das Haar undeutlich wurde.

Hannovers ungereimte Meinung, dass sich die Linse in ihrer Kapsel bewege, ist nur beiläufig zu erwähnen. Aber es ist ohne Zweifel, dass Pemberton, Camper, Hunter, Young, ferner Purkinje, Graefe, Th. Smith nach dem Vorgange von Cartesius an eine Formveränderung der Linse, in der Cramer den Grund des Accommodationsvermögens findet, gedacht haben, ohne diese Meinung mit Beweisen zu unterstützen.

Weber und Ludwig sprachen von einer Vorwärtsbewegung der vordern Linsenfläche nach galvanischer Reizung der inneren Muskelgruppen des Auges. Sie nahmen an, die ganze Linse bewege sich dabei, sie dislocire sich nach vorn.

Cramer beweist nun, dass bei der Accommodation die vordere Linsenfläche ihre Form verändert.

Max Langenbeck hat schon im Jahre 1849 (in seinen klinischen Beiträgen aus dem Gebiete der Chirurgie und Ophthalmologie, Göttingen) auf Grund der Beobachtung mit dem blossen Auge, dass sich das Bild einer Flamme auf der vordern Linsenfläche hinsichtlich seiner Gestalt als seines Sitzes verändere, ausgesprochen, dass das Accommodationsvermögen auf einer veränderten Wölbung der vordern Linsenfläche beruhe. Donders, der sich mit dem unbewaffneten Auge von der Richtigkeit dieses Ausspruches nicht überzeugen konnte, prüfte ihn (mit Prof. v. Rees und Dr. Berlin) vermittelst des Kathetometerglases. Er kam zu keiner sicheren Ueberzeugung, hielt den Weg aber für den richtigen, um zu ermitteln, ob wirklich eine Formveränderung zu Stande käme. In dem Seine schrieb er in der Ned. Lanc. 1849: „für die Lehre von der Accommodation halte ich es für sehr gewichtig, dass die beiden durch die Linsenflächen geformten Bilder,

was ihre Grösse als was ihre Tieflage betrifft, bei verschiedenem Accommodationszustande mit einem Vergrösserungsglase untersucht werden. Unter günstigen Umständen muss diese Untersuchung zu erheblichen Resultaten führen."

Langenbeck versäumte, seinem Ausspruche Eingang in die Wissenschaft zu verschaffen, wobei ihm auch die unglückliche Erklärung dieses Mechanismus durch einen improvisirten *m. compressor lentis* im Wege stand. Der erste Gedanke gehört aber ihm.

Cramer's Methode, um den fraglichen Gegenstand mit zweifelloser Richtigkeit zu beweisen, ist folgende.

Erstens lässt er das Licht von der einen Seite einfallen und beobachtet die von der Hornhaut, der Vorder- und Hinterfläche der Linse zurückgeworfenen Bilder auf die gewöhnliche Weise von der anderen Seite. Am Rande der Pupillarfläche sieht man nun das von der Cornea zurückgeworfene Bild (Fig. I, a); meistens ungefähr in der Mitte derselben Fläche das tiefliegende von der Vorderfläche der Linse zurückgestrahlte (I, b); an der a gegenüberliegenden Seite das auf der Hinterfläche der Linse oder auf der Vorderfläche des Glaskörpers entstandene umgekehrte kleine Bildchen c. Zum Wahrnehmen der Veränderungen im Stande dieser Bildchen bei der Accommodation ist es erforderlich, dass die Richtung der Gesichtsachse dieselbe bleibt und dass das Licht in dem beobachteten Auge unverändert denselben Platz behält, während es dagegen wünschenswerth ist, dass das beobachtende Auge die Bilder vergrössert sieht. Um diese Bedingungen zu erfüllen, construirte Cramer ein Instrument, dem er den Namen Ophthalmoscop gab. Die wesentlichen Bestandtheile desselben sind:

1) eine kurze kegelförmige Büchse mit zwei Seitenöffnungen, an dessen breites Ende das zu beobachtende Auge gebracht wird;
2) eine Flamme mit einem Diaphragma, die ihr eine bestimmte Form gibt und durch eine der genannten Seitenöffnungen Licht in das zu beobachtende Auge wirft;
3) ein bewegliches Mikroskop, welches auf die zweite Seitenöffnung gerichtet ist und durch welches das beobachtende Auge die Reflex-Bilder wahrnimmt.

Man lässt nun das zu beobachtende Auge sich für den Kreuzungspunkt zweier ganz in der Nähe ausgespannter Fäden accommodiren und sieht die Bilder b

Fig. I.

Fig. II.

und c (projicirt auf eine Fläche, die lothrecht auf der Gesichtsachse des beobachtenden Auges steht) fast unmittelbar bei einander (cfr. Fig. II). Lässt man nun aber in derselben Richtung über die Fäden hinaus [1]) in die Ferne sehen, dann entfernt sich das

[1]) Man ist auf diese Weise niemals sicher, vollkommen dieselbe Richtung zu erhalten, weil bei dem Sehen auf grösseren Abstand das Fadenkreuz sich mehrfach darstellt (polyopia monocularis). Für scharfe Messungen wird es desshalb nöthig sein, erst durch eine kleine, mitten vor die Pupille gehaltene Oeffnung zu sehen, um das Fadenkreuz und einen entfernteren Punkt in entsprechende Lage zu bringen und nun abwechselnd das Kreuz und den Punkt zu fixiren.

Bild b von c und stellt sich fast mitten zwischen a und c (cfr. Fig. I), während a und c unverändert ihren Platz behalten. Bei dieser Platzveränderung wird das Bild b zugleich grösser und matter. Die Accommodation für die Nähe bringt es sofort wieder nahe bei c und macht es zugleich wieder kleiner und heller.

Cramer stellte diese Beobachtungen bei 20maliger und selbst bei 10maliger Vergrösserung an.

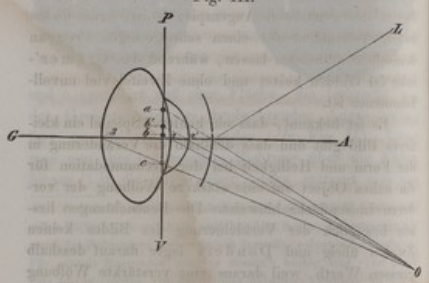
Donders, der Gelegenheit hatte, ein von Mechanicus Epkens in Amsterdam verfertigtes Cramer'sches Ophthalmoscop zu untersuchen, fand es in mehrfacher Hinsicht unpraktisch. Er liess mit seinem von Epkens ausgeführten Augenspiegel ein Ophthalmoskop in Verbindung bringen, vermittelst dessen nicht allein die Wahrnehmung der von Cramer angeführten Erscheinungen keine Schwierigkeit hat, sondern auch solche Messungen bewerkstelligt werden können, als nöthig sind, um zu berechnen, um wieviel die Vorderfläche der Linse nach Vorn getreten ist. Wer im Besitz des genannten Augenspiegels ist, kann dieses zweite Instrument für einen sehr geringen Preis an demselben anbringen lassen, während das Cramer'sche 56 Gulden kostet und ohne Zweifel viel unvollkommener ist.

Es ist bekannt, dass ein hohlerer Spiegel ein kleineres Bild gibt und dass desshalb die Veränderung in der Form und Helligkeit bei der Accommodation für ein nahes Object auf eine stärkere Wölbung der vorderen Linsenfläche hinweist. Die Beobachtungen liessen bezüglich der Verkleinerung des Bildes keinen Zweifel übrig und Donders legte darauf desshalb grossen Werth, weil daraus eine verstärkte Wölbung offenbar bewiesen ist. Die Lageveränderung des Bildes ist bei dieser Beobachtung aber noch mehr für die

Messung zugänglich, als die Veränderung hinsichtlich der Grösse des Bildes. Die Lageveränderung an und für sich selbst gibt uns wohl nicht das Recht, auf eine veränderte Wölbung der vorderen Linsenfläche zu schliessen, aber sie weist doch unwiderleglich nach, dass die vordere Linsenfläche nach Vorn getreten ist, sei es nun durch stärkere Wölbung oder durch Verschiebung der ganzen Linse.

In Bezug auf diesen Punkt kommt es ganz besonders auf deutliche und strenge Beweisführung an. Man sieht bald ein, dass die Lageveränderung des Reflexbildes in einer veränderten Form oder Lage der reflectirenden Fläche begründet ist, aber es bedarf der Erklärung, dass und wesshalb die bei der Accommodation für die Nähe wahrgenommene Lageveränderung des Bildes b nach a hin eine Vorwärtsbewegung der reflectirenden Oberfläche anzeigt. Diese nähere Entwickelung soll hier folgen.

Fig. III.



Wenn sich in L (Fig. III) ein leuchtender Punkt befindet, dann müssen die bei 1 auf der Cornea in

derselben Fläche, unter gleichem Winkel mit der Gesichtsachse G A reflectirten Strahlen das in O befindliche Auge erreichen, das Reflexbild muss also in der Richtung O 1, auf die Pupillarfläche P V projicirt, als a gesehen werden. Von demselben leuchtenden Punkte L müssen auch die bei 2 von der vorderen Linsenfläche zurückgeworfenen Strahlen das Auge O erreichen und sich in der Richtung O 2 als b darstellen. Endlich müssen die von L kommenden und von der hinteren Linsenfläche bei 3 zurückgeworfenen Strahlen in der Richtung O 3 das Auge erreichen und in c wahrgenommen werden. Auf diese Weise sieht man die Bilder als a b c in der Pupillarfläche (cfr. Fig. 1). Tritt nun die Vorderfläche der Linse bei der Accomodation für die Nähe bis nach 2' vorwärts, dann werden auch die bei 2' zurückgeworfenen Strahlen O erreichen und das Reflexbild wird nicht mehr in b, sondern in der Richtung O 2' in b' wahrgenommen, — also bei der Vorwärtsbewegung der vorderen Linsenoberfläche näher bei a. Wir haben hierbei vorausgesetzt, dass das Licht in demselben Winkel mit der Gesichtsachse einfällt, in welchem das Auge das reflectirte Licht wahrnimmt, — eine Voraussetzung, die Cramer in seiner Untersuchung versäumt hat. Donders behauptet, dass sein mit dem Augenspiegel verbundenes Werkzeug Gelegenheit gebe, beide Winkel genau zu bestimmen und also gleich zu stellen. In dieser Art also kann man die Lageveränderung wahrnehmen, ebenwohl ganz so, wie Cramer [1]) es will. Seine Annahme, dass die vordere

[1]) Der Stand der Bilder ist bei der Beobachtung unter gleichen Winkeln (c. 35°) und bei verschiedenen Objecten nicht ganz derselbe. Zuweilen ist er, wie in Fig. I und II, wo er der in Fig. III entspricht. Nicht selten ist er, wie bei Cramer (Fig. 7 u. 8), der Art, dass b im Zustande der Ruhe nahe bei c steht und bei der

Linsenfläche bei der Accommodation für die Nähe nach Vorn trete, war also wohlbegründet. Wir dürfen aber nicht versäumen, daran zu erinnern, dass bei unserer Beweisführung der Einfluss der Cornea unberücksichtigt geblieben ist, da die Strahlen nicht in dem Krümmungsradius durch diese Membrem treten, eine für die einfallenden und ausfallenden Strahlen gleiche Abweichung zur Folge hat, und wohl eine grössere, wenn die Vorderfläche der Linse der Cornea näher gerückt ist. Daraus geht aber nur hervor, dass die Lageveränderung grösser erscheint (bei Vergl. von Fig. I und II), als sie wirklich ist (Fig. III von b zu b'), aber nicht, dass sie in anderer Weise statthat. Die Thatsache bleibt also mit derselben Sicherheit bewiesen.

Diese Lageveränderung des vordersten Poles der Linse kann nun abhängen: entweder von einer Veränderung in der Wölbung der Vorderfläche oder von einer Lageveränderung der Linse oder von beiden zugleich. Cramer nimmt eine Veränderung in der Wölbung an. Zuerst sieht er in dem Heller- und Kleinerwerden einen Beweis, dass die Wölbung der Linse wirklich zunimmt. Sodann nahm er keine Lageveränderung des von der hintern Linsenfläche zurückgeworfenen Bildes c wahr, woraus er folgerte, dass diese hintere Fläche weder bezüglich der Lage noch

Accommodation für die Nähe ungefähr in die Mitte zwischen a und c rückt. Bei diesem Stande liegt die vordere Linsenfläche sicher weiter vor der Cornea, als in Fig. I und II, — ein Abstand, der sich nach Donders Beobachtungsmethode ohne Schwierigkeit berechnen lässt. Soviel scheint klar, dass die Lageveränderung von b keineswegs der Verschiedenheit der Accommodation und der Grösseveränderung des Bildes entspricht, so dass es höchst wahrscheinlich ist, dass die Linse unter gewissen Umständen nach Hinten ausweicht. Der Untersuchung bietet sich hier noch ein weites Feld.

der Wölbung eine Veränderung erleide. Diese Folgerung scheint sehr gewagt. Denn wenn auch angenommen wird, dass diese Fläche wirklich keine der beiden genannten Veränderungen erleidet, so muss doch die veränderte Wölbung der vorderen Linsenfläche eine kleine Veränderung in der Richtung der bei 3 zurückgeworfenen Strahlen, die das Auge O wahrnimmt, zur Folge haben und das Bild also wirklich eine kleine Lageveränderung zeigen. Fand also nicht die geringste Lageveränderung statt, dann würde man hieraus gerade folgern müssen, dass eine geringe Veränderung um der hinteren Linsenfläche zu Stande kam, welche die sonst nothwendig gewesene Lageveränderung compensirte. Statt so [1]) zu folgern, scheint es richtiger zu sein, anzunehmen, dass die Cramer'schen Beobachtungen nicht hinreichend genau waren.

Können wir also in Cramer's Annahme nicht einstimmen, „dass sich die hintere Fläche der Linse weder hinsichtlich der Stelle noch der Wölbung verändere", so geben wir doch gerne zu, dass die Veränderung gering genug ist, um ihn zu der Annahme zu berechtigen, dass die Lageveränderung des Bildes hauptsächlich einer Veränderung des Krümmungsradius der vorderen Fläche zuzuschreiben sei, keineswegs bloss einer Vorwärtsbewegung dieser Fläche, die von einer Vorwärtsbewegung der hinteren Fläche begleitet sein müsste.

Dass die Wölbung der vorderen Fläche eine Ver-

[1]) Es erscheint fast undenkbar, dass ein weicher, von elastischer Haut umgebener Körper seine Form durch Druck auf eine Fläche sollte verändern können, ohne dass zugleich auch die andere Fläche mehr oder weniger verändert würde.

änderung erleidet, hat Helmholtz [1]) kürzlich durch ein etwas bestimmteres Verfahren näher dargelegt. Er schlug denselben Weg ein, den Senff gewählt hatte, um den Krümmungsradius der Cornea zu bestimmen. Er besteht in der Messung des Abstandes der zwei Spiegelbilder, die von zwei zur Seite befindlichen Lichtern auf der vorderen Fläche der Linse entstehen. Auf diese Weise fand er, dass der Krümmungsradius bei dem Sehen auf Abstand ungefähr ebenso gross ist, als der auf dem todten Auge = 10—12 mm., während er bei der Accommodation für die Nähe, bei welcher sich die Spiegelbilder einander sehr nähern, nur ungefähr die Hälfte beträgt. Ferner bestimmte Helmholtz unter gleichzeitiger Beachtung des Einflusses der Cornea, als deren Brennpunkt unter Wasser er 33—34 mm. fand, wieviel die Iris nach Vorn gerückt wird und fand hierfür $\frac{1}{3}$ mm. Die Vorwärtsbewegung der Iris hatte Donders schon seit längerer Zeit mit Hülfe des Orthoscopes ohne Schwierigkeit beobachtet. Sie war auch schon früher ohne Orthoscop von Hueck untersucht und bestimmt worden. Auch Cramer bringt sie, ebenso wie die Versuche von Weber und Ludwig, mit den Ergebnissen seiner Untersuchungen in Verbindung. Von einer noch näheren Bestimmung, als die von Helmholtz gegebene ist, dürfen wir uns aber nicht zu viel versprechen, weil sie uns nicht allein Nichts lehrt in Bezug auf die Wölbung des durch die Pupille vorgetretenen Theiles der vorderen Linsenoberfläche, sondern auch selbst hinsichtlich der Lageveränderung der Iris keine Auskunft gibt, weil ja die Pupille bei der

[1]) Mon. Ber. der Königl. Preuss. Acad. der Wissenschaften zu Berlin, Febr. 1853, S. 137 u. f.

Accommodation für die Nähe sich verengert und die Iris sich desshalb auf die Linse schieben muss, auch wenn die Oberfläche der Linse, auf der sie ruht, keinerlei Veränderung erleidet [1]).

Helmholtz sah nach der von ihm befolgten Methode auch keine Veränderung weder in der Stellung noch in der Grösse der Bilder, die von der Hinterfläche der Linse zurückgeworfen werden, und er schloss hieraus, dass sich die Form dieser hinteren Fläche nicht merkbar verändere. Die Bedenken, die oben gegen die Annahme Cramer's ausgesprochen wurden, sind durch die vorläufigen Mittheilungen von Helmholtz nicht aus dem Wege geräumt. Jedenfalls ist die Helmholtz'sche Annahme weniger exclusiv.

Nachdem Cramer ausgesprochen hat, dass die vordere Fläche der Linse durch die Accommodation für die Nähe eine stärkere Wölbung erhält, untersucht er, ob noch andere Momente bei der Accommodation in Betracht kommen. Anderweitige Veränderungen in der Linse werden von ihm geläugnet. Auch die Veränderung in der Wölbung der Cornea, an die nach den Senff'schen Untersuchungen wohl Niemand mehr glaubte, werden auf Grund eigener genauer Untersuchung verworfen. Endlich will Cramer auch beweisen, dass der Abstand zwischen der Hinterfläche der Linse und der Netzhaut keine Veränderung erleidet, dass also, angenommen, dass die Hinterfläche der Linse bei gleicher Form gleiche Stelle einhält (?), die Gesichtsachse bei der Accommodation für die Nähe

[1]) Helmholtz behauptet, dass keine Lageveränderung der Iris nach Vorn wahrgenommen wird, wenn die Pupille sich allein in Folge einfallenden Lichtes verengert. Dies soll zu der Hypothese hinführen, dass bei der Accommodation auf Abstand eine hintere Augenkammer bestehe.

nicht verlängert werde. In der Beweisführung ist er aber nicht bestimmt. Erstens behauptet er, dass, wenn man das Auge für das Sehen in der Nähe accommodirt hält, zwei einander bedeckende, indirekt sichtbare Objekte bei dem Accommodationszustande des Auges für die Ferne nicht auseinander gehen. Dies würde, wenn es wahr wäre, allein beweisen, dass der Kreuzungspunkt der Richtungslinien unverändert bleibt, wird aber eine Veränderung dieses Kreuzungspunktes angenommen, insofern er als ein Punkt betrachtet werden darf, und woran wohl nicht zu zweifeln ist, so muss die Behauptung für unrichtig gehalten und wiederholt gesagt werden, „dass die Wahrnehmung in Bezug auf das Bedecken von indirekt gesehenen Gegenständen niemals scharf ist.“ — Von nicht grösserem Gewichte sind die übrigen Gründe. Indem er die entoptische Untersuchung zu Hülfe nimmt, sagt Cramer nämlich, „so sieht man, dass die Streifen und Flecken in der Linse bei der Accommodation des Auges für entferntere Gegenstände nicht schmaler, kleiner und deutlicher und ebenso wenig bei der Accommodation für die Nähe breiter, grösser und undeutlicher.“ Aber gerade von der veränderten Wölbung der Linse kann man viel eher einen Einfluss auf diese entoptischen Bilder erwarten, die allerdings schwer zu constatiren ist, als von einer Verlängerung der Gesichtsachse. Wenn Cramer sich endlich auf die Anwendung der Donders'schen Methode zur Bestimmung des Sitzes von kleinen Verdunkelungen etc. beruft und sich aus dem unveränderten Abstand der entoptischen Doppelbilder überzeugt haben will, dass der Abstand zwischen Linse und Netzhaut derselbe bleibt, so wird von Donders dagegen geltend gemacht, dass weder die Lageveränderung der Iris nach Vorn und

noch mehr die stärkere Wölbung der vorderen Fläche der Linse eine geringe Verrückung der entoptischen Doppelbilder nothwendig macht, so dass, wenn diese nicht hier gesehen werden, wir dies allein auf Rechnung der sehr mühsamen und desshalb weniger vollkommenen Wahrnehmung bringen müssen [1]).

Cramer hat also nicht bewiesen, dass bei der Accommodation für die Nähe überhaupt keine Veränderung der Gesichtsachse zu Stande kommt. Inzwischen lässt der hohe Druck, unter dem die Flüssigkeiten im Augapfel sich befinden, bei der geringen Elasticität der umhüllenden Membranen, kaum eine merkbare Verlängerung der Gesichtsachse zu, die, wie auch vielseitig behauptet wird, in jedem Falle, wo sie zur Accommodation erforderlich sein möchte, so gut wie gänzlich wegfällt.

In der dritten minder wichtigen Abtheilung seiner Schrift handelt Cramer über den Accommodationszustand für die Ferne als das natürliche Verhalten des Auges. Dass dieser Zustand wirklich der der Ruhe ist, will Cramer durch den Stand der Reflexbilder bei der Ermüdung des Auges bestätigt gefunden haben, wozu aber Beobachtung unter einem bestimmten Winkel nothwendig gewesen wäre. Cramer ist, wie hieraus hervorgeht, der Meinung, dass das Accommodationsvermögen erst bei einem Abstand von 50 Centim. zu

[1]) Donders hat die mit ein oder zwei kleinen Oeffnungen versehenen Platten zur entoptischen Untersuchung am Ende eines cylindrischen Behälters, der durch Einschieben von 1 auf 2 Centimeter Länge gebracht werden kann, befestigen lassen, um alles seitlich einfallende Licht abzuschliessen. Mit solchem Apparate kann man ohne grosse Schwierigkeit die einzelnen oder doppelten Bilder der entoptischen Phänomene auf gut erleuchtetes weisses Papier projiciren und unmittelbar mit der Bleifeder abzeichnen.

wirken anfange. Er berücksichtigt, um so zu sprechen, sicher nur kurzsichtige Augen, denn wenn wir lesen, dass bei Fernsichtigen, deren nächster Punkt 30—40 Centim. beträgt, kein Accommodationsvermögen stattfindet, so glauben wir dies so auslegen zu müssen, dass Cramer bei diesen Menschen keine Lageveränderung des Reflexbildes wahrnehmen konnte. Es wird allerdings, wenn sich im höheren Alter Fernsichtigkeit einstellt, das Accommodationsvermögen meistens in hohem Grade geschwächt, es wird aber, wie man sofort erkennt, wenn man positive Brillengläser gebrauchen lässt, keineswegs aufgehoben.

Cramer sucht ferner (im 4. Cap.) den Beweis zu liefern, dass die stärkere Wölbung der vorderen Linsenfläche bei der Accommodation für die Nähe durch contractile Elemente bewirkt wird, die im Auge selbst liegen. Er beruft sich insbesondere auf die nach Belladonnaeinträufelung beobachteten Erscheinungen. Bei derselben vermindert sich, wie man weiss, das Accommodationsvermögen für die Nähe. Zugleich wird bei starkerweiterter Pupille die Beobachtung auf jeden Abstand weniger scharf, was der geringeren Vollkommenheit der seitlichen Linsentheile zugeschrieben werden zu müssen scheint. Um zu untersuchen, ob die Beschränkung der möglichen Accommodation für die Nähe nicht vielleicht eben so sehr der Pupillenerweiterung als solcher zugeschrieben werden müsse, stellte Donders optometrische Versuche an, bei denen die Strahlen allein durch den centralen Theil der Linse gehen konnten, und überzeugte sich auf diese Weise, dass wirklich das Accommodationsvermögen für die Nähe beschränkt war. Cramer erkennt diesen Versuchen volle Beweiskraft zu und stimmt mit Donders darin überein, „dass es ungereimt sein würde, die in-

stillirte Feuchtigkeit einen Weg nach den Augenmuskeln ablegen und gefässreiche Gewebe durchdringen zu lassen, da es ja bereits früher von den Blutgefässen aufgenommen und durch den ganzen Körper verbreitet sein müsste.“ Richtig bemerkt er aber weiterhin, dass andere Beweise nicht überflüssig sind. Jedoch würde nicht allein, wie Cramer scharfsinnig anführt, wenn die Accommodation durch Druck der *mm. obliqui* bewirkt wird, dieser Druck durch Erweiterung der Pupille und Erschlaffung der Irisgefässe die gewohnte Wirkung einbüssen können, v. Graefe in Berlin hat auch noch ausserdem, bereits vor geraumer Zeit, entdeckt, dass, wie ungereimt es auch scheinen mag, die Muskeln durch Belladonnaeinträufelung direkt afficirt werden, und noch kürzlich berichtete er an Donders, dass er diese seine früheren Beobachtungen durch ganz genau angestellte Versuche bestätigt gefunden habe. Wir nehmen desshalb Cramers Untersuchungen, die unseres Erachtens allem Zweifel ein Ende machen, dankbar an.

Er bediente sich des Auges von einem kürzlich getödteten Seehunde, befreite es von den umgebenden Theilen, schnitt an der Hinterfläche Sclerotica, Choroidea und Retina in einem bestimmten Umfange, ohne das corp. vitreum zu verletzen, fort, brachte es mit nach Unten gerichteter Cornea in einem Ring unter dem Mikroskope, betrachtete das durch die Augenmedien geformte Bild einer Flamme, die sich in einem Abstande von 35 Centim. befand und vom Spiegel zurückgeworfen war, und sah nun stets, wenn der Strom eines magneto-electrischen Rotationsapparates auf die Corneawölbung geleitet wurde, die Flamme breiter, undeutlicher und weniger begränzt werden. Schon mit dem blossen Auge konnte dasselbe wahrgenommen

werden. Cramer überzeugte sich, dass durch diese Procedur das Auge für die Nähe accommodirt wird [1]).

Im 5. Cap. des Cramer'schen Werkes folgen einige anatomische Bemerkungen in Bezug auf einige Augentheile, die erste bezüglich des Standes der Iris.

Cramer sucht hier besonders zu beweisen, dass die hintere Irisfläche nicht allein unmittelbar auf der Linse ruhe, sondern auch auf den processus ciliares

[1]) Cramer bediente sich bei seinen Untersuchungen vorzüglich der Augen von Seehunden (Phoca litorea), auf die schon in der Preisfrage selbst hingewiesen war. Dass wirklich das Accommodationsvermögen bei den Seehunden sehr gross ist, bestätigt sich durch den Umstand, dass, wenn das Auge eines frisch getödteten Seehundes mit dem electromagnetischen Apparate in Verbindung gebracht würde, eine sehr beträchtliche Verkürzung der Brennweite des Auges eintrat. Zugleich geht aus diesen Versuchen hervor, dass das Auge des Seehundes wie das des Menschen von Natur und in dem Ruhezustande auf das Sehen in die Ferne in der Luft accommodirt ist, und zum Sehen in der Nähe in der Luft und noch mehr im Wasser sich erst besonders accommodiren muss. Er sah ferner, dass unter Anwendung des magnetoelectrischen Apparates die Pupille sich so schnell verengerte und bei Unterbrechung der Steigung so rasch wieder erweiterte, dass er vermuthete, es möchten animale (gestreifte) Muskelfasern in der Iris vorhanden sein; allein eine nähere Untersuchung lässt ihr bloss eine sehr grosse Anzahl organische (glatte) Muskelfasern, radiale wie kreisförmige, darin erkennen, was er als einen Beweis hervorhebt, dass sich nicht alle organischen Muskelfasern durch die Weise, wie sie sich zusammenziehen, von den animalen unterscheiden. — Dass die Seehunde nicht bloss im Wasser, sondern auch auf dem Lande gut sehen, erwähnt schon Cuvier (Ann. du Mus. d'hist. nat. I. XVII); alle Seehundsfänger bestätigen es und fügen hinzu, dass der Wind, der ihnen durch den Geruchssinn die Nähe des Jägers verrathen könne, keinen Einfluss habe. (Natuurk. Verhandel. van de Holl. Maatsch. de Wet. te Haarlem. 8. Deel. 1853. p. 60. 88. — Vergl. Fechner's Centralblatt für Naturwissenschaften und Anthropologie. N. 7. 1854. —

und der Zonula Zinnii, dass also keine hintere Augenkammer bestehen könne.

Die Meinung, dass die Iris in einer verticalen Fläche auf der Gesichtsachse stehe und dass also ein Raum als hintere Augenkammer vorhanden sei, wurde von Petit im Jahre 1728 gegen die herrschende Meinung aus scheinbar unzweifelhaften Gründen behauptet und seit der Zeit fast allgemein angenommen. Erst in der letzten Zeit wurden wieder gewichtige Gründe für die entgegenstehende Ansicht vorgebracht, so von Stellwag von Carion (cfr. Wiener Zeitschrift der Aerzte, 1850, Hft. 3. S. 125.), und auch Cramer erklärt sich auf Grund seiner Untersuchungen ausdrücklich gegen die Meinung Petit's. Zuerst sucht er zu beweisen, dass die Iris einige, wenn auch nur geringe Wölbung nach Vorn besitzt, und behauptet im Gegensatz zu dem Ausspruche von Petit und Czermak, dass die Iris eines unter Wasser gehaltenen oder durch das (Czermak'sche) Orthoscop gesehenen Auges diese Wölbung zeige. Wir wollen nicht läugnen, dass die geringe, kaum wahrnehmbare Wölbung, die man auf diese Weise wahrnimmt, für uns keine endgiltige Beweiskraft hat, und zwar in Betracht des Lichtbrechungscoefficienten des Kammerwassers und der Cornea, der von dem des Wassers noch etwas übertroffen wird, und dass desshalb, wenn auch die Iris in einer verticalen Fläche liegt, doch immer einige scheinbare Wölbung gesehen werden muss.

Sodann stellt Cramer auf, dass die Iris ungefähr 1 mm. hinter dem Ursprung des *m. tensor choroideæ*, also auch nahezu 1 mm. hinter dem canalis Schemmii entspringe, — also vielmehr hinten (wie er aus den in dieser Hinsicht sehr unvollkommenen Messungen von Krause darthut), als der hinterste Pol der Linse. Daraus

müsste mit Sicherheit folgen, dass die Iris nach Vorn zu gewölbt ist; nach den Donders'schen Untersuchungen hängt die Iris aber, mindestens dem Ursprung des *m. tensor choroideæ* gegenüber, mit den rückwärts gerichteten Fasern zusammen, die aus der membr. Descemetii entspringen, so dass also die Befestigung der Iris zu viel nach Hinten und Innen gesetzt und abgebildet wird. Es ist ganz richtig, dass die Iris mehr nach Hinten mit den Fasern der Descemet'schen Membran zusammenhängt, als wo sich die Grenze der durchscheinenden Cornea befindet, denn an der Aussenseite ist ihr Gewebe mehr nach Vorn undurchscheinend, als an der Innenseite, und hier liegt der Schlemm'sche Canal, hinter welchem (doch auch schon in dem hier an elastischen Fasern reichen, undurchscheinenden Gewebe) der *m. tensor choroideæ* entspringt. Dies erklärt die Möglichkeit, wie in einem Hohlspiegel, einen Schlagschatten der Sclerotica an der Seite, von der das Licht einfällt, zu sehen.

Können wir also mit allen zum Beweise angeführten Gründen nicht übereinstimmen, so sind wir doch geneigt, mit Cramer anzunehmen, dass die Iris allzeit eine geringe Wölbung nach Vorn besitzt und dieselbe dadurch erhält, dass sie auf der Linse ruht. Das eine Organ ist von dem andern abhängig: die Iris kann sich bei der Verengerung und Erweiterung der Pupille nicht über die capsula lentis hinschieben, ohne gewölbt zu sein, sie kann auch, wie Stellwag von Carion [1]) sehr richtig bemerkt und was schon Winslow [2]) nachgewiesen hat, wegen ihrer longitudi-

[1]) Zeitschrift der k. k. Gesellsch. der Aerzte zu Wien; 1850. 3. Hft. S. 125. — Es sind auch Carion's andere Gründe für das Aufliegen der Iris auf der Linse zu vergleichen.

[2]) Mémoires de l'Académie Royale des Sciences, 1721, p. 310.

nalen und circulairen Muskelfasern keine Wölbung besitzen, wenn sie nicht auf einem festen Körper aufliegt. Stellwag von Carion nahm an, dass sich unmittelbar um die Linse zu einige wässrige Feuchtigkeit befinde, aber wie in einem Canale eingeschlossen, der hier zwischen Iris und Zonula Zinnii übrig geblieben sei. Die alte Methode, frische Augen gefrieren zu lassen und in hinreichend gefrorenem Zustande in der Gesichtsachse zu durchschneiden, belehrte Cramer, dass es überhaupt keine hintere Augenkammer gibt. Schon Winslow war durch denselben Versuch zu demselben Resultate gekommen und selbst Petit, der gewissermassen der Schöpfer der hinteren Augenkammer ist, fand in ihr nur eine äusserst dünne Lage oder überhaupt gar kein Eis. Diese Untersuchung darf für überzeugend gehalten werden. Besonders lässt Cramer's Methode [1]), das frische Auge zuerst in Gips zu drücken, es nun frieren zu lassen, zu durchschneiden und dann in der in Gips geformten Höhlung zum Aufthauen zu bringen, a priori vollständig endgültige Resultate für das todte Auge erwarten.

Es wird Niemandem befremdlich erscheinen, dass Cramer sich so viele Mühe gab, um das Ruhen der Iris auf der Linse zu beweisen, wenn er weiter hört, dass Cramer's Erklärung des Mechanismus der Accommodation das Aufliegen voraussetzt. Aber sollte es nicht möglich sein können, dass bei der Accommodation auf Abstand wirklich ein kleiner, mit wässriger Flüssigkeit gefüllter Raum zwischen der Iris und den hintergelegenen Theilen übrig bleibt und dass derselbe erst bei der Accommodation für die Nähe verschwindet?

[1]) Cfr. Tijdsch. d. Maatschappij t. bevord. der geneesk. 1848, p. 114.

Cramer geht weiter über zu der *Choroidea*, den *proc. ciliares* und dem *m. tensor choroideae* Neue Resultate lieferte seine Untersuchung nicht. Denn schon bei Brücke [1]) und ebenso bei Bowman [2]), der diesen Muskel unabhängig von Brücke, wenn auch später als dieser, unter dem Namen von *ciliary muscle* beschreibt, liest man, dass er sich ungefähr auf der Höhe der ora serrata in die Choroidea verliert, — ein Umstand, den Cramer gefunden zu haben glaubte. Hinsichtlich des Ursprungs oder besser der Anheftung dieses Muskels merkt Donders noch an, dass die Fasern an der Innenseite des Schlemm'schen Canales grösstentheils aus der membr. Descemet. herkommen, die, was hier vorläufig mitgetheilt wird, damit anfängt, sich nicht in Fasern, sondern in Platten zu zerspalten, deren Fasern im Anfang der Spaltung einen circulären Verlauf, welcher der Cornealperipherie folgt, haben und nur zu einem kleinen Theile mit der Iris, zu einem viel grösseren Theile allein mit dem m. tensor choroideæ zusammenhängen. Auf die Bedeutung dieses Zusammenhangs für die Accommodation werden wir später zurückkommen.

Bei dieser Gelegenheit handelt Cramer auch von den Muskelfasern in Vögelaugen. Ausser den quergestreiften Circularfasern, die Krohn zuerst genauer untersucht hat, nimmt Cramer radiäre organische Muskelfasern an, die unter dem Einfluss des n. sympathicus stehen. Er scheint sie aber nicht gesehen, wenigstens nicht isolirt zu haben, ihr Vorhandensein vielmehr aus der Erweiterung der Iris zu folgern, die

[1]) Anat. Beschreibung des menschlichen Augapfels. 1847. p. 18.

[2]) Lectures on the parts concerned in the Operations on the Eye etc. London 1849. p. 51: „terminating about on a line with the ora serrata."

beim Prickeln der regio cilio-spinalis des Rückenmarks wahrgenommen wird. Dass hierin kein Beweis ihres Bestehens liegt, wird bald klar werden. Es kommt nämlich im Auge noch ein Muskel, der aus quergestreiften Bündeln besteht, vor, das Analogon des *m. tensor choroideae* und nur der Form des Vogelauges nach von besonderer Beschaffenheit. Dieser Muskel hat ein unglückliches Loos. Von Crampton entdeckt, erhielt er den Namen *m. Cramptonianus*. Treviranus hat sich sodann dergestalt von dem Nichtbestehen eines Accommodationsvermögen überzeugt, für welches dieser Muskel als ein lästiger Zeuge auftreten konnte, dass er die Natur desselben als eines Muskels bestritt, obwohl er die Querstreifen auf den Primitivbündeln wahrnahm. Hueck, der sich so viele Mühe gab, um das sog. *ligam. ciliare* bei Vögeln genau zu beschreiben, — befangen durch die Rolle, welche die processus choroideæ bei der Accommodation spielen mussten, und unerfahrener Histolog ausserdem, — behauptete geradezu, dass er „nichts weniger als muskelartig" sei. Nachdem nun Krohn seine muskelartige Natur ausser Zweifel gesetzt hat, machte Brücke aus ihm zwei Muskeln, von denen der eine zwischen der membr. Descem. und dem Knochenringe, der andere zwischen dem Knochenringe und der Choroidea liegen sollte, — der erste wäre allein der *m. Cramptonianus*, der andere müsse als analogon des neu entdeckten *m. tensor choroideae*, denselben Namen führen. Cramer sagt endlich, dass er nach genauer Untersuchung diesen anderen Muskel wirklich so gefunden habe, wie Crampton ihn beschreibt; aber er behauptet zugleich, dass der *m. Cramptonianus* nicht, wie Crampton und Brücke angeben, von der Innenseite des Knochenringes entspringe und sich an die membr. Descemet.

anhefte, sondern dass er von der Choroidea an der Stelle entspringe, an der sich inwendig die processus ciliares befinden, und dass er sich an die vordere Innenfläche des Knochenringes und an die membr. Descemetii anhefte. Bei Vögeln wäre der *m. Cramptonianus*, ebenso wie der *m. tensor choroideæ* bei dem Menschen, so innig mit der Innenwand des Canalis Schlemmii verbunden, dass dieser letztere bei der Ablösung des Muskels daran hängen bleibe, worauf auch Crampton selbst schon aufmerksam gemacht haben soll. Aus den von Crampton citirten Worten erhellt indess eigentlich etwas Anderes, nämlich, dass er den Zusammenhang mit der Choroidea nicht übersehen hat. Wie Cramer nun ausser dem m. Cramptonianus, wie er denselben beschreibt, mit Brücke noch einen *m. tensor choroideæ* annehmen konnte, ist unbegreiflich, da die beiden Brücke'schen Muskeln in der Cramer'schen Beschreibung des m. Cramptonianus so gut wie aufgenommen sind.

Die bestehende Verwirrung veranlasste Donders zu eignen Untersuchungen. Es ist ihm bereits ausser Zweifel (indem er besonders Augen von calecutischen Hühnern, dann auch von gewöhnlichen Hühnern, Gänsen, Enten und Tauben zur Untersuchung benutzte), dass es nur einen Muskel gibt, der den Namen m. Cramptonianus zu führen verdient. Er entspringt, als ein zierlicher, halbgefiederter kleiner Muskel, an der äusseren Wand des Canalis Schlemmii, an welcher Wand die membr. Descemet. kaum einigen Antheil hat, und ferner von der Aussenseite eines faserartigen Stranges, der sich von der besagten Wand isolirt, um sich ziemlich entfernt, der Krümmung des Knochenringes folgend, nach Hinten hin fortzusetzen. Die vorderen Fasern laufen nach Aussen und Hinten und heften sich

an das faserartige Gewebe der Sclerotica, das den Knochenring von Innen auskleidet; je weiter nach Hinten von dem faserartigen Strang sie entspringen, um so mehr nehmen sie eine Rückwärtsrichtung an, so dass die letzten, der Beugung der Sclerotica folgend, sich auf der Aussenfläche der Choroidea einpflanzen, an der Stelle, wo der knöcherne Ring der gewöhnlich als hornartig beschriebenen Lage von wahrer Knorpelsubstanz in der Sclerotica Platz gemacht hat. — Aus dieser kurzen Beschreibung, der Donders bald eine ausführlichere, mit Abbildungen versehene folgen lassen will, kann bereits ersehen werden, was dieser Muskel in anderweitigen Beziehungen bewerkstelligt.

Cramer handelt im 6. Cap. von **dem Nichtvorhandensein von Contractilität der Krystalllinse** (lens und capsula lentis). Die Meinung, die aus früheren Zeiten noch Einigen geläufig sein mag, dass die Linse Contractilität besitze, könnte vielleicht bei dem heutigen Standpunkte der Histologie schon a priori verworfen werden. Zum Ueberfluss hat Cramer, wie später auch Helmholtz, diese Frage erfahrungsgemäss mit negativem Resultate untersucht.

Im 7. Cap. ist nur von der Zonula Zinnii die Rede. Cramer bestätigt die Brücke'sche Beschreibung, läugnet den m. compressor lentis Langenbeck's und kommt zu dem Resultate, dass diese structurlose Membran ganz nothwendig ohne Elasticität sein müsse.

Bei dem 8. Capitel, sicher einem der wichtigsten, müssen wir länger verweilen. Es drückt das Siegel auf die Faraday'schen Worte, die ihm als Motto vorangesetzt sind: „I may well say that no man if he take industry, impartiality and caution in his investigations of science, ever works experimentally in vain."

Cramer untersucht hier, wodurch die Linse ihre stärkere Krümmung an der Vorderfläche bei dem Accommodationszustande für die Nähe erhalte. Eine grössere Versuchsreihe gab die hier erforderliche Sicherheit. — Auf einen hölzernen Ring auf der Oeffnung des Objecttisches brachte er, mit der Cornea nach Unten, das Auge eines kurz vorher getödteten Seehundes. Die Membranen der hinteren Hemisphäre waren zum grössten Theil entfernt. Auf diese Weise beschaute er das Bild eines entfernten, auf dem flachen Spiegel des Mikroskopes entstandenen Gegenstandes. Das Bild veränderte seine Stelle, wenn seitlich an die Cornea der Draht eines magneto-electrischen Apparates gebracht wurde. Dabei verengerte sich zugleich die Pupille. Mit einer Staarnadel durchschnitt er nun die Iris, so dass coloboma totale entstand, und sah nun, dass durch den electrischen Strom keine Veränderung mehr im Refractionszustande hervorgerufen wurde. — Bei einem zweiten Auge von einem Seehunde hatte er die Cornea weggeschnitten und die Iris vom orbiculus ciliaris getrennt und sah nun sogar mit dem blossen Auge, dass bei electrischer Reizung die processus ciliares angespannt wurden, was er der Wirkung des *m. tensor choroideae* zuschreibt. Mit Augen von Hunden, Kaninchen etc. glückten diese Versuche nicht, weil sich die Pupille beim Ausschneiden des Auges zu stark verengerte. Bei Vogelaugen, oft unausgeschnitten im Kopfe, der auf einer Korkplatte befestigt war, untersucht, überzeugte sich Cramer mit Hilfe seines Ophthalmoscops, dass ein seitlich durch die Cornea geführter electrischer Strom dieselbe Lageveränderung der Lichtbilder zu Wege brachte, welche bei der Accommodation entsteht. Das Corneabild erlitt keine Veränderung, so dass Cramer sowohl Crampton

als Brücke bestreitet, von denen der erste Abplattung, der andere eine stärkere Wölbung der Cornea von der Zusammenziehung des *m. Cramptonianus* erwartete. Nachdem die Cornea weggeschnitten und das Bild der vorderen Linsenfläche so heller geworden war, als für gewöhnlich das der Cornea, bemerkte er, dass durch galvanische Reizung dies Bildchen viel kleiner wurde. Er konnte diese Erscheinung wiederholte Male auf demselben Auge beobachten. Wurde aber die Iris vorsichtig mit einer kleinen Pincette abgelöst, so wurde bei der Reizung niemals eine Veränderung in dem Spiegelbilde wahrgenommen.

Aus diesen Versuchen kommt Cramer zu der Folgerung, dass „**der Eintritt einer stärkeren Krümmung der vorderen Fläche der Linse durch eine Wirkung der Iris zu Stande kommt.**“ Die vergleichende Anatomie bestätigt diesen Satz.

Es bleibt nun die Untersuchung noch übrig bezüglich der **Art und Weise, durch welche die Accommodation des Auges zu Stande kommt.** Dieselbe ist Object des 9. Capitels.

Winslow berichtet irgendwo, dass die Iris einen Druck auf die Linse ausüben könne, in Folge dessen die Linse nach Hinten ausweichen müsse, ohne dass er hierin den Grund der stärkeren Wölbung des freien, der Pupille entsprechenden Theiles der Linse erkannte. Stellwag von Carion bemerkt, dass ein derartiger Druck auf die Linse die nothwendige Folge einer gleichzeitigen Wirkung der circulären und radiären Irisfasern sein müsse, weil durch denselben die nach Vorn gewölbte Iris das Bestreben erhalte, sich in der Fläche auszudehnen. So gibt auch Cramer an, das bei der Zusammenziehung der circulären Fasern die radiären gleichsam zwischen zwei festen Punkten, dem Ursprunge

und dem Pupillarrande, ausgespannt sich befänden, demzufolge ihre Zusammenziehung einen Druck auf die in ihrer Concavität gelegenen Theile ausüben müsse. Bei weiter Pupille werden nur die processus ciliares und die Zonula Zinnii, bei engerer der Rand der Linse und bei sehr enger der grösste Theil ihrer Oberfläche gedrückt. Da nun die Linse nicht nach Hinten ausweichen kann (?), so muss der Druck eine stärkere Wölbung des in der Pupille sich hervordrängenden Theiles der Linse zur Folge haben. Die Weichheit der äussersten Linsenlagen begünstigt die Formveränderung derselben (Huschke, Forbes). Der Raum, den die (vordere) Augenkammer in der Mitte verliert, gewinnt sie zur Seite durch das Flacherwerden der Iris wieder, so dass der Raum für die wässrige Flüssigkeit derselbe bleibt. Die Elasticität der capsula lentis stellt die ursprüngliche Form wieder her, sobald der Druck aufgehoben wird.

Es ist deutlich, dass auf diese Weise bei verschiedener Pupillenweite ein Druck auf die Linse ausgeübt werden kann. Nach Cramer hängt die Kraft, mit der die Iris auf die in ihrer Concavität gelegenen Theile drückt, ab:

a) von der Länge des Bogens, den die Iris in ihrer Breite bildet, also von der grösseren oder geringeren Weite der Pupille;

b) von dem Grade des Tonus oder der Contraction, in dem sich die longitudinalen und circulären Muskelfasern befinden.

Was a betrifft, wie nämlich bei enger Pupille eine stärkere Wölbung erfolgt, bedarf einiger näheren Erklärungen. Es kommen, wie es wenigstens scheint, hierbei zwei Punkte in Betracht: 1) je kleiner die durch die Pupille hervortretende Fläche ist, einen um so

kleineren Krümmungsradius wird sie, cæteris paribus, annehmen können; 2) je kleiner die Pupille ist, desto länger sind die radiären Fasern und je weniger die Fasern bei ihrer Contraction verkürzt sind, um so grösser ist, wie schon Schwann bewiesen hat, ihre Kraft. Bei hellerem Lichte und convergirenden Gesichtsachsen, wodurch die Wirkung der Ringfasern verstärkt und die Pupille verengert wird, kann man das Auge desshalb für einen mehr in der Nähe befindlichen Punkt einrichten. Dem genannten Umstande ist es sicher auch zuzuschreiben, dass Verengerung der Pupille, durch plötzlich einfallendes Licht, das Auge für einen Moment für einen nahen Punkt, Erweiterung dagegen, durch Schliessen eines Auges bewirkt, für einen mehr entfernten Punkt accommodirt. Donders findet auch bei sich selbst, dass er, mit beiden Augen sehend, hell erleuchtete Gegenstände wenig näher gespalten sehen kann, wie mit einem Auge, wobei die Pupille etwas weiter ist.

Es ist wohl unbestreitbar, dass in der hinzukommenden Reflexion und Synergie, welche die zur Accommodation für einen bestimmten Abstand erforderliche Kraft willkürlicher Muskelwirkung bestimmen, der Grund zu suchen ist, wesshalb wir uns so viel weniger deutlich des richtigen Abstandes, für den wir unser Auge accommodirt haben [1]), bewusst sind, als dies bei andern willkürlichen Bewegungen der Fall ist, die desshalb nicht als massgeblich gelten dürfen. — Aus b folgt deutlich genug, dass bei gleicher Weite der Pupille der Accommodationszustand doch sehr verschieden sein kann; jedenfalls kann die Wirkung der

[1]) Nederl. Lancet. 3. Serie. II. p. 506.

Ring- und Strahlenfasern bei gleicher Pupillenweite eine durchaus verschiedene sein.

Oben wurde gesagt, dass nach Cramer die Linse beim Druck auf die Vorderfläche nicht nach Hinten ausweichen kann. Dies darzuthun, nimmt Cramer die Wirkung des *m. tensor choroideae* zu Hülfe und sagt: „die Folge der Contraction dieses Muskels ist deutlich. Schon früher haben wir beim Seehunde wahrnehmen können, dass durch Zusammenziehung dieses Muskels die *proc. ciliares* etwas angespannt werden. Durch die nur lose Anheftung des *m. tensor choroideae* und der *ora serrata* wird von da an, wo diese bereits aus der *Choroidea* zu entspringen beginnt, bis zu seiner Anheftung an die Wand des canalis Schlemmii und an die Sclerotica eine Contraction dieser Muskelfasern nicht behindert. Dagegen ist dieser Theil der Choroidea, aus dem die Muskelfasern des *m. tensor choroideae* entspringen, mittelst der membr. limitans sehr innig mit dem corp. vitreum verwachsen. Die pars plicata des corp. ciliare, ebenso die processus ciliares sind auch mit der Zonula Zinnii verwachsen, so dass man sich demzufolge leicht vorstellen kann, dass die genannten Muskelfasern das corp. vitreum und mittelst der proc. ciliares die Linse durch ihre Contraction verhindern können, vor dem Druck der Iris zurückzugehen, auszuweichen und dass sie sich im Leben sicher in demselben Maase anspannen, in welchem die Iris stärker drückt. — Auf diese Weise", fährt er fort, „muss es durch den *m. tensor choroideae* verhindert werden, dass der Druck, welchen die Iris auf die Linse ausübt, auch auf die Retina fortgepflanzt werde. Beachtet man, wie schnell durch geringen Druck auf die Retina die sog. phosphène [1]

[1]) Cfr. *Serre*, Annal. d'Oculist. 1850. 2. Ser. p. 31.

erregt wird, so ist es hinreichend klar, welche wichtige Rolle der *m. tensor choroideae* als solcher zu erfüllen hat". Donders sagt, dieser Erklärung gegenüber, dass nach seiner Meinung der *m. tensor choroideae*, den er lieber *musc. Brückianus* nennen will, eine ganz andere Bedeutung habe. Er sagt, für die Formveränderung der Linse sei dieser Muskel eben so wichtig, als die Muskelfasern der Iris. Ohne ihn ist die Iris nicht im Stande, einen Druck von Erheblichkeit auf die Linse auszuüben: der *m. Brückianus* liefert nämlich den festen Punkt für die Wirkung des *musc. dilatator pupillae*, dadurch, dass er den Faserring der membr. Descemetii, aus dem die Strahlenfasern des genannten *musc. dilatator* entspringen, rückwärts zieht und ihm Festigkeit gibt.

Eine lange fortgesetzte Untersuchung des gegenseitigen Verhaltens der Cornea und der sie innerlich auskleidenden Membran, der Sclerotica und der uvea (in der ursprünglichen Bedeutung des Wortes als *choroidea, m. Brückianus, processus ciliares* und *Iris* aufgefasst) hat Donders zu der Ueberzeugung geführt, dass die Befestigung des Anheftungspunktes der Iris nach Hinten die nothwendige Folge ist von einer Zusammenziehung des genannten *muscul. tensor choroideae*. Der vordere Anheftungspunkt dieses Muskels besteht hauptsächlich aus Fasern, die von der membr. Descem. nach Hinten und ein wenig nach Aussen sich erstrecken. Der hintere Anheftungspunkt ist die Choroidea, deren stroma zum Theil als perimysium dieses Muskels auftritt, in welches die Pigmentzellen, die diesem stroma eigen sind, sich fortsetzen. Von diesen beiden Anheftungspunkten ist der hintere, die Choroidea, viel weniger beweglich und ausdehnbar, als der vordere, die Fasern der membr. Descem. Diese werden bei

der Zusammenziehung des *m. Brückian.* angespannt und um so mehr nach Hinten ausgezogen, als die membr. Descem. auf den angränzenden Lagen der Cornea etwas verschiebbar ist und also mit nach Hinten weicht. Von der Innenseite dieser selben Fasern nun entspringt die Iris und auch ihr Anheftungspunkt wird also nach Hinten verschoben und angespannt. Seit Hueck, und vielleicht schon früher, stellt man sich gewöhnlich vor, dass einige Fasern der membr. Descem. als ligamentum pectinatum iridis auf die vordere Fläche der Iris übergingen. Bowman [1]) bildet dies ligamentum ab, als ob es ziemlich lang wäre, und Köllicker [2]) hat diese Abbildung nach meiner Meinung nicht dadurch verbessert, dass er zwischen *m. Brückianus* und *ligam. denticulatum* (Fig. 296. i) noch mehr Raum liess. Nach Donders Ansicht legt sich der erste unmittelbar an die Fasern an, welche nach Hinten gerichtet sind und zugleich zum Ursprung des *m. Brückianus* dienen. Wenn man, wie Hueck that, die Cornea nach Aussen umbiegt, um zwischen Cornea und Iris zu sehen, dann werden die genannten Fasern natürlich nach Vorn gedrängt und scheinen nun bogenweise auf die Iris überzugehen; ebenso kann man durch Spannung der Iris nach Hinten ein derartiges Aussehen bewirken; beim Durchschnitte wird es aber klar, dass kein eigentliches ligamentum pectinatum iridis besteht, sondern dass die aus der membr. Descem. entspringenden Fasern allein zur Anheftung des *muscul. Brückianus* dienen, an dessen Innenfläche sich, anfänglich dem Ursprung der Iris gegenüber, einige dieser Fasern fortsetzen. Nun sieht man leicht ein, dass diese Fasern dann allein einen

[1]) Lectures. p. 52.

[2]) Gewebelehre. p. 586.

festen Punkt zur Wirkung auf die radiären Muskelzellen der Iris abgeben, wenn sie nach Hinten zu gespannt sind, und dass die Iris gerade durch die Lageveränderung ihrer Peripherie nach Hinten auf günstige Weise in den Stand gesetzt wird, um auf die Vorderfläche der Linse zu drücken und die hintere Augenkammer, wenn sie besteht, zum Verschwinden zu bringen. Die Spannung der processus ciliares, welche Cramer bei galvanischer Reizung beobachtete, ist eine nothwendige Folge des Zusammenhangs der Aussenfläche der processus ciliares mit der Innenfläche des *m. Brückianus*, dessen vorderer und mittlerer Theil bei der Zusammenziehung etwas nach Hinten ausweichen. Dieser Bewegung folgen die *proc. ciliares*; ihr schwammartiges Gewebe, in den Falten der Zonula Zinnii, gewissermassen wie in einem Flüssigkeit haltenden Canale (canalis Petiti) eingeschlossen, fügt sich leicht jedem Druck- und Raumverhalten und unterstützt durch seine gespannten Blutgefässe die angrenzenden Theile wie ein elastisches Polster. Man muss annehmen, dass beim Tode, durch den die Wirkung des *m. Brückianus* aufgehoben wird, der Anheftungspunkt der Iris etwas mehr nach Vorn zu liegen kommt, — es ist desshalb im Leben seitlich noch weniger eine hintere Augenkammer anzunehmen, als in dem todten Auge. — Was nun die Spannung der Zonula Zinnii betrifft, so muss diese, als solche, um so weniger im Stande sein, das Hintenausweichen der Linse beim Druck auf deren Vorderfläche zu verhindern, als sie elastisch ist. Das Ausweichen nach Hinten wird aber grösstentheils durch den Druck verhindert, unter dem das corp. vitreum schon fortwährend steht, wie das die Spannung der Sclerotica beweist, die allem Anschein nach durch den Druck der Iris und vielleicht auch durch die Wirkung

des *m. Brückianus*, insofern er als *tensor choroideae* wirkt, noch vermehrt wird. Erheblich kann die Lageveränderung der Linse schon darum nicht sein, weil der Augenkammerraum bei unveränderter Wölbung der Cornea unverändert bleiben muss und beim Zurückweichen der Linse seitliche Theile nach Vorn treten müssten. Es scheint, dass der Druck, unter dem die wässrige Feuchtigkeit und also auch der durch die Pupille hervortretende Theil der Linse steht, bei der Accommodation für die Nähe geringer ausfallen muss, als der Druck des corpus vitreum, der auf die ganze Hinterfläche der Linse wirkt. Die Besorgniss Cramer's, dass ein gleichmässig erhöhter Druck des Glaskörpers nachtheilig auf die Retina wirken würde, scheint überhaupt ungegründet. Gerade die Erscheinung von *phosphène*, auf die er sich beruft, scheint dies zu lehren; denn wie sehr auch alle Feuchtigkeiten dabei unter höheren Druck gerathen, so tritt doch nur einige Reizung der Netzhaut ein (eben die Erscheinung von *phosphène*) und zwar auf dem Platze, wo eine veränderte Beugung der Membranen und also eine gewisse Spannung der Netzhaut verursacht wird [1]).

[1]) Das Wort *phosphène* wurde zuerst von dem unglücklichen Augenkranken Savigny, Mitglied des Institutes, gebraucht, der die Lichterscheinungen, die man beim Druck auf das Auge beobachtet, mit diesem Namen belegte. In letzter Zeit wurden die *phosphènes* wiederholt zur Sprache gebracht von Dr. Serre d'Uzès. Nach einigen Vorberichten ist kürzlich ein ausführliches Werk von ihm über diesen Gegenstand erschienen, das den Titel führt: Essai sur les phosphènes ou anneaux lumineux de la rétine, considérés dans leurs rapports avec la physiologie et la pathologie de la vision. Paris. 1853, — und in dem er die Schlusssumme seiner Untersuchungen, mit theilweiser Zurücknahme früher mitgetheilter Resultate, der Welt vorlegt. Donders sagt geradezu, dass die Bedeutung dieses Werkes für Physiologie und Pathologie zu seinem Umfange (469

Die Weise, in welcher die Accommodation in Vogelaugen zu Stande kommt, unterwirft Cramer einer

Seiten) in keinem Verhältniss stehe. Ausser dem Beweisse, dass die Empfindlichkeit erst ungefähr 1 centim. vom Rande der Cornea entfernt anfange, und der neuen Methode zur Bestimmung des Kreuzungspunktes der Richtungslinien, von der der Verfasser einen unglücklichen Gebrauch macht, findet Donders im ganzen Buche nichts für die Physiologie wissenswerthes. Vor der unvorsichtigen Anwendung in pathologischen Fällen warnt er. Serre theilt (p. 309) einen Fall von Diagnose einer Amaurosis mit, die erst am folgenden Tage zu Stande kam. Das beweist zu viel, eine Krankheit zu diagnosticiren, die noch nicht existirt! Ist es nicht viel wahrscheinlicher, dass der Kranke sich durch zu vieles Drücken auf das Auge, welches ihm vor der Nacht anbefohlen war, während er Alles gehörig sah, aber *phosphènes* zu sehen hartnäckig läugnete, seine Krankheit erst zugezogen habe? Donders hat nach dem Studium dieser Erscheinungen mehrere Tage Schmerzen in den Augen gehabt. — Der Nutzen für die Diagnostik scheint gänzlich problematisch, während der Augenspiegel sehr deutlich lehrt, wo die Augenmedien durchscheinend sind und wo dies nicht der Fall ist, und die Lichtperception durch die Sclerotica hin meistens befriedigende Endresultate gibt.

Was ferner die Erklärung vom Entstehen der *phosphènes* betrifft, so beruft sich Serre auf einen bestimmten Grad von Druck und bestreitet Brewster, der die Ursache in Ausdehnung der eingedrückten Retina sucht.

Wenn wir erwägen, dass sich allein der Umkreis eines Zirkels, ein Ring, erleuchtet zeigt, so lässt sich nicht wohl etwas Anderes, als der Rand, wo die Einbiegung beginnt, als die gereizte Stelle betrachten und hier, wo die Krümmung sich verändert, ist einige Spannung oder Ausdehnung der Netzhaut zu vermuthen. Dasselbe darf man bei tieferem Druck, gegenüber der Spitze der eingedrückten Stelle voraussetzen und dies erklärt die Lichterscheinung, die bisweilen in der Mitte des Ringes gesehen wird.

So viel ist sicher richtig, dass das plötzliche Entstehen von *phosphènes* bei Druck auf den Augapfel, durch den die Augenfeuchtigkeiten im Allgemeinen unter einen höheren Druck kommen, durchaus nicht den Beweis liefern, dass dieser höhere Druck einen nachtheiligen Einfluss auf die Netzhaut ausüben müsse.

besonderen Untersuchung. Wie schon oben erwähnt wurde, nimmt er radial geordnete Muskelzellen in der Vogeliris an, weil er bei der Reizung der regio spinalis Erweiterung der Pupille beobachtete. Es ist nicht wahrscheinlich, dass Cramer sie gesehen hat, und ausser an den Gefässwänden ist es auch Donders nicht gelungen, sie nachzuweissen. An der Peripherie der Iris findet man schon quergestreifte Muskelbündel, die auf der äusseren Gränze aber schmäler und weniger zahlreich sind. Cramer hat aber übersehen, dass durch Contraction des *musculus Cramptonianus* der peripherische Ursprung nach Aussen und Hinten gezogen wird. Nach der von Donders gegebenen Beschreibung bedarf dies keines weiteren Beweises, sobald nur noch gesagt ist, dass die *Processus ciliares* durch die elastischen Fasern, welche von der membr. Descem. herkommen, mit dem muskelartigen Streifen, der zur Anheftung des halbgefiederten *m. Cramptonianus* dient, weit nach Hinten zu verbunden sind. Die Contraction des *m. Cramptonianus* erklärt also die Erweiterung der Pupille bei der Reizung der regio cilio-spinalis, und da der *m. Brückianus*, wie oben nachgewiesen worden ist, ein Analogon des *m. Cramptonianus* bei Vögeln ist, so liegt hierin ein Argument für die jedenfalls sehr wahrscheinliche Annahme, dass auch dieser Muskel von dem *nerv. sympathicus* beherrscht wird. Aber zugleich wird es nun deutlich, wie ohne radiäre Muskelbündel in der Iris Druck auf die Vorderfläche der Linse ausgeübt werden kann. Es ist sogar sehr glaublich, dass der *m. Brückianus* dieselbe Wirkung hat, wie die radiären Muskelzellen der Iris, die mit dem *m. Brückianus* gewissermassen einen zweibäuchigen Muskel bilden. Der *m. Cramptonianus* kann beide ersetzen. Denn wenn die Peripherie der Iris nach Aussen und Hinten gezogen wird, müssen die

circulären Muskelbündel durch ihre Zusammenziehung, lediglich durch Ausübung eines Druckes auf die in ihrer Concavität gelegenen Linse, die Pupille verengern können.

Im 10. Capitel wird **die Nervenwirkung in ihrer Beziehung zum Accommodationsvermögen** in Betracht gezogen. Es ist bekannt, dass der nerv. oculomotorius direct auf die circulären Fasern der Iris wirkt, der nerv. sympathicus dagegen auf die longitudinalen und, wie wenigstens höchst wahrscheinlich ist, eben so sehr auf den *m. Brückianus*, bei Vögeln auf den *m. Cramptonianus*. Auch bei Reizung des nerv. trigeminus ist von Budge Zusammenziehung der Pupille beobachtet worden, die nicht unmittelbar bei der Reizung begann und nach der Reizung erst allmählig wieder nachliess. Offenbar stehen die radiären Fasern und der *m. Brückianus* bei dem Accommodationsvermögen direct oder indirect unter dem Einfluss des Willens. Cramer stellt nun die Hypothese auf, dass der nerv. trigeminus durch die radix longa motorische Nervenfasern nach dem ganglion ciliare sende und dass die durch den Willen gereizten Fäden sowohl die Fasern des nerv. oculomotorius als die des nerv. sympathicus im Ganglion ciliare zu erhöhter Thätigkeit veranlasse, so dass dadurch der ganze Muskelapparat für die Accommodation angeregt werde. Diese Hypothese mag rationell genannt werden, — sie schliesst sich aber zu wenig an unsere Kenntniss von der Bedeutung der Nervenknoten, um sofort angenommen werden zu dürfen. Der geringe Grad von Accommodationsvermögen, der nach Einträufelung von Belladonna und auch nach Lähmung des nerv. oculomotorius zuweilen übrig bleiben mag, würde nur als Grund angeführt werden können, wenn vorher bewiesen ist,

1) dass die in dem Stamm des nerv. sympathicus vorlaufenden Fasern nicht unter dem directen Einfluss des Willens stehen,

2) dass nicht andere unbekannte Momente ein geringes Maas von Accommodation bewerkstelligen können. — Wird es durch weitere Versuche klar, dass die Pupille sich durch Reizung des nerv. trigeminus verengert, wie Budge behauptet, dann wird die Hypothese einen festeren Boden erhalten, sobald man bei der Verengerung eine stärkere Convexität der Vorderfläche der Linse und der Lageveränderung der Reflexbilder constatiren kann. Liegt die Hypothese ihrem Aufsteller sehr am Herzen, so mag er die erforderlichen Versuche anstellen. Donders meint, dass man berechtigt sei, sie a priori zu verwerfen. Sie entspricht auch der allgemeinen Forderung für jede Hypothese, dass sie auf erfahrungsgemässem Wege geprüft werden kann. Die geringste von allen ist die in der angenommenen willkürlichen Wirkung gelegenen Schwierigkeit. Denn wie kann man daran zweifeln, dass die Bewegung der Iris etc. bei der Accommodation willkürlich ist, selbst wenn sie associirt wäre? Cramer geht indess ausführlich auf diese Frage von der Association ein. Er erinnert ganz besonders an die Donders'schen Experimente mit convexen, concaven und vorzüglich mit prismatischen Gläsern, zum Beweise, dass zwischen der Convergenz der Gesichtsachsen und dem Accommodationszustande keine nothwendige Verbindung besteht. Er macht es weiter wahrscheinlich, was de Ruiter[1]) später bewiesen hat, dass sowohl die Convergenz der Gesichtsachsen ohne Veränderung der Accommodation,

[1]) De Ruiter dissert. de actione Atropae Belladonnae in Iridem, Utrecht, 1853. — Donders wird den Hauptinhalt dieser Schrift in der Nederl. Lancet mittheilen.

als auch die veränderte Accommodation ohne bestimmten Einfluss auf die Convergenz der Gesichtsachsen mit Pupillarverengerung vereinigt ist. Da bei der Convergenz die Verengerung am stärksten ist, so glaubt Donders dies so auslegen zu müssen, dass mit der blossen Convergenz hauptsächlich die circulären Fasern der Iris associirt sind, während bei der Accommodation der ganze Muskelapparat in erhöhter Weise in Thätigkeit versetzt wird.

Im 11. Cap. fast Cramer **verschiedene Bemerkungen, die das Accommodationsvermögen betreffen,** zusammen. Zuerst kommt hier die Accommodation nach der Entfernung der Linse zur Sprache. Er sagt: man hat wiederholt Beispiele beizubringen gesucht, wo nach der Entfernung der Linse das Accommodationsvermögen fortbestanden haben soll. Haller theilt mehrere Beispiele mit, ebenso wollen auch Javin, Pellier, Gleize, Richter u. A. solche Fälle beobachtet haben.

Wurden früher solche Mittheilungen angeführt, um die noch als Hypothese geltende Formveränderung der Linse zu bestreiten, so ist es wegen der Wichtigkeit des Gegenstandes wohl der Mühe werth zu untersuchen, ob und dann wie nach der Entfernung der Linse noch Accommodationsvermögen da sein kann.

Home[1]) theilt über diesen Gegenstand Untersuchungen mit, die er an einem 21jährigen, durch Extraction von Cataract befreiten Matrosen angestellt hatte. Bei verschiedenen Prüfungen und Versuchen, die er mit Englefield und Ramsden vornahm, meinte er gefunden zu haben, dass das Accommodationsvermögen noch nach der Operation vorhanden gewesen

[1]) Philosophical transactions of the royal society of London for the year 1795.

sei. Er folgerte desshalb, dass „the adjustement of the eye to different distances could take place independent of the crystalline lens“. Klügel [1]) hat schon darauf hingewiesen, zu einem wie wenig zuverlässigen Resultate Home durch seine Versuche, dass noch Accommodationsvermögen geblieben, gelangt sei. Auch dass Home das operirte Auge durch eine sehr feine Oeffnung sehen liess, benimmt seinen Prüfungen allen Werth. Er theilt ausserdem hinsichtlich des Sehvermögens dieses Kranken noch mit, dass derselbe zur Zeit der ersten Versuche, 27 Tage nach der Operation, Alles etwas undeutlich gesehen habe, so dass schon desshalb über die Accommodation oder den Mangel derselben kein Urtheil gefällt werden kann.

Viele späteren Untersuchungen haben dem Home'schen entgegengesetzte Resultate geliefert. So hat Young [2]) an Cataract Operirte mit dem Optometer von Porterfield untersucht und als Resultat ausgesprochen, dass das Accommodationsvermögen nach Entfernung der Linse gänzlich aufgehoben sei. [3]) Auch aus den von Holke [4]) angestellten Versuchen geht zur Genüge hervor, wie beschränkt das Gesichtsvermögen wird, nachdem die Linse durch die Cataractoperation entfernt worden ist. Es ist ferner allgemein bekannt, dass die Operirten mehrere Brillen von verschiedenem Focalabstand besitzen müssen, um gut in der Nähe und in der Ferne sehen zu können.

[1]) Reil's Archiv. 1797. Bd. II. p. 51.

[2]) Philosoph. transactions for the year 1801. Part. I. p. 65.

[3]) Diogene, Hanson genannt, bei dem Young diese Versuche anstellte, hatte bereits das Alter von 63 Jahren. Dieser Umstand nimmt den Beobachtungen ziemlich alles Gewicht, da in diesem Alter Accommodationsvermögen fast nur noch ausnahmsweise vorhanden ist.

[4]) Holke, Disquisitio de acie oculi dextri et sinistri. Lipsiae. 1830.

Ebenso scheinen aber mehrere Beobachtungen dagegen zu sprechen, dass das Accommodationsvermögen durch die Entfernung der Linse gänzlich verloren gehe. Man braucht sich in dieser Beziehung nicht allein daran zu erinnern, was Maunoir [1]) mittheilt, dass Jemand, dessen cataractöse Linse discidirt und nachher resorbirt war, mit demselben Glase zugleich deutlich lesen und entfernte Gegenstände auf der Jagd erkennen konnte; es sind hier auch die Resultate der Untersuchungen des scharfsinnigen und genauen Volkmann [2]) anzuführen. Volkmann stellt verschiedene Prüfungen an und kommt endlich zu der Annahme, dass ein geringer Grad von Accommodationskraft auch nach der Entfernung der Linse noch fortbestehe.

Auch Stellwag von Carion [3]) theilt eine hierher gehörige Wahrnehmung mit. „Besonders auffallend“, sagt er, „war mir das Accommodationsvermögen eines vor der Trübung seiner Krystallkörper sehr kurzsichtigen jüdischen Religionslehrers. Nach der Operation las er kleingedruckte Schrift prompt, unterschied aber auch weitentfernte Gegenstände, z. B. das Fensterkreuz eines gewiss 300 Schritte entfernten Flügels des hiesigen Krankenhauses ganz deutlich und rein.“

Dass nach Entfernung der Linse ein gewisser Grad von Accommodationsvermögen fortbestehen kann, ist also nicht unwahrscheinlich. Viele Beobachter haben aber die Begriffe von Vorhandensein oder Fehlen des Accommodationsvermögens nicht richtig aufgefasst. Es kann nämlich etwas Gedrucktes gelesen und auch ein

[1]) Cunier Annales d'oculistique. Tom. IX. Avril-Juin 1843. p. 14.

[2]) Volkmann, Beiträge etc. p. 173.

[3]) Wiener Zeitschrift p. 195.

entferntes Object gesehen werden, während doch das Accommodationsvermögen fehlt. Sodann scheinen andere Beobachter mit Taylor [1]) der Meinung zu sein, dass es ohne Accommodationsvermögen nicht möglich sei, dass das Auge, nachdem der Krystall durch die ordentliche Art den Staar zu stechen aus der Augenachse gekommen, die in verschiedener Entfernung stehenden Gegenstände empfinden könne.

Ob auch vielleicht in einigen Fällen, z. B. in der zweiten Reihe der von Home angestellten Versuche, das mehr weniger mögliche Accommodationsvermögen nach der Cataractoperation einer Regeneration der Linse zugeschrieben werden muss, will ich unerörtert lassen. Aus den Untersuchungen von G. Vrolik [2]), Sömmering [3]), Middlemore [4]), Coiteau [5]), Löwenhardt, Davison, Werneck, Beck, später von Retzias und von Fronmüller [6]) geht hervor, dass sich bald eine neue Linse bildet. So sah man, wie auch Meyer [7]) mittheilt, dass sich bei Thieren, deren Linse weggenommen ist, während die capsula lentis bei der Operation erhalten blieb, jedes Mal schon nach

[1]) J. Taylor, Mechanismus oder neue Abhandlung von der künstlichen Zusammensetzung des menschlichen Auges. Frankf. a. M. 1750. p. 122.

[2]) Graefe und v. Walther's Journal. Bd. 18. Hft. 4.

[3]) W. Sömmering: Beobachtungen über die organischen Veränderungen im Auge nach Staaroperationen. Frankfurt a. M. 1828.

[4]) Froriep's Notizen. Bd. 34. p. 297.

[5]) Ds. Bd. 16. p. 289.

[6]) G. T. C. Fronmüller, Beobachtungen auf dem Gebiete der Augenheilkunde. Fürth. 1850. p. 65.

[7]) Graefe und v. Walther's Journal. Bd. 17. u. 18.

der vierten Woche die Linse reproducirt hat [1]). Auch Leroy d'Etiolle [2]), der mit Coiteau viele Versuche über die Regeneration der Linse anstellte, wirft bereits die Frage auf, ob diejenigen, welche nach der Cataractoperation ohne Brille zu lesen im Stande sind, dies der Linsenregeneration verdankten [3]). Jedenfalls ist es sicher, dass bei der Frage, ob das Accommodationsvermögen nach der Cataractoperation in gewissem Grade fortbestehen kann, auf die Regeneration der Linse nicht hinreichend Gewicht gelegt wird. Die entoptische Untersuchung wird besonders dazu beitragen, unsre Kenntniss von der Reproduction der Linse zu vermehren.

Eigene Beobachtungen, die auf diese Frage mehr Licht würfen, kann Cramer nicht beibringen. Er hat sich bei an Cataract Operirten niemals mit genügender Sicherheit von dem Vorhandensein eines gewissen Grades von Accommodationsvermögen überzeugen können. Wenn wir aber nach dem, was Volkmann und Stellwag von Carion hierüber mitgetheilt haben, annehmen wollen, dass nach Entfernung der Linse ein gewisser Grad von Accommodationsvermögen bestehen bleiben könne, so lässt sich dafür wohl an eine Ursache denken. So bemerkt auch Donders [4]),

[1]) Cfr. K. Textor. Ueber die Wiederersetzung der Krystalllinse. Inaugural-Abhandlung. Würzburg. 1842.

[2]) Leroy d'Etiolle, Recueil de lettres et de mémoires, adressés à l'academie des sciences, pendant les années de 1842—43. Paris. 1844.

[3]) Dass der am Cataract Operirte, der früher sehr kurzsichtig war, ohne Brille lesen konnte, spricht für sich selbst. Burow erzählt sogar, dass Jemand nach der Entfernung der cataractösen Linse noch etwas kurzsichtig geblieben sei.

[4]) In der von Donders veranstalteten holländischen Bearbeitung des Ruete'schen Lehrbuches der Ophthalmologie. p. 120.

dass dies nicht allein von der baldigen, theilweisen Reproduction der Linse, sondern vielleicht von einem durch Druck der processus ciliares bewirkten Vorrücken des Mittelstücks des corp. vitreum abhängig sein könne. Auch Stellwag von Carion glaubt [1]), dass noch ein gewisser Grad von Accommodationsvermögen nach der Entfernung der Linse möglich sei, nachdem „der mittlere Theil der Vorderfläche des Glaskörpers nach Vorn gestaucht", und dass auf diese Weise die Accommodation des Auges durch Druck der Iris auf die processus ciliares und weiter auf das corpus vitreum einigermassen bewerkstelligt werden könne.

Versuche an Kaninchen, bei denen die Linse durch Extraction oder durch Zertrümmerung und Absorption entfernt worden war, zeigten Cramer, dass der Glaskörper wirklich einige Rundung nach Vorn annimmt und auf diese Weise der Raum, den die Entfernung der Linse verursacht hat, einigermassen ausfüllt.

Bedenken wir aber, dass nach Stellwag von Carion's Mittheilung [2]) bei den an Cataract Operirten der Grenzpunkt des deutlichen Sehens in der Nähe in der Regel weit hinausgerückt ist, in mehreren Fällen auf 30—40 Zoll, und beachten wir weiter, dass erst bei Betrachtung von Objecten, die sich mehr als 50 Cent. dem Auge nahe befinden, das Accommodationsvermögen in Thätigkeit tritt, so ist es vorläufig hinreichend klar, was man von dem Fortbestehen des Accommodationsvermögens nach der Entfernung der Linse zu denken hat.

Die Fälle, bei denen als vitium congenitum die Linse fehlt, — einen solchen Fall bei einem Kalbe

[1]) l. c. p. 196.

[2]) l. c. p. 195.

beobachtete Prichard [1]) noch kürzlich, — sind hier nicht zu wiederholen. Es bestehen zugleich meistens andere Gebrechen, da das Fehlen der Linse meistens die Folge einer ophthalmia intra-uterina ist.

Donders bemerkt hierzu, dass die mitgetheilten Fälle nicht in Abrede gestellt werden könnten, dass das Accommodationsvermögen aber in der Mehrzahl der Fälle, also in der Regel fehle. Kehrt sich die schlüsselförmige Grube halbkugelig nach Vorn und ist der Lichtbrechungscoefficient des corp. vitreum wirklich etwas grösser, als der der wässrigen Flüssigkeit, so muss Druck der Iris auf die seitlichen Theilen noch wirksam sein können. An Regeneration der Linse und an eine geringe Verlängerung der Gesichtsachse durch die Augenmuskeln, besonders wenn die Augenflüssigkeiten wenig im Zustande der Spannung sich befinden, kann nach Donders Meinung auch noch gedacht werden. Er führt noch an, dass soviel feststehe, dass der geringe Grad von Accommodationsvermögen, der nach der Cataractoperation noch zurückbleiben kann, der Cramer'schen Theorie durchaus nicht gefährlich sei.

Weiter spricht Cramer in demselben Kap. von dem Einfluss der Belladonna auf das Accommodationsvermögen. Er sagt, es seien nicht alle Berichte über den Zustand der Sehkraft nach der Anwendung eines

[1]) Prichard. Provinl. Journal 1849. Nro. 8. — Bei einem halbjährigen übrigens gesunden Kinde in Kessenich bei Bonn beobachtete ich letzthin vollkommenen angeborenen Linsendefect; die Retina mit ihren Gefässen war ohne Spiegel deutlich sichtbar und schien ohne organische Destruction zu sein. Das Kind war, was die Eltern erst von mir erfuhren, absolut blind. Es starb einen Monat später an epidemischen Masern, ohne dass es mir vergönnt worden wäre, die Section der äusserst interessanten Augen vorzunehmen.

Mydriaticums gleichlautend. Allgemein wird aber anerkannt, dass das Refractionsvermögen abnimmt, dass sich namentlich der kürzeste Gesichtsabstand des Auges in höherem oder geringerem Grade entfernt. Dafür erklären sich Ray, Schmidt, Wells, Hutchinson, Jüngken, Hall, Dunglisson, Ritter, Volkmann, Hueck, Donders u. A. Hinsichtlich der Accommodation des Auges für die Ferne sind die Ansichten, ob die Anwendung der Belladonna Einfluss ausübe, verschieden. Dass der Gränzpunkt des Sehens für die Ferne jedenfalls nur wenig verändert wird, geht hinreichend daraus hervor, dass Normalsichtige eben keine Veränderung wahrnehmen.

Die meisten Beobachter sprechen desshalb auch hiervon nicht oder verwirren es mit dem, was allein als Folge der bedeutenden Pupillenerweiterung anzusehen ist. Es kann wegen der nur so geringen Verkürzung des fernsten Gesichtsabstandes desshalb auch wenig befremden, dass Bartelds und Hueck aussprachen, der fernste Gesichtsabstand werde durch die Anwendung von Belladonna nicht entfernt, er bleibe, obwohl sie sich als Kurzsichtige von der Verkürzung des fernsten Gesichtsabstandes hätten überzeugen können. Volkmann dagegen, der als Kurzsichtiger wegen des bei solchen vorhandenen Grenzpunktes für das Sehen in die Ferne im Stande ist, über eine unbedeutende Verringerung der Accommodation des Auges für die Ferne zu urtheilen, behauptet, dass sich nach Belladonnaeinträufelung der fernste Gesichtsabstand ein wenig verkürze. Er sagt, die Wirkung der Belladonna bestehe: 1) in der Beschränkung der Fähigkeit in der Nähe deutlich zu sehen, 2) in der Beschränkung der Fähigkeit, in der Ferne deutlich zu sehen, 3) in der Steigerung der passiven Sehweite, d. h. derjenigen,

in welcher ohne vorgängige Anstrengung am deutlichsten gesehen wird. Auch Stellwag von Carion ist der Meinung, dass der Grenzpunkt durch ein Mydriaticum etwas näher gebracht werde. Cramer machte den Versuch mit einer Belladonnaauflösung bei einem Individuum, dessen kürzester Abstand deutlichen Sehens in der Nähe vorher 25 Centim. gewesen war. Als sich nach einer halben Stunde die Pupille sehr erweitert hatte, betrug derselbe 40 Centim. In der Ferne wurde aber ebenso deutlich gesehen, wenn nur die grosse Erweiterung der Pupille durch eine in der Grösse der normalen Pupille durchbohrtes Kupferplättchen beschränkt war. Bei den Versuchen an sich selbst gebrauchte Cramer das Optometer von Donders [1]), ein schwarzes Linial, an einem Ende mit einem schwarzen Kupferplättchen versehen, das auf und nieder geschoben werden kann und in dem zwei kleine Oeffnungen angebracht sind, $^2/_3$ m. m. gross und ebenso weit von einander. Eine Stunde nach der sehr erfolgreichen Einträufelung war der kürzeste Gesichtsabstand von $7^1/_2$ Centim. auf $9^1/_2$ Centim., dagegen der Grenzpunkt beim Sehen in die Ferne von $12^1/_2$ auf $11^1/_2$ Centim. gebracht. Diese Resultate stimmen vollständig mit denen überein, die Volkmann durch Versuche beim Lesen erhielt, Donders sagt aber, dass sie beide ihn nicht vollständig überzeugt haben. Er fand die Schärfe des Sehens auf jeden Abstand verringert, was er der stärkeren Krümmungsabweichung der seitlichen Linsentheile zuschreibt. Bei gewöhnlichen Leseproben kann man desshalb nur schwer mit Sicherheit urtheilen. Das Optometer erklärt er bei hinreichender Uebung für brauchbar, um den Grenzpunkt

[1]) Ned. Lancet. 1851. April. p. 601.

des Sehens in der Nähe zu bestimmen, für ungeeignet aber, um den mittleren Abstand deutlichen Sehens anzuzeigen, der überhaupt zu veränderlich ist, um viel von ihm zu sprechen, — und wenigstens ungenügend, um den Grenzpunkt für die Ferne zu bestimmen, weil man unwillkürlich geneigt ist, beim Lesen durch kleine Oeffnungen sein Accommodationsvermögen ein wenig in Thätigkeit zu setzen. Wenn Donders nach Belladonnaeinträufelung durch eine Oeffnung von der Grösse einer normalen Pupille sah, dann konnte er ebenso gut Objecte auf grossen Abstand unterscheiden, als vor der Einträufelung, was beim Sehen durch ein schwach positives Glas von ungefähr 100 Centim. Focalabstand schon nicht mehr gut gelingt

Weiter bemerkt Donders, dass die Belladonnawirkung eine paralytische sei auf die Ringfasern der Iris, sicherlich aber keine beschränkende auf die radialen Fasern und den *m. Brückianus*, dass diese beiden letzteren vielmehr höchst wahrscheinlich in hohem Grade gereizt würden. In der ungeschmälerten Wirkung dieser Beiden muss der Grund des noch übrig gebliebenen Accommodationsvermögens gesucht werden, wenn auch in geringerem Masse, und ist wirklich, wie Volkmann, Stellwag von Carion und Cramer wollen, der Grenzpunkt für die Ferne näher gerückt, dann kann man dies mit dem gereizten Zustande der radialen Fasern und des *m. Brückianus* sofort in Zusammenhang bringen. Zur Erklärung des noch übrig gebliebenen Accommodationsvermögens verliere man nicht aus den Augen, dass der Rand der Iris stets breiter bleibt, als er, durch die Cornea geschen, zu sein scheint. Es lässt sich indess nicht läugnen, dass unsere Kenntnisse in dieser Hinsicht noch keineswegs befriedigender

Art sind. Um von dem übriggebliebenen Accommodationsvermögen nach Cramer's Theorie Rechenschaft zu geben, müssen wir annehmen, dass wenigstens die radialen Fasern und der *m. Brückianus* noch unter dem Einfluss des Willens geblieben sind.

Es ist Donders aber wahrscheinlich geworden, dass diese Fasern ein Maximum ihrer Thätigkeit erreichen, weil Reizung des nerv. sympathicus bei Kaninchen, deren Pupille durch Belladonnaeinträufelung weniger stark erweitert wird, keine fernere Erweiterung der Pupille bewirkt. Cramer will einmal beobachtet haben, dass eine durch Belladonnaeinträufelung erweiterte Pupille bei der Accommodation für die Nähe jedes Mal, statt enger zu werden, sich ein wenig erweitert habe. Er sagt, „obwohl dies nur wenig bemerkbar wurde, so war es doch zu deutlich, als dass ich mich darüber hätte täuschen können". Diese Beobachtung würde desshalb als eine wichtige betrachtet werden müssen, weil sie eine stärkere Spannung der radialen Fasern und des *musc. Brückianus* beweisen würde; aber alle Versuche mit den besten Hülfsmitteln, von Donders und de Ruiter angestellt, um diese Beobachtung bestätigt zu finden, schlugen fehl.

Man stösst nicht auf dieselbe Schwierigkeit, um das Fortbestehen des Accommodationsvermögens nach der Lähmung des *nerv. oculo-motorius* zu erklären, auf die Cramer sodann zu sprechen kommt. In allen Fällen, die Donders beobachtete, und später ausführlich mitzutheilen verspricht, waren der normale Refractionszustand und das Accommodationsvermögen auf der leidenden Seite geringer. Donders sagt mit Cramer, dass eine einzige Beobachtung von Ruete nicht beweisen kann, dass das Accommodationsvermögen fast ganz unbeeinträchtigt bliebe; gegen die Möglich-

keit, dass die circulären Fasern der Iris in diesem Falle nicht gelähmt waren, spricht indess die stärkere Weite der Pupille. Donders beklagt die Untersuchung versäumt zu haben, ob bei bestehender Lähmung des *nerv. oculo-motorius* die Pupille bei der Accommodation für die Nähe etwas erweitert war; er fügt hinzu, dass der Mechanismus der Accommodation es vermuthen lasse.

Was das Accommodationsvermögen bei Irideremie betrifft, so führt Cramer an, dass das Accommodationsvermögen bei theilweisem Irismangel und coloboma iridis fortbestehe. Die Fälle von totaler Irideremie scheinen nicht genau genug auf das Fortbestehen der Accommodation untersucht worden zu sein, um folgern zu können, dass es ungeschmälert bestehen bleibe. Cramer fand übrigens den Ausspruch von Carion von Stellwag bestätigt, dass bei Entzündung und Exsudation in der Iris die Accommodation aufgehoben ist.

In Bezug auf Presbyopia führt Cramer an dass bei Seeleuten und anderen Personen, die keine Uebung im Sehen in der Nähe haben, der nächste Grenzpunkt 45 und noch mehr Centim. betrage und dass bei diesen Personen auch keine Lageveränderung des Reflexbildes auf der vorderen Irisfläche beobachtet werde. Die Cornea fand er nicht flacher.

Das Entstehen von Myopia bei fortgesetztem Sehen in der Nähe erklärt Cramer aus dem Mangel an Elasticität in der capsula lentis und aus bleibender stärkerer Thätigkeit, resp. Entwickelung der Muskelfasern. Als Beweise führt er an: die bleibende stärkere Wölbung des durch den Pupillarrand begrenzten Theiles der Linse eines Seehundsauges, dessen Accommodationsapparat er längere Zeit gereizt hatte, und

die durch ihn beobachtete Thatsache, dass die Rundung der Cornea nicht zugenommen hatte, was er aus der Feststellung des Abstandes der Spiegelbilder zweier Lichter entnahm. — Dagegen musste es aus dem Stande der drei Reflexbilder bei dem gewöhnlichen Versuche (gewöhnlicher Abstand zwischen a und c, kleiner Abstand zwischen b und c, grosser Abstand zwischen a und b) ersichtlich werden, dass die vordere Fläche der Linse bei Kurzsichtigkeit voller würde. Donders macht darauf aufmerksam, bei vorkommenden Gelegenheiten die Augen von Personen, die durch Missbrauch des Sehens in der Nähe kurzsichtig geworden sind, mit gewöhnlichen Augen zu vergleichen und sagt, dass man eine solche Veränderung, die sich allmählig bei der Accommodation für die Nähe entwickelt habe, als eine bleibende antreffen würde.

[*From the* Annals and Magazine of Natural History *for October* 1854.]

ON THE MECHANISM OF AQUATIC RESPIRATION AND ON THE STRUCTURE OF THE ORGANS OF BREATHING IN INVERTEBRATE ANIMALS.

By THOMAS WILLIAMS, M.D. Lond.,
Licentiate of the Royal College of Physicians, formerly Demonstrator on Structural Anatomy at Guy's Hospital, and now of Swansea.

[With three Plates.]

Structure of the Branchiæ in the Lamellibranchiate Mollusks.

The *mist* upon this branch of natural history which has survived the brightening science of a bright century may indeed refuse to be dissipated even by the achromatic microscope—the potent wand of the modern observer. That which the calm eye discerns with clearness, and the understanding interprets with confidence, though amplified many hundred diameters, is as likely to be an immutable objective truth as any "instance" within the sphere of the unassisted vision. Faith in the verity of microscopic facts is a fundamental article in the scientific creed of every living philosopher. The sphere of the naked vision is exhausted: another is opened by the microscope. Minute descriptions of subtle and complex structures, rendered possible only through its instrumentality, will prove of as great service in the hands of the future lawgivers of science, as the grosser narratives of the fathers of anatomy have already proved in the founding of the temple in which the high priests of natural theology now chant her service.

The branchial structures of the Mollusca have never yet been unravelled. The problem, though not impracticable, still awaits solution. The system of the gills is a conspicuous element in the molluscan organism. In apparent size they are considerable.

If function were expressed in numeric amount by the dimensions of the organs, the physiologist would assign to this class of animals a high degree of respiration. Minute structure is a factor in the estimate. The gills of the Lamellibranchiate mollusk are singularly and peculiarly formed: they admit of comparison in structural characters with no other organ found amongst Invertebrate animals. The *meaning* of a part is an inference of the intellect. When exact, it is founded upon a correct appreciation of structure. A 'law' is upraised upon the basis of particulars. Let the following difficult inquiry be conducted in rigid compliance with this *regulus philosophandi*. Though abundant, the elder literature upon this subject has bequeathed little that is accurate and true. Baer* alludes in a special manner to the pectinated character of the branchiæ in the Lamellibranchiata; he illustrates his description by the gills of *Mytilus*. Meckel† depicts and describes in general terms a comb-like structure in the gills of *Spondylus*, *Pecten*, and *Arca*. Cuvier's figures and descriptions‡ delineate the same formation. In his valuable notes, Siebold§ describes the branchiæ in *Pectunculus*, *Mytilus*, *Arca*, *Pecten*, *Avicula*, and *Lithodomus* as consisting of a system of parallel vessels. In the text of his work, however, Siebold, like Mr. Hancock, speaks of the trellis-like network of the branchial structures. Among the older authors by whom allusion is made to the pectinated arrangement of the branchial vessels, the names of Bojanus, Treviranus, and Poli may be enumerated.

The contributions of Mr. Hancock upon this subject are the most recent, special, and distinguished‖. By this observer three types of structure are recognized. They are thus defined in his own language:—"There appear to be *three distinct modifications* of gill-structure in the Lamellibranchiata. In the first the laminæ forming the gill-plate are composed of filaments either free or only slightly united at distant intervals, as in *Anomia* and *Mytilus*; in the second they are formed by a *simple vascular network*, as in *Mya*, *Pholas*, &c.; and in the third the laminæ of the gill-plate are complicated by the *addition of transverse plicæ composed of minute reticulations of vessels*, as in *Chamostrea*, *Myochama*, *Cochlodesma*, &c. Other modifications may exist,

* Meckel, Arch. 1830, p. 340.

† Syst. der Vergleich. Anat. vi. p. 60.

‡ Règne Animal, nouvelle edit., Mollusques, pl. 74. fig. 2 *a*.

§ Anatomy of the Invertebrata, translated by Burnett, p. 211.

‖ The excellent papers of Messrs. Alder and Hancock, to which repeated reference is made in the text, will be found in various Numbers of the 'Annals and Magazine of Natural History' for the years 1852 and 1853. To the attentive perusal of these valuable essays the student of the subject is earnestly advised.

but these are all that have come under my observation*." In each of these "modifications" one common character is said to prevail—the vessels *reticulate*—in the first only "slightly," in the others more "minutely." Such is the structural law of the branchiæ of the Lamellibranchiates as expounded by Mr. Hancock. It is at direct variance with the prevalent and accepted definition. Dr. Sharpey says—"Each gill (of *Mytilus*) or leaf consists of two layers, which are made up of vessels set very close to one another like the teeth of a comb or like parallel bars, &c.†" "These bars are connected laterally with the adjacent ones of the same layer at short intervals by round projections on their sides," &c.‡

Here this accurate observer conspicuously indicates the difference between the solid projections interposed between the vascular bands, tying them together into a horizontal lamina, and the "transverse plicæ" of Mr. Hancock, which transform a matchless system of parallel bars into one of "minute reticulation" (Hancock), which neither the eye nor the understanding can unravel. M. Deshayes§ stands in this anomalous position:—he has figured accurately what he has interpreted wrongly. Albeit to this author merit is due. He has pointed out clearly by the pencil—what really exists in nature; what he himself misunderstood; what neither Dr. Sharpey nor Mr. Hancock seem at any time to have recognized—a structure without which the gill of the Lamellibranch could not architecturally be what it is; a marvellously woven fabric, refined in the utmost degree in its mechanism, adapted with incomparable skill to the purpose in view—a structure which no observer either anterior or posterior to the time of M. Deshayes has even suspected to exist—that apparatus of transverse scaffolding (Pl. VI. fig. 1 *c*, *c*, *d*) situated between the lamellæ of the gill, crossing at right angles the axes of the interlamellar water-tubes, *j*, *j* (of the existence of which M. Deshayes had not the slightest knowledge), and doubtfully described by him as the true *blood-channels* of the branchiæ! M. Deshayes mistook the laminæ formed by the real branchial vessels for "*membranous* layers or laminæ, *within the substance of which the branchial vessels* are arranged with great regularity." His *eye* caught with correctness nearly *all the parts* of this exquisite apparatus; his reasoning then enveloped them in

* Annals and Magazine of Natural History for April 1853.

† Art. "Cilia," Cyclop. of Anat. and Phys.

‡ This concise description is rendered still clearer by the original figures which accompany the famed article of Dr. Sharpey, to which I have adverted in the text.

§ See art. "Conchifera," Cyclop. of Anat. and Phys.

confusion*. Nothing less than a *rediscovery* of these skeletal parts, by which the branchial vessels are maintained *in situ* and the whole tubular system preserved in shape, could suffice to render complete and consistent the demonstration of the anatomy of the Lamellibranchiate gills.

With the eye stedfastly fixed on these parts, it is surprising that M. Deshayes could see in them no meaning, could read in them no purpose. In defining the outline and office of the interlamellar water-tubes, it is matter of wonder that Mr. Hancock did not suspect the mechanical *necessity* for a supporting apparatus such as this, without which the water-tubes could not sustain their patency or their form. Such is the history of progress in all the manifold paths of scientific observation. Discovery must literally be *prefigured* in the intellect of the thinking observer. In the absence of the foreshadowing conception, wondrous things in nature rendered manifest by accident are vacantly gazed at, left unfathomed, and then forgotten, or mentioned only as incidents or episodes in the drama. The merit which belongs to rediscovery is too often withheld from its author. It is morally, in equity, not less worthy of honour than the first discovery.

The law was formerly stated that the blood-channels in the gills of the Lamellibranchiate mollusks occur always in every species in form of straight, parallel, independent, non-communicating tubes, supported on the two opposite sides by hyaline cartilages, generally membraniform and semicylindrically curved (Pl. VI. figs. 6 & 6^a; and Pl. VII. fig. 15). These blood-channels never reticulate. At the free border of the gill the afferent channel returns into the efferent in a looping manner (Pl. VI. fig. 1 *e*, *f*). The efferent like the afferent channel preserves its individuality from one border of the lamella to the other (*m*, *e*). The blood-current, therefore, preserves unmixedly its singularity and independence from the beginning to the end of its branchial orbit (Pl. VIII. figs. 23 & 24). This is a striking and remarkable characteristic. It is a mollusean peculiarity. Its prevalence in this class is universal. It stands in contrast with the crustacean. The network plan is here the type. In some species of Annelids the branchial vessels observe a straight, parallel, looping mode of division. In the Annelid the blood is coloured and non-corpuscular. In the mollusk it is replete with globules. The blood-globules travel through the branchial 'bars' in a single series, or two abreast. In the crustacean the blood-channels are impartetal sinuses. In the mollusk each

* The reader is requested to refer to fig. 352, article "Conchifera," in the first volume of the 'Cyclopædia of Anatomy and Physiology.'

vessel is elaborately carved and wonderfully protected (Pl. VI. figs. 6 & 6^a). The difference is as essential in kind as it is conspicuous: it may serve hereafter to establish the true direction of equivocal affinities. Subtle analogies, like deeply hidden differences concealed amid the profoundest recesses of the organism, are often more conclusive in disputative questions of specific and generic relationships, than diversities or resemblances graven prominently in the manner of the outward form.

In now entering upon the narrative of the minute structure of the gills in the conchiferous mollusks, it must be premised that illustrative types only can be comprehended in the story. Specific varieties and modifications must be left to the specific inquiries of individual observers. There prevails, however, such a remarkable uniformity in the architectural principle on which the breathing organs in all Lamellibranchiate mollusks are constructed, that departures from the central plan never involve a change of type. Such variations are apparent, not radical. Though a concise description, aided by illustrations, may enable the author to convey a readily intelligible statement of these parts, the reader must not infer that his task has been easy or his labour light. He has traversed dark and tangled controversies. For long he could pilot his course by the magnetism of no clearly-defined principle. Evidence conflicted, assertions bewildered; the subject was intricate, the clue of *principle* was wanting. He would fain trust that the history which he is about to write will transform a pre-existing chaos into the cultivated scene of exact demonstration.

The minute structure of the gills in the Conchifera may be conveniently described under the heads severally of the constituent parts of which they are formed.

1. The parallel bars or vessels forming the lamellæ.
2. The borders of the lamellæ, (*a*) attached, (*b*) free.
3. The transverse connective parts—intervascular, or intervectal.
4. The interlamellar water-tubes and the *intra-tubular framework* of support.
5. The ciliary system of the gills.

1. In the Acephalous mollusk the branchial vessel is sculptured upon one essential plan. All deviations from this plan are inessential varieties. So singularly do these blood-canals differ from ordinary blood-vessels, that they will be henceforth described under the name of "*branchial bars*." The word 'bar' implies, first, straightness, and secondly, rigidity, two properties which belong to the branchial bars. The word 'bar' involves the idea of separateness, individuality and independence—characters which apply to the branchial bars. Rigid bars arranged

in parallel directions on the same horizontal plane would form a *stratum of bars*—such is the branchial lamella. Disposed on two coincident planes, one above the other, two parallel lamellæ would result. Between parallelly-arranged rigid bars the *interspaces* would be parallel and equal—such are the *intervectal** water-passages of the branchiæ. If traversed by cross threads at frequent intervals, a long fissure would assume the form of oblong foramina (Pl. VI. fig. 1 *g*, fig. 2 *e*). Such sometimes are the varieties which occur in the intervectal passages. If the parallel lamellæ be tied together at regular points by bands running with the bars, the space between the lamellæ would be divided into tubes. Thus are formed the interlamellar water-tubes (figs. 7, 9). The picture is faithful to nature. It mirrors the reality of a complex apparatus. It represents in simple outline the machinery of the branchiæ in the bivalve mollusk.

The details are now neither intricate nor unintelligible, because the *constructive idea* is clear to the intellect. In all investigations a tangibly-grasped *mental* picture must forerun the clear perception of the outward reality.

A branchial bar is a *tube* whose sides are comparatively rigid, and whose diameter is uniform (Pl. VI. fig. 6 *a*, *a*). It is clothed externally by a membrane, the continuation of the mantle, of which the epithelium is evolved at certain regular lines into cilia-bearing scales (*b*). The opposed sides of each bar are formed of, and supported by, cartilages (*a*, *a*). If these two cartilages were far removed apart, the blood-channel would be broad and flat (Pl. VII. fig. 15 *b*, *b*, *b*). These cartilages are slender in the extreme in texture; they are membraniform and exquisitely hyaline; curved at the edges, they assume the figure of a hollow semicylinder; they possess just enough rigidity to preserve the straightness of the bar; they are *continuous* throughout the whole length of the bar (Pl. VIII. fig. 17). Being placed on the opposed horizontal sides of the bars (not on the upper and under aspects), they must necessarily circumscribe a tubular channel of unbroken continuity. The sides are not perforated by openings of any description. If the transverse structures (Pl. VIII. fig. 22 *a*), afterwards to be described, be *vessels* or blood-channels, as conceived by Mr. Hancock and some of the elder anatomists, the bore of such channels cannot communicate with that of the parallel bars. The transverse parts must therefore, if they be blood-channels at all, constitute an independent system. But they are *not* so. They are connective fibrous structures (Pl. VIII. fig. 19). In almost all species of bivalve mollusks, the branchial bars more or less closely approach the cylindrical in figure. To this rule of structure those of the common Mussel form a remarkable exception: they are here blade-shaped (figs. 17 & 20). The section of the bar is frequently oval. In the genera *Cardium*, *Unio*, *Ostrea*, &c. this form is exemplified. The subcylindrical canal, circumscribed by the hyaline cartilages just described, is the true blood-channel*. All naturalists have conjectured this fact; the existence is now only for the first time *proved*. The cartilages bounding these channels are now first announced. They do not enclose the whole circumference of the vessel: they form a third of the opposite halves (Pl. VI. fig. 6[2]). The rows of cilia correspond with their edges: the intervals between these edges are membranous. The real osmotic movement of the gases concerned in respiration is limited to these intervals. Along these intervals, extending with beautiful regularity from one end of the bar to the other, there travels a cilia-driven current. In *Mytilus* the bars appear to swell (Pl. VIII. fig. 17 *o*, *o*, *o*) at the points at which they are joined together by the transverse structures. The real blood-channel does not bulge. The cartilages of the bars at the base of the lamella are lost in and identified with that embracing the trunk common to the whole series (*c*, *c*). Traced carefully to the proximal border, they will be observed to have this disposition: the cartilages of contiguous sides of *adjoining* bars form one piece, being so bent as to become continuous at the proximal border of the lamella. The bars are thus held firmly *in situ* and in relative connexion.

At this point it becomes extremely interesting to inquire, whether the *lamella* is composed of a single series (Pl. VI. fig. 4), laid side by side, of parallel bars, or of a double series arranged in two separate planes? (fig. 5). The answer to this question will implicate an important point of function. It is difficult to convey clearly the idea of a double series of bars constituting a *single* lamella. This undoubtedly is the disposi-

* From the Latin *vectis*, a bar. Since it is proposed to distinguish the branchial blood-channels under the name of bars, it is only consistent to mark the spaces between them as *intervectal*, rather than as intervascular.

* A very recent study of the minute structure of the gills in the Tunicates and Ascidians has enabled me to resolve completely the homology of the *branchial bars* in the bivalve mollusks, to explain demonstratively why it is that in the gills of some Acephalans the blood-conduits are placed like membranous channels between *alternate bars* (as is shown in Pl. VII. fig. 15, *b*, *b*, *b*), and that in others the blood-canal (as in Pl. VIII. fig. 22) occupies the *axis* of each bar. Though there exist in the gills of the Tunicata a system of large *transverse* trunks, with which the *parallel* ultimate blood-channels (the homologues of the "bars" in the Acephala) openly communicate, in a *supplementary note on this subject* in the next paper, it will be shown that the ultimate elements of the branchiæ in Tunicata and Acephala are really arranged on the same type.

tion of the branchial bars in some species of Acephala. If a "bar" be bent once upon itself (fig. 8), and if then one limb only be rested upon a flat surface, the other limb will be on the same vertical plane, but on a different horizontal plane. If a second, then a third bar, and so on, be placed in coincident directions, the limbs will form two horizontal series or laminæ, between which a free undivided horizontal space will exist (*e*, *f*); but there will also exist vertical spaces between each two adjoining bars having the same vertical planes. In words this arrangement is complex, in illustration simple. Now it may at first be supposed that of mechanical necessity this must be the order in which the bars are arranged in all the examples of double gills* (Pl. VI. figs. 1 & 7), as it is really that in which the afferent and efferent limbs of the same looped bar are disposed in all instances, without exception, of *single* (Pl. VI. fig. 2) gills. But it is truly the case only in a very few genera. It is so in the Mytilidæ (Pl. VIII. fig. 24). It follows that under the latter circumstances the interlamellar water-tubes must be bounded by two concentric walls (Pl. VI. fig. 5), each wall being composed of a single horizontal series of bars. Of this disposition another apparent example is afforded in the Ostreadæ; if the disposition of the loops at the free margin *only* be considered. In nearly *all* other genera, known to the author, the limbs of the same looped bar are placed on the same horizontal plane (Pl. VI. fig. 7 *f*). The plane of the loop notwithstanding at the distal border of the lamella is not horizontal, but vertical. It results that each lamella is composed of a single series of bars, though the contiguous limbs alternate in function, one conveying a centripetal, the other a centrifugal current (Pl. VII. figs. 9 & 11). But it must be remembered that a single lamella (*a* or *b*, Pl. VII. fig. 11) of a double gill is not the exact equivalent of an entire single gill (fig. 14). In *all* single gills the limbs of the same bar rest on vertical planes; those of a single lamella of a double gill are placed on the same horizontal plane (fig. 12). In the single gill the physical conditions are more favourable to the complete aëration of the blood. The water-currents are different, not the same. It will greatly facilitate the comprehension of the preceding history if now *the minute anatomy of the free or distal borders of the branchial lamellæ be carefully and accurately studied.*

The structure of the extreme free edge of the lamella furnishes a ready key which unlocks at once the whole mystery of the branchial apparatus; and yet this wondrous part of the organ

* The meaning attached in these papers to the *double* as opposed to the *single* gill is afterwards explained.

has never arrested the curiosity of the anatomist. In *Mytilus* and in *Mytilus* only, Dr. Sharpey figures correctly the manner in which, at the distal margin of the lamella, the bars of the upper become continuous with those of the lower lamella. In *Mytilus* the structure of the gill is almost unique (Pl. VIII. fig. 17). The order which obtains in nearly all other genera could not be deduced from the anatomy of the Mytilidan gill. It is a singular exception. It is the rare exception only that Dr. Sharpey has pictured. The *rule* of structure remained really to be discovered. If the blunt and acute edges of the penknife-shaped branchial bar (fig. 20) carry each a blood-channel, then each *gill* in *Mytilus* will be a double gill, for the upper and lower are identically formed. If, on the contrary, the blood-channel exists only at the blunt edge (*a*) of the blade, the current travelling peripherally along the bars of the upper lamella (A, fig. 17) must turn round (as shown in fig. 24 *a*, *a'*) at the free margin through the loop and move centrally along the bars of the lower lamella (B, fig. 17). In the latter case the gill would be single, in the former double. The bars of the upper lamella when the gill is single carry currents moving in the same direction (Pl. VI. fig. 2; Pl. VII. fig. 14) from one border of the gill-plate to the other; those of the lower, oppositely tending currents (Pl. VIII. fig. 23 *e*, *f*). This point is the wonder-striking feature of the branchial engineery. No writer has ever given to it a single thought. It deserves to be further elucidated. In *Pholas* (Pl. VI. figs. 1 & 2), *Gastrochæna*, *Mya*, *Tellina*, *Mactra*, *Cyprina*, *Cardium* (Pl. VII. figs. 13 & 14), *Ostrea* (Pl. VIII. fig. 21), and probably in many other genera, the inner gill is *double* and the outer is *single*. The Pandoridæ and Lucinidæ are families in which the outer gill is altogether suppressed. In *Solen*, *Pecten*, *Unio*, *Venus*, *Kellia*, *Arca*, &c., the two gills on both sides are equal in size and *double* in structure.

Every gill-plate, whether single or double, is composed of two lamellæ, between which the excurrent water-tubes (see large arrows in Pl. VII. fig. 13, Pl. VI. fig. 1, and figs. 7 & 8) are situated. In the example of the double gill *each lamella* is the scene of a double system of opposed currents of blood, since the two limbs of the same looped bar lie on the same horizontal plane in the same lamella (Pl. VII. fig. 9 *c*). The adjoining limbs are thus alternately afferent and efferent, or venous and arterial. *Each lamella* then of every double gill is a complete and independent gill. Its system of circulation is distinct, and totally unconnected with that of the other lamella. Nevertheless, a single lamella of a double gill is not identical in anatomical characters, or structurally, or perhaps officially, equi-

valent to an entire single gill. As formerly intimated in the example of a single gill, the limbs of the same looped bar, respectively venous and arterial, are placed on different horizontal planes (Pl. VII. fig. 14), the planes of the *loops* (*a*) at the free margin being vertical, and not horizontal as they are in general in the double gill (Pl. VI. fig. 1; Pl. VII. figs. 9 & 11). The single gill, like the double, is composed of two lamellar planes (fig. 14 *b*, *c*) bounding intermediate water-tubes. But in the single gill each lamella is single in function, since it consists of the afferent or efferent limbs separately and exclusively of the looped bars. In either lamella therefore the adjacent bars belong to separate and independent loops. The component bars of the lamellæ in all single gills are separated from one another by intervectal water-fissures (Pl. VII. fig. 15 *c*, *c*, *c*). In the double gills in which the two limbs of the same loop lie adjoined on the same horizontal plane, such limbs are united together by a *continuous membrane* (Pl. VI. fig. 5). In such case the intervectal water-fissures exist only between the limbs of different contiguous loops, not between those of the same looped bar. By this arrangement the volume of water which traverses the gill at any given time is reduced by exactly one-half. The functional value of the organ therefore sinks in the same degree. A *double* gill (Pl. VII. figs. 13, 9 & 11; Pl. VI. fig. 1) in structure is not necessarily twofold in physiological import. In official activity it exceeds little the single gill. In the latter the blood is more intimately brought into contact with the respiratory medium, and this medium is more readily and rapidly renewed. To the single gill (Pl. VI. fig. 2; Pl. VII. fig. 14) conchologists have applied the term *supplementary*. It is difficult to understand in what sense this term should be received. In *structure* the single gill is *not* supplementary. It is a perfect and complete organ. No constituent element is deficient or suppressed. In function it is complete. It is not a supernumerary organ. Both these designations are significant of what is untrue. It is as much an integer of the organism as the upper or inner gill. A *law* hitherto undiscovered does, however, affect the presence and dimensions of the outer or single gill which does not influence the inner or double gill. If, as in the Pandoridæ, Lucinidæ, and some other families, there exist only one gill, it is invariably the single or out-gill that is wanting. The principle of suppression or non-development affects exclusively the latter. When only one gill exists, that is, one on either side of the foot and body, it is always *double* in structure. It contains the same number of bars and loops as any other double gill. It is quite erroneous to conceive that in such a case the absent or suppressed gill has been fused into and identified with the present solitary gill. The latter is the same in essential structure as if the single gill were present.

In *Pholadomya* and *Anatina*, Professor Owen describes the two branchial lamellæ of either side as having been united to form a single gill*. Valenciennes states that the solitary gill of the family Lucinidæ resembles that of *Anodonta*; it is larger, and formed of thicker and more prominent pectinations. *Lucina Jamaicensis*, *L. tigrina*, *L. columbella*, and *L. lactea*, are examples in which only a single branchial organ exists on either side. The solitary gill differs from the ordinary double gill only in *apparent* characters. The free border is composed only of two rows of loops; but these loops are soldered together by an obvious longitudinal band or cord, running in shape of a deep water-groove from one end to the other of the free margin (Pl. VI. fig. 3 *b*). It is this character which occasions the appearance of doubleness and fusion. In the solitary gill of the Pandoridæ and Lucinidæ, the pectinations† of the lamellæ are coarse and large to the naked eye. This circumstance is due to the greater size in these cases of the interlamellar water-tubes. It is repeated, that the vascular elements, in the solitary gills, are the same in number and disposition with those of any other double gill. If, in the example of the solitary gill, the outer single gill were really organically united to the inner double gill, an organ should result consisting at the free border of *three* rows of vascular loops, *two* distinct systems of parallel interlamellar water-tubes, *four* separate lamellæ, *three* layers of afferent and *three* of efferent bars! Such, of mechanical necessity, should be the anatomical characters of a gill which owed its formation to the union of one already double to another struck on the single plan. Such a monstrosity is not illustrated in nature. It is a fabulous branchia, born of hypothesis. But it *may appear* quite reasonable to explain the anomaly of a solitary gill, on the supposition that it is the natural and necessary product of the fusion of two *single* gills. A glance at the illustrations depictive of the type of the latter, will at once convince the mechanician that two single gills could not in any manner be fused in order to make a double gill,—such a double gill, that is, duplex in mechanism, twofold in function, as actually exists in the real animal. Let two single gills be brought together (Pl. VI. figs. 7 & 8),—the water-movement and the ciliary action would cease at once on the two adjoined, apposed faces. Thus the power of each would be reduced by one-half. Two singles united make a single! Such is the clumsiness of human handi-

* Forbes and Hanley, British Mollusca, vol. ii. p. 42.

† It should be distinctly understood, that the word '*pectinations*' is not synonymous with an ultimate branchial bar, but with that *set* of bars which form an interlamellar water-tube.

craft: attempting to mimic nature, it is lost in caricature. Nature does not reach her ends by the "fusion" of organs. An existing organ is *modified* to fulfil a collateral purpose. A solitary gill has its own peculiar characters. The component vessels remaining unchanged in number and arrangement, a solitary organ is rendered equivalent to a double one, by augmenting the dimensions of the passages and tubes in such a manner, that the aërating element brought into relation with the blood can be increased almost to any amount. *Function* is thus intensified, while structure remains unaltered.

The *loops of the vascular bars*, as they project at the free margin of the lamellæ, are differently joined and variously figured and sculptured in different genera, and frequently in different species of the same genus. In *Pholas* (Pl. VI. figs. 1 & 4), the free border of the double or inner gill presents two rows of loops (*e, f*). The plane on which the loops of the upper lamella rest is horizontal, coinciding with the *length*, as opposed to the breadth, of the gill. Those belonging to the lower lamella of the same gill, form a row on a plane an eighth of an inch below the former. Between these two projecting scalloped edges, a groove (fig. 3 *b*) runs from one end of the gill to the other. The cilia which fringe this groove (Pl. VIII. fig. 24 *h*; Pl. VII. fig. 10, *a, b*) are very much larger in all species than those which are distributed over the bars at the plane faces of the gills. They excite a vigorous current, bearing towards the mouth. Those of the flat surface (Pl. VIII. fig. 20; Pl. VI. fig. 6 *b, b*) raise streams, tending towards the free border of the gill. Both are subservient to alimentation and respiration. In *Pholas*, then, the double gill (Pl. VI. fig. 1) is composed only of two lamellæ, like the single gill (Pl. VI. fig. 2); but in the former, each lamella is composed of two orders of bars, in the latter of one order only. The two limbs (fig. 4 *b, b, c, c*) of each looped bar in the former are placed on the *same* side of the intermediate water-tube: the afferent and efferent limbs of the same bars (fig. 2 *h, f*), in the instance of the single gill, are so *opened* or separated at the free margin as to form respectively the *opposite* walls of the included water-tube. The vascular *loops* at the margin of the double gill in nearly all genera are disposed flatwise (Pl. VI. fig. 1 *e, f*; Pl. VIII. fig. 21 *e, b*; Pl. VII. fig. 9 *a, b*, fig. 11 *a, b*), so that all the loops of the same lamella form one horizontal plane. Those of the single gill (Pl. VII. fig. 14 *a*; Pl. VI. fig. 2) are placed vertically, so that the plane of each loop is separated from, though parallel with, that of the adjoining loops. In the double gills of the Cardiadæ an exception occurs, and probably in other families. The loops at the distal margin are disposed here on vertical planes (Pl. VII. fig. 14 *a*); but though standing verti-

cally, they do not enclose two systems of interlamellar water-tubes, but only *one*. The *mechanical* problem presented by the gills of *Cardium* proved extremely difficult of solution. *When understood*, it challenged any living mechanism for beauty and perfection. In *Mytilus* the loops of the two lamellæ are soldered into union at the free borders; they stand vertically (Pl. VIII. figs. 17 & 24); they circumscribe a deep intermediate gutter. In *Solen* (Pl. VIII. fig. 23) the loops expand. In *Venus* they also somewhat exceed in diameter that of the bars, of which they are *the bend*. In *Mytilus*, the inner and outer gills exhibit the same formation. In the Ostreadæ (Pl. VIII. fig. 21), the loops at the margins of the gills are so closely packed together horizontally, as to appear like a continuous membrane bounding an angular groove. Numerous other varieties in the mere shape and size of the loops occur in different families of Bivalves—the *type* of structure never changes.

The proximal or attached border (Pl. VI. figs. 1 & 2 *a, b*) of the gills occurs under many varieties of anatomical plan. *Pholas* exemplifies one type. The two lamellæ* are attached to the pallial tunic. All the vascular bars terminate in a common trunk (Pl. VI. fig. 2 *o, b*) which runs at right angles to their axes, and parallel with the length of the entire gill. There are two of these trunks, one afferent, the other efferent. They occupy respectively the proximal margins (Pl. VI. fig. 1 *a, b*) of the two lamellæ of which each gill is composed. In *Pholas* these trunks are supported by the framework of *solid* structure (*c c* and *d d*), which occupies the interlamellar spaces. In *Solen* (Pl. VIII. fig. 23 *a*) and *Mytilus* (fig. 17 A) another plan of formation is observed. Here the proximal border of the superior lamella of the upper gill, and inferior lamella of the under gill, are *unattached*, floating in the mantual cavity. In such instances the interlamellar framework is wanting. The vascular bars at this border, for some distance up the breadth of the gill, are tied together by means of a *continuous* membrane (*e, e*). Here the interlamellar scaffolding, and the water-tubes which the former assist to form, exist only where the two lamellæ are adherent; viz. over the two-thirds of the breadth of the gill nearest the free border.

* It should be clearly explained that the word *lamella*, as applied to the gill of the Acephalan Mollusk, should signify, one of the two plates of which the gill, whether double or single, is composed. The gill is the whole organ. In those instances in which (as shown in fig. 5, Pl. VI.) the bars stand vertically on the same lamella, then of course each lamella would be composed of two plates, or finer lamellæ. I am not quite certain that such an arrangement exists in nature. In several genera—in *Cardium* especially—when care is taken to avoid *pressure* upon the margin, such is the true position of the loops, if not of the bars proceeding backwards from them.

Intervectal and Interlamellar Framework of Connective Structures.

These structures constitute the true skeleton by which is sustained the vascular fabric of the gill. Of the latter, they determine the shape and the form. They preserve the blood-carrying bars in position. They hold apart the component lamellæ of the gills. They thus *form* the interlamellar tubes, since without these structures the lamellæ would fall together into contact and obliterate the tubes. Messrs. Alder and Hancock recognized the tubes, but overlooked the framework system by which they were constructed*. M. Deshayes has *figured* this framework (Pl. VI. fig. 1 *c*, *c*, *c* & *d*, fig. 2 *i*, and fig. 8 *d*, *d*) apparatus in a conchiferous (*Pecten* or *Arca*?) mollusk. Not a sentence is written descriptive of its characters, or interpretative of its meaning†. Attention was drawn to it by no allusion whatever, direct or incidental. Philippi‡ has this observation with respect to the branchiæ of *Solenomya*, which probably refers to the interlamellar structures in question:—"Branchiæ duo non quatuor, non lamelliformes, sed pectinatæ vel potius pennam exacte referentes, lamellis transversis perpendicularibus, carina media corpori per totam longitudinem adnatæ, versus apicem *ope ligamenti*." Ill-defined reference to the same parts is made by Carus, Blainville, Garner, and others. To be known descriptively, and comprehended physiologically, they remained really to be rediscovered,—to be read by a *new* eye, from a *new* point of view. The author believes that the following is the first systematic exposition on record of the anatomy and significance of the *non*-vascular elements of the lamellibranchiate gill.

They are classifiable under two heads. Those parts which are placed between (Pl. VI. fig. 1 *g*, fig. 2 *e*, fig. 8 *d*; Pl. VIII. fig. 19 *c*, *c*, *c*) the parallel bars (*the intervectal*), uniting them into the form of a leaf, constitute a separate order. Those, *secondly*, stronger, coarser, in some genera very conspicuous, in others very concealed, which separate the lamellæ, forming and bounding the excurrent interlamellar water-tubes, to which in many species the ova adhere, the basis of the whole gill, the wonder of the whole enginery, the last of the branchial constituents to be described and understood, are really a distinct and unknown class of structures.

The first class vary the apparent anatomy of the gill more than the second. They cut the fissural spaces (Pl. VI. fig. 7 *d*) between the individual bars, or individual loops, into oval stigmata (fig. 5 *d*), elliptical perforations, or lengthened parallelograms (Pl. VII. fig. 12 *d*). In the absence of them, as in *Thracia* (Pl. VII. fig. 15), the intervectal water-fissures are continuous from the free margin of the gill to the proximal. In *Mytilus* (Pl. VIII. fig. 17 *o*, *o*, *o*) they appear under the character of fleshy nodules; in *Cardium* (Pl. VII. fig. 12 *c*, *c*, *c*) they are almost invisible; in the Veneridæ they consist of a flattened bundle of slender threads, running from bar to bar at equal intervals; in *Pholas* they assume almost a membranous form (Pl. VI. fig. 4 *e*), perforated at regular distances by oval holes; they exist only between alternate loops. In a physiological sense, the highest value attaches to these intervectal parts. They determine the dimensions of the water-stigmata. If they are small, the water of respiration is very much subdivided; if large, the lamella is readily traversed by the aërating element. In calculating the quantum of respiration in the Conchifera, *two factors* demand to be estimated: first, the amount of blood entering the breathing organ; secondly, the volume of water by which, in a given time, it is capable of being traversed. The latter will depend upon the dimensions of the water-passages.

The intervectal connective structures have been mistaken for half a century by the best observers for *vessels, blood-canals crossing the bars*,—deceiving the observers into the idea that each lamella in the lamellibranchiate gill is really composed of a *network of blood-vessels*. This idea as regards the Acephala involves a fundamental error; it envelopes everything in unresolvable confusion. The orbit of the branchial circulation cannot be explained. It contradicts the anatomical arrangement conspicuous in other parts. A consistent sentiment cannot be shaped of this

* It is very probable, from the following passage, that Messrs. Alder and Hancock have mistaken the thick solid cords which at short intervals cross the *tubes*, for real blood-channels: "The laminæ forming the walls of these tubes were now examined through the microscope, when the whole was observed to present a regularly *reticulated* structure composed of blood-vessels; *those passing transversely being the stronger and more prominent*."—Annals and Magazine of Natural History, paper on Currents in *Pholas* and *Mya*," 1852.

† The following is the only passage which occurs in the excellent article (Conchifera, Cyclop. Anat. Phys.) of M. Deshayes having reference to the structure of the gills:—"In the greater number of genera, the branchiæ are formed of two membranous layers or laminæ (*a*, *b*, fig. 352), within the substance of which the branchial vessels descend with great regularity. In several genera, as the *Arcidæ* and *Pecten*, the branchial vessels, instead of being connected parallel to one another *within the thickness of a common membrane*, continue unconnected their entire length, and they are thus formed of a great number of extremely delicate filaments, attached by the base within the membranous pedicle on which the branchial veins pursue their way towards the auricle." Nothing is said of the distinct and independent structures which separate the laminæ. The condition, namely the *separation* of the laminæ—upon which depends the existence of the interlamellar water-tubes—is here *accidentally* stated; but neither the existence nor the meaning of such parts seem in the slightest degree to have been imagined by M. Deshayes.

‡ Moll. Sicil. i. p. 16.

most perfect mechanism. They are *not* blood-channels. They are elastic, fibrous structures, enacting a purely ligamentous part. They derive their supply of blood from that of the branchial bars. Their office is mechanical, not chemical.

The *intra-tubular structures* (Pl. VI. fig. 1 *k*, *k*, *k*, fig. 8 *d*, *d*; Pl. VIII. fig. 21 *f*, &c.) are neither less remarkable nor less important. Upon this interlamellar framework depend the whole characters of the gill. They hold the lamellæ apart at *definite* distances. They unite closely together the loops of these lamellæ at the free margin (Pl. VI. fig. 7 *a*, *b*); thus they *close up* cœcally the tubes at this border of the gill *. This single point of structure is the pivot whereon turns the action of the gill. If the tubes at this extremity were open (as suppositionally at Pl. VII. figs. 9 & 11), it is hydraulically certain that the water would take this course to pass from the extra- to the intra-branchial cavity; none would pass between the bars which contain the blood; the function of respiration could not proceed; and this calamity, further, would ensue—no food could be carried to the mouth. Men do not value health until it is lost! Spectators see not, *feel* not, the perfection, the unimprovableness of organic mechanism until an element is ideally removed—until some deviation from nature's method of working is *supposed*! The *argumentum ad absurdum* startles by the bungle and foolery which it is sure to introduce; *then* philosophers realize the inimitableness of her certainty and refinement.

As the proximal borders of the lamellæ (Pl. VI. fig. 1 *a*, *b*) are separated by the whole diameter of the water-tubes, and as the distal margins are fused together, it follows that these tubes, like rivers, are small and shallow at their commencement, deep and broad at their termination. This arrangement favours their *suctorial* action. The water, as first explained by Mr. Hancock, is undoubtedly *drawn into* (properly *pushed* into) these tubes through the lamellar stigmata (Pl. VIII. fig. 17 *f*, *f*, *f*) from the pallial cavity. The water is discharged from the tubes by ciliary agency, which is constant (arrows in Pl. VII. figs. 9, 11, 13 & 14). They are thus constantly being emptied. If they were not refilled from without, they would become *vacuous*. During the action of the gill, there is momentarily generated a tendency to a *vacuum*. The pressure that is on the tubular side of the

* In the accompanying illustrations, in several instances (Pl. VII. fig. 9 & 11, Pl. VIII. fig. 21), these tubes are represented as if they were *open* at this margin of the gill. This method of illustration was adopted only for the sake of clearness, and in order that the disposition of the loops and bars of each lamella may be readily understood. In all cases, without a single exception, the tubes are *closed* at this border of the gill by the apposition of the loops of the two constituent lamellæ.

lamella is diminished; on the other it remains the same as long as the animal continues in the water. It is hydraulically inevitable, even *without* the assisting agency of cilia, that the water must transude the lamella by way of its intervectal fissures and perforations. This mechanism could neither be conceived nor explained before the nature and office of the interlamellar framework were brought under clear demonstration. It is important to understand, that that surface of the lamellæ which faces the intermediate tubes, namely the *internal* walls (Pl. VI. fig. 8 *e*) of the water-tubes themselves, is far less richly ciliated than the external surface. The excurrent movement of the respiratory water is much aided by the action of the connective structures of the bars and tubes. The intervectal pieces—those which pass crosswise from bar to bar by approximating the latter—are capable of stopping up the intervectal stigmata,—of suspending, therefore, the act of respiration. Thus is prevented the passage of irritating substances through the branchial lamella. The alternate movements of the shutting and opening of the bars is as important to the sieving operation of the gill, its prehensile function, as the cilia themselves. The transverse intervectal pieces (Pl. VII. fig. 12 *c*, *c*, *c*; Pl. VIII. fig. 19 *c*, *c*, *c*) consist of irritable and contractile tissue. They are capable, in part, of voluntary contraction. Thus, although the chemical act of breathing is in itself uncontrollable, it may be interrupted by the exercise of those connected parts which are subject to the will.

The skeleton of *solid* pieces (Pl. VI. fig. 8 *d*, *d*, fig. 1 *c*, *d*, fig. 2 *i*, *j*, &c.) by which the *tubes* and the *lamellæ* are supported and held apart, exists probably in the branchiæ of *every lamellibranchiate mollusk*. It constitutes a framework system, though anatomically distinct from, having a mechanical action concurrent with, the intervectal. At the free border this interlamellar substance is thin, slender, and difficultly detected by the eye, admitting of the falling together of the lamellæ and of the closing of the tubes. At the opposite attached border, the interlamellar substance is much thicker, coarser, and more conspicuous (Pl. VI. fig. 1 *k*, *k*, *k*); the parts being quite apparent *through* the lamellæ. Here, therefore, the lamellæ are further separated, and the tubes of the greatest diameter. This framework consists of two distinct pieces,—those, first, which run parallel with the vascular bars (Pl. VI. fig. 1 *e*, fig. 2 *i*); and, secondly, those which transversely connect these longitudinal pieces (fig. 1 *d*, fig. 2 *j*, *j*). The former limit the breadth of the interlamellar water-tubes. The tubes are capacious when the lamellæ are far apart, small when they are near each other. As the exterior appearance of "pectinations" in the gill is due to

the presence of these tubes, the pectinations are obvious, as in *Cardium, Solen, Pecten, Thracia*, &c., when the tubes are large, invisible to the naked eye when they are small. The *cross pieces* tie together the longitudinal at regular intervals. The latter run with the tubes, and *divide them* from one another, the former cross them. If the transverse pieces were so thick and large as to fill up the tube, and interrupt its continuity, the excurrents of water of course would be arrested, and the function of the gill would be suspended. It is far otherwise. They traverse the tubes in form of cords. Their extremities are attached to the opposed points on the horizontal sides,—to those very lines along the sides of the tubes at which the chemical act of breathing is passive. From this arrangement there flows this most beautiful result: the water, having permeated the lamella and gained the *interior* of the tubes, in its course towards the excurrent siphon, *is made to keep continually in contact with the branchial bars*. By this simple arrangement, the *two* sides, in fact the four sides, of each individual blood-carrying bar are *persistently* embraced by a moving current of the respiratory element! If the cross cords did not exist, every drop of water which entered the tube would collect at the most depending side, and flow out as *a useless and unused stream*. In the œconomics of nature, the subtlest œconomist may well marvel at her care!

The long pieces of this interlamellar framework are capable of shortening the length of the water-tubes, the cross pieces of diminishing their diameters. These actions impel, interrupt, facilitate, &c. the breath-giving currents. While they complicate the branchial machinery, they double the certainty of the process; they provide against accidents; they preserve in the required position the slender, tender, beautiful parts of which the apparatus is composed.

Endless diversities occur, in different species and genera, in the size, the figure, the visibleness, &c. of this interlamellar framework. In no single instance is there observable the slightest departure in *principle* of structure, in intention, in purpose, from the typal plan unfolded in the preceding description.

The *cilia-bearing epithelium* (Pl. VI. fig. 6 *b*) of the branchial lamellæ in the conchiferous bivalves is well known. It has been well described by trustworthy observers, from Leeuwenhoek to Quekett. The cilia in all cases are distributed in rows on the bars (Pl. VII. fig. 15; Pl. VIII. figs. 20 & 22). There are two rows on each external hemi-cylinder of each bar. On the *external* aspect of each bar, therefore, there are four lines of cilia (Pl. VII. fig. 10 *b*, *a*). They drive two currents in intersecting directions. On the interna aspect of each bar, that namely which faces the

interlamellar water-tubes, these rows of cilia are single on the opposite sides. The excurrent streams are consequently driven by a power which is one-half in amount of that by which the water is propelled through the lamellæ into the tubes. No cilia are detectable on the supporting framework between the lamellæ. The current raised by the cilia which are distributed on the external surface of the lamellæ tends in the direction of the free border; that excited by the internally-placed cilia bears towards the proximal border, coinciding with the outlet of the interlamellar tubes. It is a true ciliary current; but it is reinforced, quickened from time to time, by the contractile, voluntary action of the musculo-fibrous parts which constitute the intervectal and interlamellar framework. The cilia which fringe the free border of the gill propel the water in the direction of the mouth, at right angles, consequently, with that raised by those covering the flat surfaces of the gill. It is an alimentary, not a respiratory current. It is powerful enough to bear on its waves pellets of food for the mouth. The true aërating currents travel along the *naked* lines between the rows of cilia. These are the ultimate scene of the respiratory process.

Thus ends an imperfect sketch. It is but a rude outline of a beautiful picture. Much is left to the industry of observers coming after the author. Magnetic thoughts, indicating the pole of truth, have been but hastily projected. May they stimulate others to a truth-admiring repetition of his labours!

EXPLANATION OF PLATES VI. VII. AND VIII.

PLATE VI.

Fig. 1. Complete view of the *double* gill of *Pholas candida*. The attached border is held upwards as it were. *a*, *b*, are the large afferent and efferent trunks (of which there is a *set* in the border of each lamella) communicating with the parallel vessels, as shown at (*a*) and (*b*); *c*, *c*, *c*, mark the longitudinal pieces of the intra-tubular framework of solid structures; *k*, *k*, *k*, show the mode in which these longitudinal pieces form and bound the interlamellar water-tubes (*j*, *j*, *j*); *d*, *d*, *d*, are the transverse pieces of the framework on which the lamellæ (*m*, *c*) of parallel vessels rest, and by which the latter are held apart, and which cross at right angles the axes of the interlamellar water-tubes; *e*, *f*, denote the *two* series of loops of which the free border of this *double* gill is composed; they lie on two distinct planes one above the other; they are shown *as if they were few in number and far between*; they exist in nature in innumerable multitudes and packed with dense closeness. *m*, *n*, represent the course of the bars in the upper lamella; *e*, *i*, that of the lower. The arrows at the base of the gill emerge out of the interlamellar water-passages. *g*, intervascular *fibrous connecting* pieces.

Fig. 2. Is a view of a small portion of the entire *single* or *supplementary* (sic) gill in *Pholas candida*. *a*, *b*, afferent and efferent trunks giving and

receiving the blood of *two separate lamellæ*, not that of the *same* lamella as in the case of the double gill; *h*, *h*, bars of the upper lamella; *f*, those of the lower; *e*, connecting fibrous threads tying together the bars; *d*, the bars of the lower lamella, *i*, *j*; *c*, *c*, the interlamellar framework as explained in the double gill. *g* & *f*. show the *single* row of loops of which the free margin of this single gill is composed: but here the *plane of each loop* is placed *vertically*, not *horizontally* as in the double gill.

Fig. 3. A diagram representing the arrangement of the loops, *a*, *a*, *c*, *d*, at the free margin of the gill in some species; *b*, is the water-channel conveying a current towards the mouth. The loops are placed on vertical planes. Each lamella in this case is composed of afferent or efferent vessels *exclusively*.

Fig. 4. Two loops from the free margin of the double gill of *Pholas*, showing the mode in which the *continuous* membrane, *a*, *a*, which in some bivalves, if not in *Pholas*, really forms the blood-channel. In such cases the bars, *b*, *b*, *c*, *c*, would be *solid*. The water-stigmata (intervectal orifices), *d*, are in such instances present only between alternate pairs of bars.

Fig. 5. Plan representing the manner in which the bars, *a* & *b*, of two separate lamellæ placed on the *same* vertical planes, but different horizontal planes, are tied together by vertical partitional perforated membranes (*e*); *d*, shows the water-orifices in that connecting structure (*e*) which unites the bars of the same lamella *horizontally*.

Fig. 6—6ª. Minute anatomy of the branchial bars in those cases in which the hyaline cartilages, *a*, *a*, are so closely approached as to form the boundary of the channel (*c*); *b*, *b*, pieces of the cilia-bearing epithelium stripped off.

Fig. 7. View of a piece of the entire gill of *Thracia convexa*. The free, distal margin is turned upwards. *a*, *b*, the double rows of *vertically disposed* loops of which this margin is made up; *g*, the contraction which occurs between the interlamellar water-tubes (*e*); *f*, one of the transverse pieces of the interlamellar framework in view, crossing the water-tube, indicated by the arrow.

Fig. 8. The same cut horizontally along the length of the bars. The free edge (*b*, *c*) of the upper lamella is left. *d*, *d*, show the mode in which the transverse pieces of the intra-tubular framework lie between the lamellæ, *a*, *b*, which constitute the walls of those tubes; *e*, *e*, arrows denoting the direction of the water-currents in the tubes.

Plate VII.

Fig. 9. A small piece of the free edge of the double gill of *Venus striatula*. *a*, *b*, loops of the bars of the two component lamellæ lying horizontally; the loops lobed (fig. 10) as they are in *Mytilus*. *c*, the afferent and efferent bars of a single loop. The arrow descends from the interlamellar water-tubes.

Fig. 10. One of the loops from fig. 9, enlarged, representing the lines and disposition of the vibratile cilia, *a*, *b*: *c*, is a fleshy nodule tying the loop to its neighbour.

Fig. 11. The free margin of the gill of a minute freshwater bivalve. It is produced with a view to illustrate the *continuous* membrane which in some cases ties the branchial bars together at the *free* border of the gill. *a*, *b*, the loops of the two lamellæ are shown as if they were separated by the intervening tube (*c*), but in *nature* the lamellæ are fused together at the free border, and the intermediate tubes are cæcal.

Fig. 12. A few looped bars from the preceding, magnified, showing the delicate transverse threads, *c*, *c*, *c*, which cross the branchial bars at distant intervals. They lie on the *inside* or *tubular* aspect of the lamellæ, and sometimes supersede the intra-tubular framework. The open spaces (*d*) between these cross threads are the "intervectal" water-orifices. *a*, *b*, are either vascular bars, or two rigid sides bounding an intermediate membranous channel, which then is the blood-channel.

Fig. 13. A small portion from the distal edge of the double or upper gill of *Cardium*. *a*, *b*, the upper and lower loops of which this border is composed. The planes of the loops have a vertical position in relation, that is, to the plane of the whole gill. *c*, the fleshy or membranous structure which unites the loops. It belongs probably to the intra-tubular apparatus, *d*, *d*. Although in *this* double gill, as in *all* double acephalan gills, the free edge is double, the interlamellar water-tubes *f*, and arrows *g*, are *single*. The double row of loops runs into one at a little distance from the margin, in order to form *one* system of tubes. This union of the bars is shown at *i* and *e*. *h*, indicates the fleshy structures,—a part of the intra-tubular cross bars by which the *lobes* or "pectinations" of the gill are held together.

Fig. 14. A portion of the free margin of the inferior or *single* gill of *Cardium*. This figure exhibits perfectly the manner in which the limbs (*b*)—(which, arranged in a linear series, form the upper wall of the tube (*d*), or the upper *lamella* of the gill)—of the *same* system of loops pass, by looping *vertically*, as shown at *a*, into those of the lower wall of the tubes, or, which is the same thing, into the lower *lamella* of the gill.

Fig. 15. Shows the *alternate mode* by which the membranous blood-channels (*b*, *b*, *b*) are formed by the *solid* bars (*a*, *a*, *a*, *a*) of contiguous, but distinct loops. In such a case, which is the normal type in the branchiæ of all Tunicata, the cilia are disposed in lines *only* on that side of each *bar* which is nearest to the *water-fissures* (*c*). These fissures, in such examples, are not crossed by transverse connecting threads.

Fig. 16. Represents one of the lobes, or tubular pectinations, from the single gill of *Cardium*, cut longitudinally, illustrating the mode in which the water-tubes are formed.

Plate VIII.

Fig. 17. The "bars" from the gill of *Mytilus*, followed throughout the whole of their minute anatomy. *a*, *b*, are the afferent and efferent blood-trunks, running along the attached margin of the upper lamella A. This border of the gill in *Mytilus* is represented as including *two* trunks, on the theory, as yet not quite proved, that the *two* edges, *c* and *c*, would then be the beginning of the blood-channel along the *blunt* edge, and *a*, *a*, *a*, would mark the termination of that, travelling along the acute margin of *the same* penknife-shaped process. *f*, *f*, are the corresponding trunks at the proximal border of the inferior lamella; *e*, *e*, mark the *continuous* membrane by which, in this gill, the bars are tied together at the proximal border, so that no water can pass between the bars; *f*, *f*, *f*, *f*, are open orifices between the fleshy nodules, *o*, *o*, *o*, *o*, by which the aërating water enters from without into the inter-

lamellar water-tubes; *q*, *q*, *q*, are the superior *lobes* of the *single* loop, of which the free margin of the gill in *Mytilus* is composed; *p*, is the lower lobe. The deep groove between these lobes is the great alimentary water-channel, bearing a current moving in the direction of the mouth. *l*, is a fleshy nodule, by which the loops are united into a series; *k*, *k*, *k*, are *horizontal* water-passages; *m*, *m*, fleshy nodules, which connect the contiguous bars. The system of arrows, *g*, *g*, *g*, indicate the direction of the great respiratory current, along the upper surface of the lamella; *i*, *i*, *i*, the lower: *both* having the same direction—towards the free margin of the gill.

Fig. 18. Is a transverse section of one tube from the gill of *Mytilus*, exhibiting the mode in which the laminæ *separate*, in order to form a tube (*a*, *b*).

Fig. 19. Four bars from the same gill, illustrating further the *vertical* parallel planes on which the penknife-shaped bars are placed, and the mode more exactly in which they are tied together.

Fig. 20. A magnified view of a minute portion of a *single* blade-like bar from the gill of *Mytilus*. It illustrates the distribution of the cilia; and the water-currents, denoted by the arrows, set in motion by them. *a*, *b*, are intended to show the position of the blood-channels in the axes of the thickened lines of either edge of the blade. If the upper lamella in the exceptional gills of *Mytilus* should be hereafter proved to carry only a *single system* (afferent or efferent) of blood-currents, the channel carrying such a single current must prove to be a *flat* passage, whose transverse section would extend from *a* to *b*; *e*, *e*, *&* *c*, fleshy nodules.

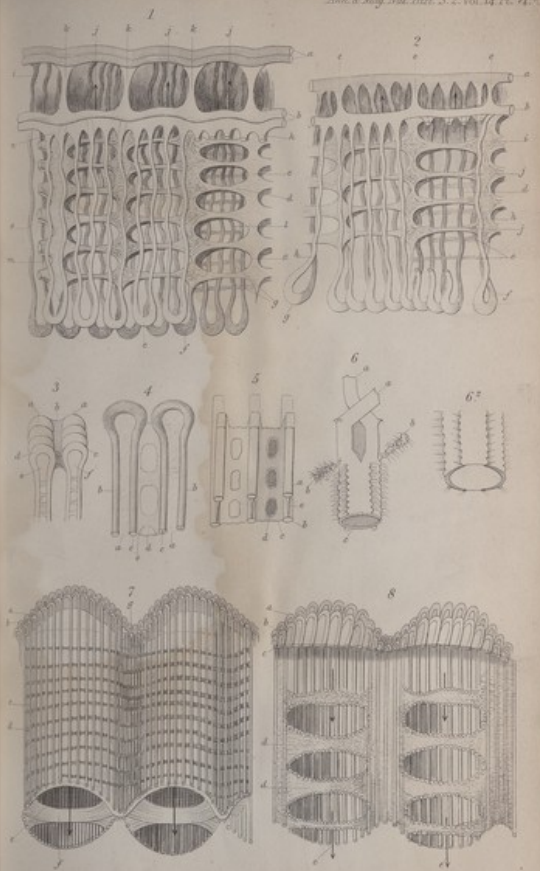
Fig. 21. Two longitudinal lobes, or pectinations, from the gill of the common Oyster. *a*, *c*, the *double* loops of which the free margin is composed. They are drawn as if separated from each other, in a tubular form; but, naturally, the two planes of loops lie in close apposition. At this border in *Ostrea*, the branchial bars are soldered together by a *continuous* membrane over the interval included in the dotted lines *g*. *f*, are the transverse pieces of the intra-tubular framework. By these transverse pieces, the lamellæ forming the water-tubes are sometimes drawn into quadrilateral figures, *d*, *e*.

Fig. 22. A few of the "bars" from the same gill, showing that each bar is an independent vessel. The component hyaline cartilages, *d*, *c*, are brought *close* together, so as to form a cylindrical channel, *e*. *a*, *b*, exhibit the transverse structures, as running along *one* (the internal) of the tubes. This arrangement proves that the latter cannot be transverse *vessels*.

Fig. 23. A portion of the *single* gill of *Venus*, drawn as an outline plan. *e*, *d*, *f*, mark the course of the blood, and the character of the blood-channel, from the attached margin (*a*) of the superior lamella, to the end of its course at the proximal border (*b*) of the inferior lamella.

Fig. 24. A second plan of the gill of *Mytilus*, constructed on the supposition that each *lamella* carries only a single blood-current, of which the beginning is shown at *d*, margin *a*, and the end at *d*, margin *b*. The border, *h*, depicts the order of the vibratile cilia.

[To be continued.]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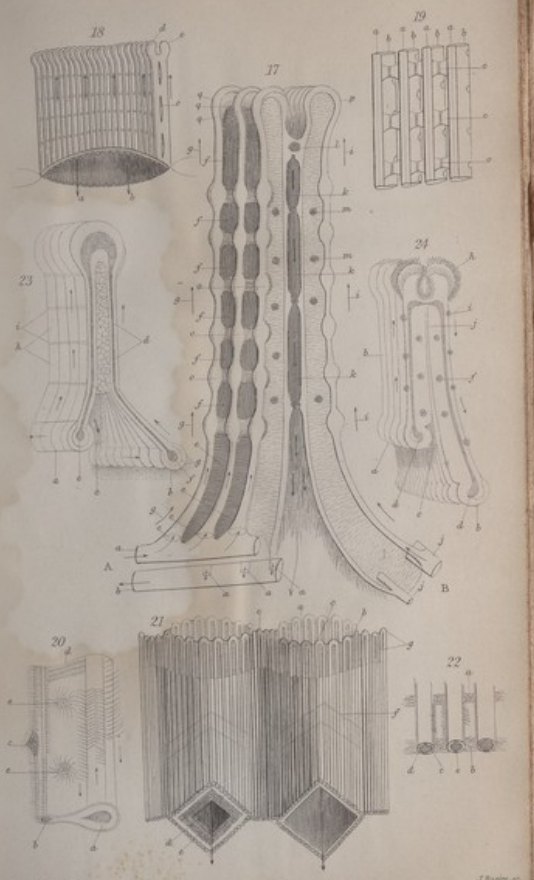
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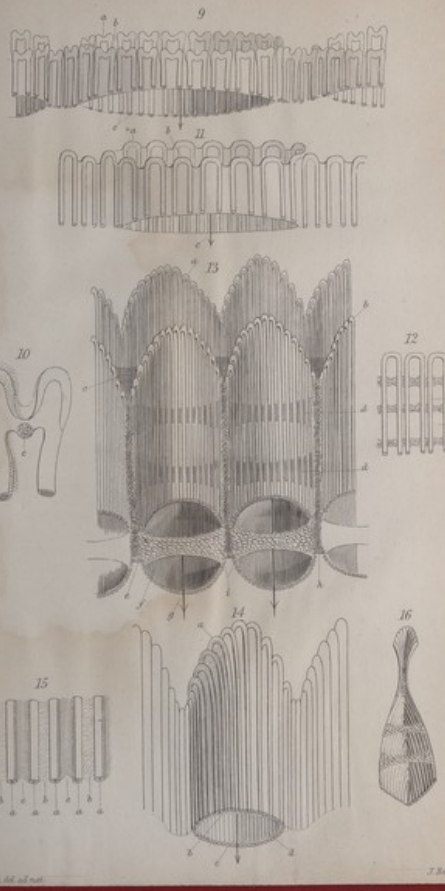
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LETTRE

DE

CHARLES MATTEUCCI

A Mr. H. BENCE JONES

F. R. S. &. &.

Editeur d'une brochure intitulée

ON ANIMAL ELECTRICITY

OU EXTRAIT DE DÉCOUVERTES

DE Mr. DU BOIS-REYMOND.

FLORENCE.

IMPRIMERIE LE MONNIER.

1853.

Monsieur.

Je viens de parcourir un livre intitulé: *On animal electricity: being an abstract of the discoveries of Emil Du Bois-Reymond*, et c'est à Vous, qui en êtes l'éditeur et le commentateur, que je crois juste d'adresser quelques réclamations contre les assertions erronées sur mes travaux d'électro-physiologie et les fausses insinuations sur mon caractère, qui sont contenues dans ce livre.

Je commence par déclarer que ce n'est pas une discussion scientifique sur mes travaux et sur ceux de M. Du Bois-Reymond que je veux entreprendre aujourd'hui; je ferai cela dans la nouvelle édition de mon Traité sur les phénomènes électro-physiologiques des animaux, dont les premières feuilles sont déjà imprimées. Je me bornerai seulement à prouver que tous les passages de ce livre où j'ai été cité, contiennent évidemment une exposition imparfaite ou erronée de mes recherches, à repousser tout ce qu'on m'attribue injustement, et à réclamer ce qui véritablement m'appartient.

En commençant par ce qui me blesse le plus dans votre livre, et qui est sous tous les rapports indigne d'une œuvre de science, je trouve à la page 23, Chap. II, qu'on dit en parlant de mes premiers travaux, qu'il y avait non seulement *a want of clearness but sometimes of fairness*. Je vous déclare qu'on ment dans ce passage et que cette opinion est sans fondement. En effet, la seule preuve que vous en donnez c'est que j'ai pris à M. Becquerel, ce que vous appelez une théorie des poissons électriques, et qui consiste à supposer que le cerveau de la torpille développe de l'électricité. Cette *théorie* a dû exister depuis le temps qu'on a observé la décharge électrique de la torpille; il serait aussi facile de l'abattre que de la créer, et certainement ni M. Becquerel ni moi n'y avons jamais attaché aucune importance:

c'est là cependant le fondement sur lequel vous établissez l'accusation de manque de bonne foi.

Vous prétendez dans ce même 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; mais d'un autre côté personne n'admettra qu'on doit, sans aucun intérêt pour la science, chercher dans ces publications des motifs pour affaiblir ou détruire les droits acquis par des travaux postérieurs. Je doute, que si on faisait l'histoire des travaux scientifiques avec cet esprit, on arriverait à diminuer le mérite de ceux qui sont bien autrement importants que les miens.

Commencez par admettre, qu'il est parfaitement prouvé par des documens incontestables, qu'avant l'année 1843 j'ai démontré l'existence du courant musculaire et de ses lois principales, et celles du courant propre et de la contraction induite à l'aide d'expériences d'autant plus évidentes qu'on peut les obtenir avec des instrumens ordinaires par la méthode des piles musculaires. (*Comptes-Rendus*, Séances du 6 Septembre 1841, du 21 Février et du 24 Octobre 1842; et *Annales de Chimie et de Physique*, 1838 et 1842.)

Parmi mes expériences de cette époque sur le courant propre, il y a celles qui prouvent évidemment les modifications souffertes par le courant de la grenouille sous les contractions tétaniques (*Essai sur les phénomènes électro-physiologiques des animaux*, pag. 81, 82), et il est dit dans le même Essai (pag. 85), que des courants électriques semblables à ceux de la grenouille se montrent chez tous les animaux dans la même direction. Enfin, dans le Mémoire de Décembre 1842 des *Annales de Chimie et de Physique*, se trouve la description d'une expérience faite sur une patte de lapin récemment tué, et qui est identique à l'expérience fondamentale de Galvani.

De tout cela vous n'avez pas dit un mot dans le Chapitre qui paraît destiné à donner l'histoire de mes recherches sur l'électricité animale.

Rapportez-vous à l'état de l'électro-physiologie à cette époque, à laquelle il n'était plus même permis, excepté parmi les médecins et les magnétiseurs, de parler de l'existence de l'électricité dans les animaux, et puis demandez à Faraday, à Sir David Brewster, à Wheatstone, à Grove, à Bowmann, à Todd, et à tant d'autres qui ont vu mes expériences en Angleterre, s'ils n'en ont pas tiré la conviction qu'on ne pouvait plus conserver le moindre doute sur l'existence du courant musculaire et de ses lois, et sur l'influence de la contraction musculaire sur un nerf excitable. Les vérités qui se déduisaient alors de mes expériences restent toujours les mêmes.

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.

Il suffit de lire mes Mémoires d'électro-physiologie de cette époque 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 avait cru d'abord propre de la grenouille seule.

A la page 25 de l'*Abstract* on trouve ce passage: « Matteucci » alors conclut étrangement, que puisque le courant de la grenouille » traversait une assez longue colonne d'eau salée, il était en consé» quence capable d'une action chimique, et il essaya de le prouver » avec la solution d'iodure de potassium sans employer des électrodes » métalliques. » On a toujours admis, et on admet toujours que la conductibilité d'un liquide et sa décomposition électro-chimique sont des circonstances inséparables, et quelle que puisse avoir été la cause de la coloration jaunâtre du nerf dans l'expérience citée, il serait impossible d'admettre *a priori* que les filets nerveux ne peuvent en aucune manière jouer le rôle d'électrodes, lorsqu'on sait que même des liquides peuvent agir ainsi.

A la page 26 je trouve, en parlant de moi, sans autres observations et avec les deux derniers mots soulignés, le passage suivant: « Il affirme aussi que le courant est absent dans les grenouilles lors» qu'elles sont sous l'influence du tétanus, aussi bien que lorsqu'elles » sont refroidies par la glace, pendant *quelques minutes*. » Je persiste entièrement dans ces propositions.

J'ignore à quel propos dans cette même page on parle de mon silence sur une hypothèse *du courant propre des nerfs et du sang*, qu'on m'attribue et qu'on regarde comme mon point de départ. J'ai recherché dans mon Mémoire sur la torpille, qui est imprimé dans les *Annales* (année 1837, pag. 430), ce que j'avais dit alors sur le courant propre, et je n'ai trouvé qu'un fait très connu à propos d'une goutte de sang frais répandu entre le muscle et le nerf, et qui rend plus vives, comme le ferait aussi l'eau salée, les contractions dues au courant propre dans les expériences de Galvani. Dans la suite de la même page du Mémoire que je viens de citer, je donne les résultats, encore imparfaits de mes premières expériences sur le courant musculaire.

Dans la même page 26 de votre livre, on dit que dans mes premières expériences sur le courant propre j'ai employé la méthode

qui, suivant vous, devait conduire à une connaissance plus profonde des lois de l'électricité animale. Evidemment cela a été dit pour ajouter après, que j'ai abandonné ensuite la bonne méthode pour une mauvaise. Or 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, la seule qui ait démontré le courant musculaire, par la raison toute simple que c'est elle seule qui augmente la force de ce courant sans augmenter les courants dus aux causes étrangères à l'expérience. La seule raison, qui est bien loin d'être scientifique, par laquelle on a 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. 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.

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 les piles musculaires.

D'ailleurs je ne puis croire que la différence entre les deux méthodes soit celle de fermer le circuit avec les lames de platine du galvanomètre, au lieu de le faire avec un morceau de muscle. En employant, comme je l'ai toujours fait, de l'eau distillée ou de l'eau de source au lieu d'une solution saline, on n'a pas à craindre des courants entre les lames de platine et le liquide; et d'ailleurs les courants musculaires peuvent être rendus aussi forts que l'on veut.

A la page 27 il est dit que mon Mémoire de 1838, reproduit sans altération dans mon *Essai sur les phénomènes électriques des animaux*, imprimé à Paris en 1840, forme le *starting point* pour les recherches de M. Du Bois-Reymond. Je demande la permission de répéter encore, que mes travaux sur le courant musculaire et ses lois, sur le courant propre et sur la contraction induite, ont été communiqués à l'Académie de Paris dans la Séance du 6 Septembre 1841 et dans celles du 21 Février et du 24 Octobre 1842, et publiés dans les *Annales de Chimie et de Physique* de la même année.

Vers cette même époque j'ai montré mes expériences à un grand nombre des membres de l'Académie, tels que MM. Humboldt, Becquerel, Dumas, Magendie, Despretz etc. Or, tout cela a été oublié

dans l'histoire que vous faites de mes travaux d'électro-physiologie qui précèdent ceux de M. Du Bois-Reymond, puisqu'ils ont été publiés dans le cahier de Janvier 1843 des *Annales* de Poggendorff.

Au Chapitre VI vous parlez de la grenouille galvanoscopique, comme si son application convenable aux recherches d'électro-physiologie ne m'appartenait pas et n'avait pas d'importance: en lisant mes Mémoires on sera obligé d'admettre le contraire.

Je suis cité au Chapitre VII, dans lequel on donne ce qu'on appelle la loi générale de l'excitation des nerfs par le courant électrique, et qui n'est, en vérité, que la conclusion des expériences de Ritter, de Marianini, de Nobili, de moi et d'autres, exposée avec des dénominations et des modes de représentation empruntées aux mathématiques. L'objet de la citation est de démontrer que j'ai commis une grande erreur dans une Note que j'ai communiquée à l'Académie sur la mesure de la force nerveuse développée par le courant électrique. Dans cette Note j'ai eu bien soin de déclarer combien le sujet était difficile, et que les nombres que j'avais trouvés devaient être vérifiés par des recherches ultérieures: en un mot, je n'ai voulu avec ces premières expériences que rendre évident que l'effort musculaire ou la quantité de travail développée par une certaine quantité d'action chimique qui agissait sous la forme de courant électrique, était beaucoup plus grand de celui que cette même action chimique pouvait produire, ou sous forme de la chaleur correspondante, ou dans les machines électro-magnétiques.

Quand même la loi de l'excitation nerveuse serait celle qu'on admet dans ce Chapitre, ce qui n'est fondé que sur une analogie avancée par Marianini et que ce savant physicien s'est bien gardé de présenter autrement que comme une simple hypothèse, cela ne pourrait jamais rendre absolument erroné le principe sur lequel mon expérience est fondée. Pour me rapprocher autant que possible de la vérité et du but de mon expérience, j'ai justement employé un appareil très ingénieux, construit par M. Bréguet, dans lequel le passage du courant qui a excité la contraction ne se prolongeait que pendant la durée de cette contraction. (*Comptes-Rendus*, tome XVIII, pag. 563.)

Je viens de dire dans quel but théorique cette recherche était tentée, et l'on conçoit facilement que les erreurs que l'expérience y introduit sont dans le même sens, et ne peuvent par conséquent détruire la conclusion à laquelle je voulais arriver.

Il fallait donc, pour toutes les raisons, être moins tranchant à juger la portée de ces recherches, dont j'avais eu bien soin moi-même de déclarer l'imperfection et le vrai but.

Il est dit, page 76 de l'*Abstract*, que j'ai « toujours préservé,

» dans la formation de mes piles musculaires pour étudier le courant » propre, les troncs nerveux et un morceau de la colonne épinière, » ce qui est non seulement inutile mais véritablement nuisible, parce » qu'il *augmente la résistance à la conductibilité.* »

Je n'ai besoin pour détruire une assertion si fausse, et que je suis presque tenté d'appeler un MENSONGE, que de vous renvoyer à mon Mémoire des *Annales de Chimie et de Physique*, 1842, pag. 317 et suivantes, et de vous prouver ainsi que j'ai bien établi, en opérant sur le seul muscle de la jambe de la grenouille, quel était le vrai élément électro-moteur de ce courant, et quel était le rôle du nerf dans toutes ces expériences.

Ce qu'il y a de plus étrange dans ce qui est dit dans ce passage, c'est qu'on imagine que je n'ai pu employer un liquide bon conducteur pour y plonger les extrémités de mes piles musculaires, parce que je voulais toujours laisser les filets nerveux en expérience, et que ces filets en contact avec certains liquides conducteurs, dites-vous, auraient excité des contractions dans les muscles.

Or 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; que j'ai imaginé de recourir à des piles musculaires justement pour supprimer les liquides conducteurs qui n'agissent pas dans ces expériences sur les filets nerveux, mais bien sur les lames du galvanomètre pour en troubler les résultats.

A la fin de la même page il est dit, que M. Du Bois-Reymond « trouvant incroyable l'opinion de Matteucci, que la grenouille seule » est douée du pouvoir de causer ces courants, s'est mis à faire des » recherches sur d'autres animaux. »

Pour juger de l'importance de cette considération, qui peut se répéter à chaque moment dans les sciences expérimentales, il faut dire qu'en 1838, surtout en se rappelant les phénomènes des poissons électriques, on pouvait bien suivre l'opinion de Galvani, de Humboldt et de Nobili, et que c'est justement dans le but d'étendre l'étude des phénomènes électriques des muscles, que je suis parvenu dans la suite, c'est-à-dire en 1841 et 1842, à découvrir l'existence du courant musculaire et de la contraction induite.

Je suis bien aise de répéter encore une fois, qu'à cette époque je n'avais pas vu encore, sur les muscles tendineux des autres animaux, ce que j'appelais toujours le courant propre de la grenouille, et que c'est dans le Mémoire de M. Du Bois-Reymond, publié en 1843, que ce fait se trouve publié pour la première fois.

Dans le Chapitre XII je suis cité plusieurs fois. Il est vrai que j'ai dit en 1842, ce que je persiste à dire, que le courant musculaire est dirigé de l'intérieur du muscle à la surface dans le muscle

même. J'ai également dit, et cela reste toujours, qu'en laissant, dans mes piles, aux muscles en expérience leur filet nerveux, le courant se montre dirigé du nerf au muscle. J'ai eu pourtant soin de décrire dans mon Mémoire de 1842, les expériences qui prouvent que les filets nerveux se comportent comme des simples conducteurs, semblables à un fil de chanvre ou à un morceau de papier imbibé d'eau.

Il m'était impossible d'ignorer que la surface tendineuse d'un muscle se conduit comme l'intérieur du muscle, car c'est en cela que consiste le cas du courant propre de la grenouille.

Comment donc peut-on croire sérieusement, que parce que je n'ai pas appelé section longitudinale du muscle et section transversale artificielle ou naturelle, ce que tout simplement j'ai appelé et je continue d'appeler surface du muscle, intérieure et tendon, que je n'aie pas effectivement donné la loi du courant musculaire?

Il faut dire au contraire, que je n'ai pas voulu admettre, sinon comme analogie, que la surface tendineuse était identique à la section transversale naturelle du muscle. J'ajoute maintenant, qu'après avoir écouté la lecture des deux volumes de M. Du Bois-Reymond, et avoir suivi avec attention tous les efforts qu'il fait pour considérer les courants musculaires comme des courants dérivés, développés par les faisceaux musculaires qu'il se représente comme des cylindres de cuivre couverts de zinc et plongés dans un liquide, je n'ai pu y trouver qu'une simple et pure hypothèse, qui n'est appuyée effectivement par autre chose que par les faits mêmes pour lesquels elle a été inventée.

Je n'ai pas besoin de répondre aux deux autres passages dans lesquels je suis cité, page 97, car 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.

J'ignore maintenant dans quel esprit vous dites que les expériences de M. Du Bois-Reymond étaient indépendantes des miennes; la vérité est, que mes expériences furent publiées en 1842 et les siennes en 1843, et que j'ai adopté pour l'interprétation des phénomènes d'électricité musculaire une hypothèse différente de la sienne.

A la page 98 vous avez décrit une de mes expériences tentée en employant pour liquide en contact avec les extrémités de mes piles musculaires, tantôt de l'eau salée, tantôt de l'eau pure, par lesquelles on obtient, comme cela doit être, une différence dans l'intensité du courant. Cette description prise isolément est certainement très peu instructive pour vos lecteurs, et évidemment elle n'a été donnée que pour pouvoir ajouter ensuite, que la méthode de M. Du Bois-Reymond était supérieure à la mienne, puisqu'il obtenait avec son galvanomètre *sur un morceau microscopique* de muscle une déviation plus grande que celle que j'obtiens avec mes piles musculaires.

D'abord, et pour être vrai, au lieu d'un morceau microscopique il faut désormais entendre qu'on parle d'un morceau de muscle qui est employé à fermer le circuit du galvanomètre.

Il n'y a rien qui doive surprendre, si avec des galvanomètres différents on obtient du même élément électro-moteur des indications différentes. Ce n'est pas par la déviation plus ou moins grande de l'aiguille du galvanomètre qu'on peut juger de la valeur des méthodes employées pour étudier l'électricité musculaire. Toutes les expériences sur le courant musculaire que j'ai publiées dans mes Mémoires de 1838 à 1842, et auxquelles on n'a rien changé depuis, peuvent se faire avec un seul élément musculaire et un galvanomètre très sensible, aussi bien qu'avec des piles musculaires et un galvanomètre quelconque. Mais, je l'ai déjà dit, et la chose se conçoit très facilement, avec la méthode des piles on est moins exposé aux erreurs de l'expérience, et la démonstration peut être rendue aussi évidente qu'on le désire.

On parle aussi à la même page 98 de la *confusion* que j'ai fait régner longtemps dans la science en admettant une différence entre le courant propre de la grenouille et le courant musculaire de tous les animaux. Il est à peine nécessaire de faire remarquer que cette confusion n'était autre chose que l'état vrai de la science à l'époque où l'on savait que le courant musculaire entre l'intérieur du muscle et la surface se trouvait dans tous les animaux, et qu'un courant dans un muscle entier entre le tendon et la surface n'avait été aperçu jusqu'alors que dans la grenouille.

D'ailleurs, si on entend par confusion la considération distincte de ces deux courants, distinction que j'ai conservée, même après avoir trouvé dans tous les animaux un phénomène semblable à celui de la grenouille, c'est-à-dire, ce que plus justement j'appellerai une prudente réserve à accepter une simple analogie pour une identité parfaite, j'avouerai que cette confusion règne toujours dans mon esprit.

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 différens muscles entiers de la grenouille. Je me rappelle avoir lu quelque part dans son Mémoire, qu'il regardait la constance de la direction du courant propre de la grenouille comme *un hasard trompeur*.

Lorsque je pense que la disposition qui donne plus facilement et plus distinctement le courant musculaire consiste à appliquer directement les extrémités du galvanomètre sur la section transversale artificielle du muscle et sur le tendon, qui est considéré par M. Du Bois-Reymond comme la section transversale naturelle; que cela a lieu avec la même intensité sur une demi-cuisse ou sur un demi-gas-

trocnemien de grenouille; il m'est impossible d'admettre sans réserve l'identité du rôle du tendon avec celui de l'intérieur du muscle. Qui sait quand les résultats des expériences sur l'électricité musculaire pourront être exprimés par des nombres! Mais certainement ce n'est que quand cela aura lieu que la théorie de ce grand phénomène de la vie animale pourra être fondée.

Je ne veux pas répéter ici un reproche que je me suis permis de faire une autre fois aux recherches de M. Du Bois-Reymond: je veux seulement faire remarquer, que n'ayant jamais donné de résultats numériques dans toutes ses expériences, il aurait dû exprimer avec moins d'assurance ses hypothèses.

Dans mes Mémoires publiés dans le *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 le phénomène appelé jusqu'alors du courant propre, existait dans tous les animaux, et que l'extrémité tendineuse fonctionnait comme l'intérieure du muscle. Il est vrai qu'à cette époque je connaissais le Mémoire que M. Du Bois-Reymond avait publié en 1843, et j'avais déjà dit en 1845 (*Annales de Chimie et de Physique*; tome XV, pag. 67) que M. Du Bois-Reymond avait publié le premier qu'on obtenait sur les muscles entiers de différens animaux le courant qu'on avait regardé comme propre de la grenouille. Mais c'est justement par la lecture de ce Mémoire, dans lequel on ne décrit ni la méthode, ni le galvanomètre employé, ni aucun résultat numérique, et dont plusieurs des expériences rapportées sont certainement imparfaites, et dans lequel 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, et qu'il fallait *renoncer complétement à une exposition exacte de la manière dont il se fait que le courant de la grenouille entière et de la grenouille préparée à la manière de Galvani a une direction constante des pieds à la tête dans l'animal;* et, plus que toute autre chose, par l'impossibilité où l'on était de comprendre, alors plus que maintenant, comment on pouvait déterminer avec exactitude la direction des courants électriques dans une masse musculaire très petite; c'est, dis-je, justement 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.

Au Chapitre XVI il est dit que je n'ai pas prouvé l'existence du courant musculaire dans les animaux vivans: 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, et cela est dit dans mes Mémoires cités.

Au Chapitre XVII on commence par affirmer que d'abord j'ai découvert l'influence de la contraction sur le courant musculaire, mais qu'après, par des expériences faites sur des piles de grenouilles, je n'ai pu démontrer qu'il y eût développement du courant électrique pendant la contraction; et qu'enfin je n'ai pas su profiter de ma découverte de la contraction induite malgré les conseils de M. Becquerel.

L'explication de la contraction d'une grenouille par le passage d'un courant électrique dans ses nerfs, quoique facile à être donnée, n'exigeait pas moins d'être démontrée vraie par l'expérience. Vous auriez été plus juste et plus exact en disant, que n'ayant pas un galvanomètre aussi sensible que celui de 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.

A propos de l'explication de la contraction induite, on me fait le grand tort d'avoir longuement expérimenté sur ce phénomène, et d'avoir modifié à mesure mes hypothèses. Certainement il aurait été préférable de ne jamais faire des hypothèses, et c'est en suivant ce principe que vous auriez dû admettre avec plus de réserve, *que la vraie origine du courant induit consiste dans la variation de densité du courant musculaire qui parcourt le nerf de la grenouille galvanoscopique.* Vous ne pouvez pas ignorer les expériences qui sont contenues dans mon dernier Mémoire (*Philosophical Transactions*, Seconde Partie, 1830), expériences qui d'ailleurs sont très faciles à répéter et d'un succès certain, et qui conduisent à une toute autre conclusion. A ce propos il est curieux de remarquer qu'à la page 213, N° 13, de l'*Abstract*, il est dit que M. Du Bois-Reymond n'a pas réussi à prouver si pendant la contraction il y a *diminution* ou *cessation* ou *inversion* du courant musculaire. C'est que la vraie nature du phénomène ne pouvait être mise en évidence avec l'aiguille du galvanomètre dont le mouvement est trop lent en comparaison de la presque instantanéité de la contraction induite, de sorte qu'il fallait recourir, comme je l'ai fait le premier, et comme il est dit dans le Mémoire que je viens de citer, à la grenouille galvanoscopique convenablement appliquée.

Au Chapitre XVIII sur la contraction secondaire (pour lui donner une dénomination différente de celle que j'avais adoptée) il est dit, page 140, que la forme la plus simple et la plus philosophique de mon expérience fondamentale de la contraction induite, est celle de comprendre le galvanomètre dans le circuit de la grenouille induisante, et d'avoir une pile et une roue d'interruption pour y exciter les contractions. (Voyez *fig.* 36, pag. 141.) J'espère que tout le monde comprendra facilement que la forme la plus simple de l'expérience

de la contraction induite est celle qui laisse le phénomène exempt de toute cause d'erreur, et qui consiste à poser le nerf de la grenouille galvanoscopique sur le muscle d'une autre grenouille, et à employer une irritation mécanique pour exciter les contractions.

A la page 143 du même Chapitre il est dit, que pour que mon expérience de la contraction induite réussisse, il faut que le nerf de la grenouille galvanoscopique établisse le circuit entre le tendon et la surface du muscle qui doit se contracter. Or, j'ai déclaré bien des fois, et l'expérience peut facilement se répéter, qu'il serait impossible de déterminer quelle est la position plus favorable du nerf de la grenouille galvanoscopique pour la production du courant induit. De même il est facile de s'assurer que la contraction induite s'obtient sans que le nerf de la grenouille galvanoscopique soit d'avance parcouru par une partie du courant musculaire qui serait ainsi dérivée. Ce n'est donc pas par des *variations de densité* de cette partie du courant musculaire que la contraction induite peut être expliquée.

Je persiste à croire, parce que des nouvelles expériences l'ont confirmé, que le phénomène de la contraction induite ne cesse pas d'avoir lieu par l'interposition entre le nerf et la surface du muscle d'une couche très mince d'un liquide isolant capable, non seulement d'arrêter la portion dérivée du courant musculaire qu'on suppose circuler dans le nerf de la grenouille galvanoscopique, mais aussi ce courant tout entier.

A la page 146 je suis cité pour dire que je n'ai obtenu qu'une contraction induite du quatrième ordre, et que M. Du Bois-Reymond, *en conséquence de sa connaissance de la meilleure position du nerf sur le muscle*, l'a obtenue du cinquième ordre: j'avoue que je ne puis comprendre l'intérêt de cette *découverte*.

J'arrive au Chapitre XXI, dont le titre est: *De la durée du décroissement du courant musculaire après la mort.* Quiconque a répété les expériences principales sur le courant musculaire a dû trouver que ce courant décroît en général plus rapidement dans les premiers moments après la mort, et que dans les muscles de différents animaux cette diminution suit la même loi de l'excitabilité musculaire, comme je l'ai fait remarquer dans mes Mémoires. Cette analogie, qui était certainement importante et facile à démontrer, vous dites, page 158, qu'on devait se contenter de l'admettre, mais qu'il y avait des difficultés pour la prouver avec des expériences.

J'ai toujours cru, à la suite de mes recherches, que le courant musculaire est un phénomène lié à l'excitabilité du muscle: ces recherches ont été dirigées à étudier l'influence du système nerveux des différens poissons, des milieux gazeux, de la température, de la nutrition, etc., et ont donné ce que j'ai appelé les lois du courant musculaire.

Tout cela n'entre pas dans l'exposition que vous faites des circonstances qui influent sur le courant musculaire: seulement vous y glissez (page 159) la remarque, que puisque l'irritabilité musculaire persiste plus longuement dans les oiseaux que dans les mammifères, j'ai eu tort de trouver avec l'expérience, que le résultat, quant à la durée du courant musculaire après la mort, était dans l'ordre inverse. Permettez-moi de persister dans mon opinion, jusqu'à ce qu'elle soit prouvée fausse par des expériences plus étendues, plus variées et plus exactes que les miennes.

Au Chapitre XXIII sur le courant nerveux je suis cité pour dire, qu'après beaucoup de résultats contradictoires, j'ai enfin en 1844 nié l'existence du courant électrique des nerfs et dans les nerfs. La vérité est, et tout le monde peut s'en assurer facilement, que depuis mes expériences sur la torpille (1836) *jusqu'à ce moment*, j'ai toujours déclaré très explicitement que je n'avais pas réussi à trouver un courant électrique dans les nerfs intacts des animaux vivans. J'avoue qu'après avoir entendu la lecture de deux volumes de M. Du Bois-Reymond, et quand même on voudrait admettre, comme le fait cet Auteur, un courant nerveux, dont les lois seraient les mêmes que celles du courant musculaire, il me serait impossible de concevoir qu'avec le galvanomètre le plus sensible du monde on puisse obtenir un courant dérivé en appliquant les deux extrémités du galvanomètre sur deux points rapprochés de la surface d'un filet nerveux intact.

Avec un galvanomètre très délicat on parvient toujours, soit sur des morceaux de foie, de poumon, de reins, de nerf, de cerveau, etc., à avoir des signes du courant électrique, mais excessivement faibles, incertains et souvent dans des sens contraires. Ce n'est pas là le cas du courant musculaire.

A l'aide de la grenouille galvanoscopique, qui est certainement, dans des circonstances convenables, l'instrument le plus sensible pour découvrir un courant électrique d'une très courte durée, je n'ai jamais pu trouver aucun courant dans les nerfs ni pendant qu'ils étaient excités ni avant.

Fidèle au principe que j'ai déjà déclaré de vouloir suivre en vous adressant ces observations sur votre Extrait des recherches de M. Du Bois-Reymond sur l'électricité animale, je dois m'arrêter, après avoir examiné *tous* les passages du livre où se trouve mon nom, n'ayant eu d'autre but que de défendre mes droits et de réfuter vos attaques, sans entamer une discussion sérieuse sur les expériences et les hypothèses de M. Du Bois-Reymond, ce que j'espère pouvoir faire bientôt dans ma nouvelle édition du *Traité des phénomènes électro-physiologiques.*

Je déplore amèrement la perte de temps que la rédaction de

cette lettre m'a causée, car je ne connais rien de plus pénible que d'être obligé, sur des sujets scientifiques, de défendre soi-même des droits acquis, ou de réfuter des attaques injustes. En se bornant, dans l'exposition des recherches de M. Du Bois-Reymond, à décrire ce que vraiment ce Savant a fait sur l'électricité animale, et à donner ainsi un résumé de ce qui est contenu dans ses deux gros volumes, sans rallumer une polémique qui malheureusement n'a que trop retenti dans le monde scientifique sans aucun avantage pour la science, vous auriez fait une bonne œuvre dans l'intérêt de vos confrères et dans celui de M. Du Bois-Reymond, et vous auriez épargné à vous et à moi le désagrément de cette lettre.

Pour tout juge impartial il sera clair comme le jour, que je n'ai rien dit dans tous mes Mémoires d'électro-physiologie pour m'attirer les accusations grossières et calomnieuses que M. du Bois-Raymond lance contre moi dans ses deux volumes. Si le *génie irritable* de ce Savant pouvait lui permettre une fois de relire avec calme son Mémoire de Janvier 1843, il se convaincrait, que si dans mes travaux successifs je n'ai pas justement apprécié l'importance des faits nouveaux et des déductions tirées qui se trouvent dans son unique Mémoire de 1843, la faute en était à lui, qui au lieu de décrire minutieusement, comme le sujet et l'état de la science à cette époque l'exigeaient, les appareils, la méthode et les expériences qu'il avait faites, s'était étendu plutôt sur des analogies plus ou moins vagues et à présenter des hypothèses comme des théories.

Lorsque, longtemps après la publication de ce Mémoire, j'appris par l'édition française du *Manuel de Physiologie* de M. Muller, 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, et qu'enfin il avait 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 desquels il avait été conduit à admettre que les extrémités tendineuses des muscles jouent le rôle dans les électro-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.

A cette époque, c'est-à-dire depuis l'année 1843 jusqu'en 1846 et même plus tard, j'ai répété à Paris, à Londres, à l'Association Bri-

tannique de York, mes expériences sur le courant museulaire. Je citerai MM. de Humboldt, Mitscherlich, Liebig, comme les Savants Allemands qui ont vu mes expériences. J'ai le souvenir le plus certain qu'aucun de ces Savans ne m'a parlé du Mémoire de M. Du Bois-Reymond, ni fait aucune remarque sur mes expériences qui pût me faire soupçonner le moins du monde que j'avais des torts envers ce jeune Physiologiste. Ma conscience me dit que je n'en ai pas, ce qui me fait supporter avec résignation la peine que pour cette fois, certainement la dernière, je me suis donnée de défendre des droits qui sont incontestables.

Agréez etc.

Pise, 25 Janvier 1853.

CH. MATTEUCCI.

OBSERVATION

D'UN

CAS DE PÉRITONITE PURULENTE

avec

ABCÈS ENKYSTÉ DANS LE PETIT BASSIN,

suivie de réflexions;

PAR LE DOCTEUR **E. DÉGRANGES,**

médecin honoraire de l'hôpital Saint-André.

BORDEAUX,

HENRY FAYE, IMPRIMEUR DU JOURNAL DE MÉDECINE,

rue Sainte-Catherine, 139.

—

1848.

OBSERVATION

D'UN

CAS DE PÉRITONITE PURULENTE

AVEC

ABCÈS ENKYSTÉ DANS LE PETIT BASSIN,

suivie de réflexions.

Agée de vingt-neuf ans environ, d'un tempérament lymphatique, nerveux, maigre et grêle de forme, enceinte pour la première fois, la femme Lacaribe accoucha, à la Maternité, d'un enfant à terme, sans que l'accouchement fût précédé ni immédiatement suivi d'aucun symptôme grave. Quatre heures de douleurs suffirent pour expulser le fœtus. Pendant les douze premiers jours qui suivirent la délivrance, Lacaribe n'offrit d'autres symptômes que ceux que l'on observe en pareille circonstance : apparition des lochies; fièvre de lait; gonflement des mamelles; abondance de lait qui permet l'allaitement de l'enfant pendant douze jours. La femme Lacaribe était sortie de l'hospice et avait repris son domicile ordinaire.

Ces douze jours passés, tout prit un caractère sérieux : tuméfaction et douleur dans toute la cavité abdominale, surtout dans la région hypogastrique où la moindre pression détermine une vive sensation; diarrhée; fièvre; les lochies ne coulent plus; le lait n'est pas aussi abondant, etc., etc.; tout, en un mot, confirme une inflammation péritonéale, inflammation qui fut combattue par des cataplasmes émollients sur les points douloureux, et des liniments opiacés, qui apaisèrent pendant quelques jours les symptômes graves que la malade venait d'offrir.

Si Lacaribe voyait avec plaisir les douleurs abdominales se dissiper, elle voyait d'un œil inquiet son ventre grossir de jour en jour d'une manière effrayante; aussi songea-t-elle à venir à l'hôpital Saint-André de Bordeaux, et s'y rendit (le 6 mars 1843.)

Couchée au n° 4 de la salle 8, voici quels étaient les symptômes qu'elle présentait à ma première visite : peau naturelle; pouls petit et fréquent; langue un peu muqueuse sur ses bords et disposée à être sèche à son centre; abdomen indolore et d'un développement double de celui d'une grossesse à terme. Le choc qu'une main détermine lorsqu'elle frappe un côté des parois abdominales, en même temps que l'autre main est placée diamétralement du côté opposé, ne laisse aucun doute sur l'existence d'un liquide renfermé dans la séreuse de l'abdomen. Quelques nausées, pas de vomissements; un peu de diarrhée; excrétions d urine rares.

En face d'un développement si considérable de l'abdomen, on ne pouvait retarder plus longtemps à pratiquer la paracenthèse; opération qui ne détermina pas

la plus légère douleur à la malade et qui donna issue à 3 kilog. de liquide. C'est ici le lieu de faire remarquer que ce liquide, au premier jet de sa sortie, formé par une sérosité roussâtre, devint opaque dans les jets suivants, et enfin comme purulent vers la fin de l'opération. Le fluide obtenu par la ponction, mis à reposer et décanté ensuite, présentait au fond du vase 500 gr. environ d'un pus crémeux et roussâtre.

L'ouverture abdominale du trois-quarts ne laissa pas échapper toute la sérosité que contenait la cavité péritonéale, car le lendemain je trouvai le ventre volumineux et diminué seulement à peu près de moitié. Cependant la malade se trouva soulagée de beaucoup, dormit la nuit, et exprima son contentement.

Quelques jours après l'opération, et pendant que la malade était assujettie à un traitement diurétique (digitale, scille, et nitrate de potasse), je fis appliquer un large vésicatoire sur les régions ombilicale et hypogastrique, points légèrement douloureux; mais rien n'empêcha la quantité du liquide d'augmenter. Les parois abdominales devinrent aussi distendues qu'elles l'étaient primitivement, je dirai même davantage; car Lacaribe, par la compression des organes respiratoires que le liquide refoulait en haut du thorax, était gênée de la respiration et toussait; en même temps, ses membres inférieurs, les jambes spécialement, devenaient œdématiés.

D'après ces symptômes, je fis de nouveau pratiquer la paracenthèse, le 21 mars; l'opération donna issue à une matière en entier purulente (5 kilog.), bien liée, roussâtre et inodore; cette seconde ponction, comme

la première, amena du mieux chez la malade; la gêne de la respiration et la toux disparurent, le calme général se rétablit; mais malheureusement ce fut pour bien peu de jours, car, du 1er avril au 11 de ce même mois, tous les premiers phénomènes apparurent *avec une intensité plus grande*. Voici, du reste, les symptômes qu'elle présentait à cette époque : peau sèche, pouls petit et assez fréquent; l'œdème, qui primitivement occupait les jambes et les cuisses, a envahi les parois du ventre, surtout du côté droit; l'abdomen a de nouveau pris un développement extraordinaire et empêche la malade de respirer, au point qu'elle est forcée de rester assise sur son séant pour mieux accomplir cet acte fonctionnel. L'auscultation fournit l'existence bien marquée du souffle vésiculaire à la partie supérieure des deux poumons, et une égophonie très-prononcée au-dessous du sein droit, où il y a matité. Les battements du cœur sont réguliers, avec un bruit de clapottement; cependant, non-seulement l'oreille les entend dans tout le côté gauche du thorax, mais encore sous la clavicule droite (signe pour moi d'un épanchement pleurétique). L'état devient tellement grave que je songe à faire pratiquer la *thoracenthèse*.

Au traitement déjà employé, j'ajoutai l'application de deux bandelettes vésicantes à la base de la poitrine; pensant ainsi amener 1° vers la peau une révulsion favorable; 2° pratiquer un dégorgement de sérosité à l'aide des phlyctènes; vain espoir, les symptômes s'aggravèrent, l'épigastre devint bombé, la malade était prête à suffoquer; il fallut, pour la troisième fois, recourir à la ponction abdominale; elle fut pratiquée le

21 avril, et 2 kilogr. de pus *bien lié* sortit par la canule de l'instrument.

Aussitôt que l'opération fut faite, c'est-à-dire le jour suivant, Lacaribe ressentit un mieux général; puis arriva une diminution de l'œdème des jambes et des cuisses, mais il faut le noter, la toux resta la même; ce qui était dû, sans doute, au liquide que l'on avait diagnostiqué comme existant dans le thorax. Cependant sous l'empire d'un traitement convenable associé toujours aux diurétiques, la malade était revenue à un état satisfaisant, lorsque le 1er mai, après une fièvre de vingt-quatre heures de durée, un érysipèle apparut au nez et envahit la face dans l'espace de quatre jours, au milieu de phénomènes morbides qui firent craindre la mort immédiate : pouls comprimé, face considérablement tuméfiée et de couleur lie de vin, *langue sèche* et râpeuse, etc.; ces phénomènes se dissipèrent *encore* sous peu de jours, pour être remplacés par tous ceux d'une vaste collection de fluide dans l'abdomen, qui me forcèrent de pratiquer de nouveau la paracenthèse, le 22 mai : 3 kilogr. de pus très-épais s'écoulèrent par l'ouverture.

Les jours qui suivirent cette opération furent meilleurs; l'œdème des extrémités disparut presque entièrement, et l'état général s'était considérablement amélioré par l'usage de la décoction de quinquina, prise régulièrement tous les matins. Mais voilà que tout à coup Lacaribe ressentit de violentes coliques qui l'empêchaient de se tenir debout : lorsqu'elle marchait, elle avait la précaution de porter en avant la tête et le haut du tronc; un phénomène digne de remarque, c'est qu'au milieu de ces coliques, et à trois reprises différentes,

Lacaribe eut des selles très-copieuses, presque *en entier formées par du pus crémeux et roussâtre*, au milieu duquel nageaient quelques rudiments de matière fécale, durs et noirâtres. Ces selles, en général, soulageaient la malade. Enfin, Lacaribe sortit de l'hôpital, le 20 juillet 1843, après avoir eu un abcès froid au-dessus de la clavicule; elle pouvait marcher avec assez de facilité, mais néanmoins son corps ne pouvait se redresser en arrière, car alors elle éprouvait un tiraillement douloureux dans l'abdomen.

Le 26 novembre suivant (quatre mois après), Lacaribe se représenta à l'hôpital, ayant, depuis un mois environ, une petite fièvre intermittente qui n'était pas bien caractérisée, mais résistait à l'emploi du sulfate de quinine; une toux existait plus forte dans certains moments, revenant par quinte, mais n'étant accompagnée ni de sueurs nocturnes, ni de crachats sanglants; seulement on constata une petite expectoration bronchique mélangée avec de la salive.

Poitrine indolente depuis sa sortie de l'hôpital, en juillet; les extrémités des doigts ont subi une modification, consistant dans le boursoufflement de l'incurvation du rebord de la matrice de l'ongle. Dans tous les doigts le tissu même de l'ongle est altéré.

Percussion plus sonore à droite qu'à gauche; souffle vésiculaire beau partout; vers la cinquième côte à gauche, bruit égophonique, ou bien résonnance de la voix naturelle qui est très-*sifflante*.

Près de deux mois s'écoulèrent!....

Le 14 janvier 1844, nouvelle auscultation.

Souffle sain, mais un peu dur sous la clavicule à

droite, moins fort au niveau du mamelon, sensible au dehors de celui-ci, insensible au-dessous. *Matité* à la base; battements du cœur, retentissant au sommet de la poitrine. A gauche, mêmes résultats.

Bruit de râpe au premier temps des contractions du cœur : la matité précordiale s'étend jusque sous le sternum.

L'état général de cette malade prend de jour en jour des caractères plus fâcheux.

16 Janvier. Anasarque avec couleur luisante de la peau, sur les membres abdominaux en entier et sur une partie des membres thoraciques; le ventre devient très-ballonné; pouls très-accéléré; excitation morale; craintes; plus tard anxiétés.

22 Janvier. Évacuations alvines diarrhétiques extrêmement fétides. Les personnes qui levaient Lacaribe pour la mettre sur le bassin, ont remarqué (et m'ont dit) que la matière des évacuations était blanchâtre, purulente, et semblable en tout à celle des évacuations alvines qui eurent lieu lors de son premier séjour à l'hôpital.

L'anasarque, ayant diminué dans les membres supérieurs, augmenta considérablement dans les membres inférieurs; bientôt une tympanite très-prononcée se manifesta; cours d'urine ordinaire; amaigrissement général, il arriva au marasme; la peau devint blafarde, écailleuse dans plusieurs régions, se plissa atoniquement; face hippocratique, couleur terne; la mort arriva le 28 janvier, après trente-deux jours d'alitement à l'hôpital.

L'autopsie cadavérique fut pratiquée vingt heures après la mort.

NÉCROPSIE DE LACARIBE.

Habitude extérieure. — Les muscles sont dans un état de relâchement; amaigrissement; œdème des membres inférieurs et du côté droit du tronc. Dans ce dernier point, la peau est séparée des muscles par une couche de tissu cellulaire œdématié de 20 millimètres d'épaisseur; le ventre est ballonné, proéminant vers l'ombilic, et comme conique.

Thorax. — Du côté droit, épanchement de sérosité limpide (environ 300,00); quelques adhérences du poumon; du côté gauche, adhérences aussi, mais pas d'épanchement; le *cœur* est petit, *peu consistant, pâle, et ne contient qu'un peu de sang décoloré et séreux;* les *ganglions lymphatiques*, thoraciques, *sont volumineux, durs*, d'un blanc jaunâtre à l'intérieur, semblables aux *fibro-cartilages intervertébraux;* des ganglions de même nature accompagnent les bronches jusque dans leurs dernières ramifications; à l'origine des bronches, il y en a quelques-unes *contenant une matière d'un gris sale*, comme caséeuse, semblable à la céruse employée par les peintres, assez analogue aux tubercules qui commencent à se ramollir.

Abdomen. — L'ouverture de l'abdomen donna issue à 5 ou 600 grammes de sérosité jaune citrine, sans filaments gélatineux, située entre le paquet formé par l'intestin grêle et la paroi postérieure de l'abdomen. Toutes ces anses intestinales sont adhérentes soit avec les parois, soit entre elles, soit avec les autres viscères (foie, rate, estomac); les unes dans une grande

étendue de leur surface, les autres par des points plus ou moins nombreux : tantôt c'est une fausse membrane interposée à deux surfaces adhérentes; tantôt ce sont comme des ponts plus ou moins longs, plus ou moins larges, jetés d'un point à l'autre. Il est à remarquer que toutes ces adhérences, de même que les fausses membranes qui les établissent, sont anciennes. Celles-ci sont d'un blanc mat, d'une assez bonne consistance, épaisses de 1 à 2 millimètres.

Le foie, adhérent à la paroi antérieure de l'abdomen par une grande partie de son lobe droit, est étendu, d'une consistance à peu près normale, un peu cassant, de couleur brique jaune; la vésicule, de même que la bile, est décolorée; la rate n'offre rien à signaler; *idem* pour les reins; le tube digestif, de l'œsophage au milieu de l'S du colon, est, soit à l'intérieur, soit à l'extérieur, d'une pâleur extrême; on n'y remarque pas la moindre trace de vascularisation. On pourrait hasarder pour ces organes l'expression de pâleur chlorotique.

La moitié inférieure de l'S du colon et le rectum, présentent à leur surface interne une couleur rouge, presque lie de vin, avec un peu de liquide épais, formant sur la muqueuse un enduit de même couleur. Un morceau de cette muqueuse, mis en macération pendant quarante-huit heures, a été complétement décoloré. La paroi supérieure du rectum, au niveau du vagin, vers le point où le péritoine abandonne celui-ci pour se replier sur le rectum, a subi deux perforations qui établissent une libre communication entre le rectum et une cavité accidentelle dont nous parlerons bientôt. Ces perforations, de forme à peu près circulaire,

présentent un diamètre d'environ 6 millimètres, et ne sont séparées que par une bride longitudinale de deux ou trois millimètres de largeur. Immédiatement au-dessous de ces deux orifices, on voit une perte de substance oblongue, de 15 millimètres de long sur 5 de large, dont les bords peu élevés sont taillés à pic; il est assez difficile de dire, d'une manière certaine, quelle est la partie anatomique qui en forme le fond : est-ce la musculeuse, est-ce la séreuse, ou bien est-ce le résultat d'une cicatrice?

La vessie est petite, ratatinée; ses parois sont un peu épaisses, pâles comme celles des intestins.

La substance de la matrice n'offre rien à noter; sa cavité contient une matière blanchâtre, molle, granulée. Sa surface externe sera décrite dans une autre circonstance.

Au-devant de la colonne vertébrale, et un peu à gauche, dans toute la région lombaire, existe un cordon très-épais repoussant l'aorte à droite; le *cordon* paraît être formé *par une chaîne de ganglions lymphatiques passés à un état comme fibro-cartilagineux*, réunis et enveloppés par du tissu cellulaire induré.

En dehors de la cavité abdominale, et seulement séparée par le péritoine et par une membrane propre, existe une autre cavité assez vaste, limitée en haut par une ligne qui s'étendrait de l'hypocondre droit à l'épine iliaque antérieure et supérieure; à droite par le flanc, à quelques centimètres en avant du bord externe du grand oblique; à gauche par la fosse iliaque, le trajet de l'S du colon; en bas par les os pubis, la face postérieure de la matrice, les obturateurs internes; en

avant par les muscles droits, l'aponévrose abdominale; en arrière par le péritoine.

La ponction de cette cavité, faite pendant l'ouverture de l'abdomen, a donné issue à environ 200 gr. d'une *sanie noirâtre*, assez analogue, pour la consistance et la couleur, au *liquide épais qu'on fait couler en pressant une rate ramollie*. Dans l'intérieur on a trouvé des *caillots de sang volumineux*, allongés, d'un rouge assez vermeil, dont on peut évaluer le poids à 150 gr.

La surface intérieure de cette cavité est inégale, comme plissée; on y voit, dans certains points, des élevures que l'on peut en quelque sorte comparer pour la forme aux colonnes charnues de la troisième espèce, que l'on trouve dans les ventricules du cœur. La couleur de cette surface intérieure est d'un brun noirâtre, beaucoup plus foncée vers le bas-fond du bassin, au voisinage de la matrice, lieu où étaient surtout situés les caillots de sang. La nature de cette membrane interne révèle une muqueuse accidentelle; elle est friable et ne présente pas, vue à l'œil nu, de vaisseaux lymphatiques sanguins dans son tissu. Après quarante-huit heures de macération, la surface interne de cette cavité était devenue d'un gris clair. La paroi postérieure était épaisse d'environ 2 millimètres; on y distinguait facilement trois parties : 1° de dedans en dehors une pellicule mince, peu consistante, se laissant facilement déchirer; 2° un tissu dense mais friable, qui paraissait être le résultat de l'induration d'une couche de tissu cellulaire; 3° le péritoine, que l'on pouvait aussi séparer avant la macération. On conçoit quelles modifications la paroi antérieure doit subir.

Si on étudie la partie inférieure de cette cavité, on voit qu'elle s'avance sous le pubis, qu'elle règne sur la face antérieure de la vessie, que la matrice proémine au milieu de la cavité, adhérente seulement par son sommet, au moyen d'une bride assez courte, à sa paroi postérieure. Le corps est dénudé, recouvert seulement par la pellicule qui tapisse toute la surface interne de la cavité. En arrière de la matrice et un peu à gauche, on aperçoit une espèce d'*infondibulum* assez large, qui s'étend jusqu'au rectum; c'est dans ce point qu'existent les perforations de cet intestin. A gauche, la paroi postérieure de la cavité est adhérente à la fin de l'S du colon; ainsi cette adhérence a lieu entre les séreuses.

Réflexions. — La description symptomatologique et nécropsique du fait que l'on vient de lire, conduit à se poser et à chercher les moyens de résoudre les questions suivantes, autour desquelles viendront naturellement se grouper quelques détails intéressants.

Y avait-il chez Lacaribe un kyste proprement dit, ou bien un abcès enkysté? Dans l'une ou l'autre hypothèse, cette cavité anormale était-elle située en dedans ou en dehors du paritoine?

Etait-ce un kyste proprement dit? Essayons de répondre en examinant sa situation, sa consistance et son développement.

Sa situation. — Elle pouvait faire croire à la présence d'un kyste ayant son origine sur l'un des ovaires, organe qui si souvent produit ces sortes de dégénérescence. Peut-être les deux ovaires ont-ils été affectés simultanément, et produit deux cavités qui se

sont confondues en une seule? On doit s'expliquer avec doute et réserve sur ce point, car aucun vestige des ovaires ni de leurs dépendances n'a pu être retrouvé d'une manière certaine, et ainsi n'a pu être précisé le point d'insertion de la cavité anormale qui nous occupe.

Sa consistance. — Elle repousse la croyance en un kyste : comme chez presque tous ces derniers, on ne rencontre pas une enveloppe extérieure de nature fibreuse, ou au moins semi-aponévrotique, tapissée à l'intérieur d'une sorte de séreuse; on ne rencontre pas les cloisons si nombreuses et si ordinaires dans les kystes ovariques; on ne rencontre pas encore ces débris tantôt adipeux, tantôt cartilagineux, quelquefois même pileux, ou même d'une autre nature *sui generis*, si fréquents dans ses poches pathologiques. Dans notre fait c'est au contraire une membrane épaisse, plissée, de nature muqueuse, qui tapissait l'intérieur de la cavité. Sa teinte était brunâtre et noirâtre, au lieu de présenter l'aspect albuginé des fibreuses. Au lieu des sécrétions plus ou moins transparentes et liquides, se trouvait un épanchement épais, noirâtre, et comme oléagineux. Son odeur était infecte, au lieu de n'impressionner en rien l'odorat.

Son développement. — Il offrait un contraste marqué avec celui des kystes ordinaires. Ceux-ci, nés dans un point, n'étant le plus souvent que l'exagération d'une des petites cavités sébacées, soit du tissu cellulaire, soit de tout autre tissu membraneux, s'avancent graduellement au milieu des tissus qui les environnent, repoussant ces tissus, repoussant les organes qu'ils

trouvent sur leur route, tantôt formant des adhérences sous formes de brides, et très-souvent n'étant que juxta-collées aux parties voisines. Ce qui les distingue, c'est l'isolement de leur cavité au milieu de tous les tissus circonvoisins, isolement qui fait qu'à l'aide d'une dissection légère, ou même d'une simple énucléation, on peut les extraire en totalité avec leur contenance, et ne constater à leur place qu'un vide où l'on signale avec facilité les rapports anatomiques qui s'étaient pathologiquement produits. Les kystes ovariques sont globuleux aussi, ou ovalaires et plus ou moins *mobiles*. On leur imprime un mouvement en agissant sur l'utérus. Enfin, dans certains cas, on peut reconnaître qu'ils ne produisent pas les symptômes de compression observés dans les abcès iliaques.

Donc, de tout ce qui précède on doit conclure que nous n'avons pas à faire maintenant à un kyste ordinaire et ovarique.

Etait-ce un abcès enkysté? Voyons : Cet abcès était-il de la nature de ceux appelés chauds ou froids? Ce n'est pas un abcès chaud, puisque la malade n'avait jamais présenté les symptômes qui accompagnent et caractérisent ces affections phlegmoneuses. Ainsi, point de douleur, de tension, de chaleur, de gonflement locaux; point de nuance fébrile avec pouls dur, fort et plein; point de congestion capillaire générale. Ç'aurait été plutôt un abcès froid, si on se rappelle les précédents morbides et les détails autopsiques ayant trait au développement des ganglions du ventre et de la poitrine. Au mois de juillet dernier un abcès de cette nature s'était formé au-dessus de la clavicule, région

cervicale latérale, et la ponction avait donné issue à une quantité assez considérable de pus.

Mais n'y a-t-il rien qui vienne affaiblir l'opinion d'un abcès froid? Malgré la présence, dans les environs du bassin, d'une grande quantité de ganglions lymphatiques, la haute région existant entre l'ombilic et le pubis, la crête de l'os des îles d'un côté et la crête de l'os des îles du côté opposé, n'est pas celle où viennent ordinairement ces abcès froids. Le tissu cellulaire n'y est pas assez abondant, et c'est plutôt aux environs de la région profonde des psoas que ces collections purulentes se forment. D'un autre côté, le pus provenant de tous les abcès situés aux environs du petit bassin, et qui a séjourné quelque temps contre les parois intestinales du cœcum, colon ou rectum principalement, exhale une odeur de matière fécale qui n'a point été perçue chez Lacaribe.

Il n'est donc pas sans contestation que la cavité sur laquelle nous faisons ce commentaire soit *un abcès* dans l'intérieur duquel s'est formée comme une membrane pyogénique, pas aussi distincte des parties voisines que la coque fibreuse des kystes, mais ayant procédé comme certains amas d'acéphalocistes qui se creusent un domicile pour ainsi dire au centre des fibres d'un organe qu'ils détruisent graduellement.

L'abondante exhalation sanguine rencontrée à la nécropsie, exhalation d'un liquide comme artériel, et qui pouvait bien avoir accéléré la mort, si on en juge par la décoloration de tous les tissus, et surtout de ceux de la masse intestinale entière; cette abondante exhalation, disons-nous, est assez difficile au premier abord

à expliquer dans une membrane pathologique qui paraissait dépourvue de vaisseaux. Mais cependant, *malgré ce qui s'est passé vers le rectum, circonstance sur laquelle nous reviendrons*, on doit croire que cette hémorragie intérieure avait eu lieu dans la membrane pyogénique ci-dessus mentionnée; l'expérience vient analogiquement appuyer ce fait et cette opinion. Le lendemain du jour où l'autopsie de Lacarribe a été pratiquée, une autre femme anémique, dans un état d'affaiblissement intellectuel, succombait et présentait un abcès dans la région lombaire, abcès vaste, arrondi, de 15 centimètres de diamètre, situé entre les muscles et la peau, lequel, ponctionné la veille, avait donné issue à beaucoup de pus, et quatre heures après cette ponction était devenue le siége d'une hémorragie qui s'était écoulée au dehors et avait produit un grand nombre de caillots intérieurs que l'on a retrouvés à l'examen du cadavre. Les circonstances qui avaient entouré la ponction de cet abcès, la région qu'il occupait, l'amincissement de la peau qui le recouvrait, ne pouvaient faire soupçonner la lésion traumatique d'un vaisseau, et laissaient subsister l'hypothèse de l'exhalation. L'intérieur de l'abcès était coloré par le sang, mais n'offrait point une teinte phlegmasique inhérente au tissu même. Cette hémorragie a été une des circonstances qui ont marqué les derniers instants de la vie et ont accéléré la mort.

Cette exhalation sanguine n'est pas cependant une circonstance catégorique d'un *abcès;* on la retrouve aussi quelquefois dans l'intérieur d'un kyste, ainsi qu'un exemple s'en est présenté dans la clinique de M. Gintrac, observation septième mentionnée dans la thèse inaugurale de M. Henri Gintrac.

Si on admet un moment comme fait, au lieu d'une simple hypothèse, l'existence d'un abcès ayant son siége dans le tissu cellulaire du bas abdomen, on pourrait se livrer aux détails explicatifs qui suivent et qui rendraient compte de la manière dont cette cavité s'est comportée pour ainsi dire.

Le péritoine aurait été séparé par la cavité de l'abcès des autres parties de la paroi antérieure et supérieure de l'abdomen, suivant les limites qui ont été indiquées. Dans l'hypogastre et le petit bassin les modifications anormales sont devenues plus complexes, et leur complexité s'explique par le nombre et la forme des viscères que revêt la poche séreuse. Cependant, si on se laisse entraîner par cette croyance que la cavité abdominale ne communique pas et n'a jamais communiqué avec la cavité de nouvelle formation dont il s'agit, ce que paraît indiquer l'absence de toute perforation et cicatrice, on pourrait comprendre comment la membrane séreuse, après avoir été séparée par décollement des parois de l'abdomen, s'est aussi séparée lentement et de la même manière de la vessie, de la matrice, de la portion supérieure et postérieure du vagin qu'elle tapisse; on pourrait expliquer comment l'espèce de cul-de-sac qu'elle forme entre la vessie et la matrice a été effacé, comment la matrice s'est trouvée ainsi dénudée et presque isolée de ses ligaments; enfin comment ses replis ont pu être détruits en partie du côté gauche, presque en totalité du côté droit, sans que néanmoins il y ait eu perforation du péritoine.

J'arrive à la question la plus pâtilleuse et qui résume presque toute la partie anatomique de ce petit travail. Cette question aurait dû peut-être fournir sujet à mes premières réflexions; mais n'importe le rang qu'elle occupe, elle arrive maintenant éclairée par les détails qui précèdent et qui en forment comme l'introduction.

La tumeur était-elle intrà, ou extrà-péritonéale?

Des impressions anciennes, datant du premier mois de la maladie, m'avaient disposé à admettre dans l'intérieur du péritoine, cloisonné déjà dans beaucoup de points, une cloison beaucoup plus large, limitée par des adhérences séreuses (c'était la cavité dont je m'occupe) et dans laquelle une sérosité purulente ayant séjourné s'était corrompue, s'était déposée en couches pour former une membrane anormale, avait donné lieu à une exhalation sanguine, enfin avait subi les nombreuses variétés de transformation que produisent des circonstances pathologiques pareilles.

Les paracenthèses opérées sur le côté gauche de l'hypogastre et qui avaient donné lieu à l'issue de sérosité purulente semblable à celles formées dans les péritonites puerpérales, venaient s'arranger parfaitement avec mes croyances de diagnostic.

Mais le sévère examen nécropsique, qui ne donne rien au hasard ni aux prévisions, et qui est inflexible comme un fait, m'oblige à changer de manière de voir. La cavité anormale décrite plus haut était évidemment extrà-péritonéale; car, cherché avec le plus grand soin avant et après macération, il est impossible de retrouver, sous la membrane muqueuse de nouvelle formation, aucun vestige de membrane séreuse.

Nous avons déjà mentionné l'absence de toute cicatrice ou trace de déchirure dans cette cavité anormale, ce qui repousse le soupçon d'une communication antérieure avec le sac péritonéal, et du passage des produits séreux de celui-ci dans l'intérieur de celle-là.

Il serait aussi assez difficile d'admettre que les adhérences produites par l'inflammation, et dont nous avons rencontré tant de formes diverses entre tant d'organes, n'aient pas, dans les limites qu'elles auraient tracées à l'entour de la cavité factice, compris et emprisonné pour ainsi dire quelques-uns (en partie) des organes renfermés dans la poche séreuse. Il n'en était pourtant rien; car toute la masse intestinale, les reins, la rate, étaient refoulés en haut et en dehors de l'abcès. Il est aussi à remarquer que l'investigation la plus scrupuleuse n'a pas retrouvé le feuillet séreux qui tapisse, dans le petit bassin, une partie de la matrice et du vagin.

On pourrait bien tenter d'objecter que le vaste désordre ayant son siége dans l'hypogastre et ayant détruit les ovaires, une partie des ligaments larges a bien pu comprendre, dans une désorganisation au sein de laquelle il est devenu impossible de faire descendre l'analyse, le feuillet péritonéal. Mais cette objection n'est pas sérieuse; car, d'un côté, on l'a retrouvé à l'extérieur, et, d'un autre côté, il était absent dans toute l'étendue de ce même kyste, circonstance qui serait extraordinaire s'il eût existé; car un mal quelconque ne ronge pas partout également.

La cavité accidentelle que portait Lacaribe était donc extrà-péritonéale. Nous en avons décrit le mé-

canisme un peu plus haut et sous forme d'hypothèse, maintenant nous l'admettons comme explication exacte. Celle-ci entraîne, comme conséquence, la preuve d'un abcès de cause spéciale (nous dirons bientôt laquelle), développé dans le tissu cellulaire du petit bassin; car nous ne pouvons nous arrêter (la donnée d'un kyste ayant été repoussée) à la pensée d'un kyste ovarique, qu'il nous a d'ailleurs été impossible de reconnaître, les ovaires ayant été entièrement détruits, soit primitivement, soit secondairement; abcès qui s'est développé de bas en haut en repoussant devant lui le péritoine, les organes que cette membrane enveloppe.

Cet abcès est resté clos de partout pendant longtemps. On peut soupçonner que c'est dans son sein qu'est arrivée la canule du trois-quarts dans quelques-unes des dernières paracenthèses qui ont eu lieu. Il est croyable encore que les déjections purulentes qui se sont montrées dans une période de la maladie de Lacaribe, provenaient de cet abcès ouvert dans la partie du rectum qui lui correspondait en bas et à gauche. La communication trouvée à l'autopsie entre cet abcès et le rectum, vient confirmer cette croyance et presque la prouver.

Puisque nous sommes arrivé à parler de la communication entre le rectum et l'abcès, nous allons compléter ce qui a trait à ce sujet.

La fin de l'intestin rectum (8 centimètr. de l'anus) se trouvait en contact avec le foyer de l'abcès, et il a été dit qu'une libre communication existait entre eux. Deux ouvertures séparées par un pont (selon toutes les apparences ancienne *cicatrice*) formaient cette communication. C'est par elle que, dans les premiers temps de la maladie, l'abcès paraît s'être vidé. C'est encore par elle que, dans les derniers jours de la vie, sortait un liquide très-fétide, et que les infirmières assurent avoir été de couleur blanchâtre et purulente. Les détails de l'affection qu'a présentée Lacaribe font penser que la bandelette organisée qui réunit les deux ouvertures, n'est qu'un produit réparateur formé par la nature, pour une première guérison, mais qui plus tard a été détruite à ses deux extrémités, pour venir en aide, par des ressources dont l'art ne dispose pas, à des désordres subséquents.

Cet intestin rectum était rempli et tapissé d'une sécrétion sanglante; l'abcès recélait aussi une sérosité sanglante et des caillots fibrineux. Y avait-il identité dans la cause de cette exhalation? Y avait-il seulement simple coïncidence? c'est ce qu'il n'est pas aisé de prouver. Si le sang épanché dans le rectum venait de l'abcès, comment expliquer les dernières évacuations alvines purulentes? Si le sang venait du rectum dans l'abcès, n'aurait-on pas dû trouver autre chose qu'une simple imbibition de la muqueuse? Mais dans les ulcérations quelques vaisseaux ont pu être ouverts? c'est possible.... Cependant les vaisseaux devaient être bien petits et les caillots fibrineux équivalaient à un poids de 150 grammes. Nouvelle difficulté pour le cours logique des idées. En résumé, et néanmoins, de tout cela je pense que l'exhalation sanguine a eu lieu dans la poche de l'abcès, et s'est glissée légèrement dans le rectum par les ouvertures de communication. Cette exhalation n'est arrivée que tout à fait en dernier lieu,

au moment de la mort ; et l'on comprend ainsi la possibilité des déjections purulentes venant de l'intérieur de l'abcès, et en même temps la présence des caillots allongés et dont la date ne remontait pas très-haut.

L'ulcération rectale avait été autrefois une perforation, mais la nature en avait restauré le fond. Les communications existantes étaient d'une époque plus rapprochée ; et de ces dernières circonstances il résulte que la nature avait fait d'admirables efforts pour se dégager ; qu'elle avait vidé un premier abcès qui s'était presque oblitéré, et que, plus tard, cet abcès se reproduisait dans une plus grande étendue : elle avait rouvert d'autres issues pour conduire en dehors des sécrétions dégénérées.

Un accident formidable, l'hémorragie, est venu anéantir tout ce travail de conservation.

Disons un mot sur la cause première des désordres qui viennent d'être analysés. Ils remontent bien évidemment au fait de l'acte de la conception chez cette femme, et des phénomènes de grossesse et de parturition qui en ont été la suite. Il est à croire, en étudiant l'effet de ces dernières, que l'abcès a commencé à se former dans les derniers mois de la grossesse, ou pendant le travail de l'accouchement. Entre laquelle de ces deux époques faut-il opter ? Rien ne peut guider vers un choix, ces deux époques n'ayant présenté rien que d'ordinaire. L'abcès s'est-il développé après l'accouchement et par quelque disposition spéciale ? nul ne peut le dire. Tient-il à une disposition métastatique ou sympathique qui l'aurait lié à la même cause que la péritonite puerpérale ? je l'ignore. Est-il consécutif ou primitif à la phlegmasie séreuse ? l'observation met dans l'impossibilité de résoudre ces questions. Ce qui est certain, c'est que la présence de l'abcès s'est perdue dans le fait de l'inflammation séreuse et du symptôme puerpéral. Il a même été méconnu ; car on rapportait l'issue du pus, dans l'opération de la paracenthèse, à une péritonite purulente ; et, en dernier lieu, l'état de ballonnement et de tympanite de l'abdomen masquait le développement de la cavité anormale, qu'aucune douleur ne pouvait faire soupçonner.

Ces abcès néanmoins, d'une nature traumatique spéciale, se rencontrent quelquefois à la suite des accouchements ; ils sont désignés sous le nom d'*abcès puerpéraux*, qui s'ouvrent ou dans le vagin, ou dans l'intestin, ou se font jour au dehors vers le périnée, ou s'épanchent dans le péritoine ; de là des classifications dans certains auteurs. Leur formation est très-facile à comprendre : elle tient aux tiraillements énormes qu'imprime au tissu cellulaire du petit et du grand bassin le développement de la matrice, attachée à ces liens celluleux par l'intermédiaire de ses ligaments, et qui est entraînée jusque vers la région épigastrique. C'est dans les régions du petit bassin intermédiaire, entre la matrice et le rectum en bas, la matrice et la vessie en haut, sur les côtés du vagin, que se développent ces phlegmons que l'on peut dire traumatiquement occasionnés, et auxquels dispose l'abondance d'une trame large et élastique. A cette extension graduellement forcée, et qui par ce dernier moyen seul peut exister, joignez les contractions si violentes

qui marquent l'acte de l'accouchement, joignez encore les mouvements de disjonction et d'ébranlement qui se passent dans toutes les articulations des os du bassin, et qui nécessairement se communiquent aux parties molles qui, comme le tissu cellulaire, enveloppent, joignent et matelassent tous les tissus.

Sans l'admirable loi de prévoyance par laquelle la nature se met à l'abri d'accidents qui détruiraient un être en parturition, je ne concevrais pas la rareté qui existe dans les désordres sus-décrits.

Je me suis appesanti seulement dans cette observation sur les détails qui avaient rapport à l'*abcès* formé dans la partie inférieure de l'abdomen; je n'entre pas ici dans les réflexions qu'inspireraient la péritonite et les circonstances qu'elle a offertes, ses suites et sa guérison (car les brides organisées qui ont été trouvées entre les circonvolutions intestinales lors de l'autopsie, prouvent évidemment ce triomphe des forces vitales; de plus, Lacaribe est restée hors de l'hôpital pendant quatre mois). — Je mets aussi de côté les commentaires que me suggéreraient les derniers symptômes thoraciques observés chez cette malade, ainsi que cette prédisposition à une sécrétion purulente dans plusieurs organes, circonstances pathologiques qui ont été éclairées par l'autopsie, car l'auscultation a été justifiée. — Je m'en remets à l'attention judicieuse de mes lecteurs pour remplir les lacunes que j'ai laissées.

PAMPHLETS

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