**Contributions to the pathology and therapeutics of typhus fever / by Joseph Bell.**

THE

# GLASGOW MEDICAL JOURNAL.

VOL. VII.] JANUARY, 1860. [No. 28.

## ORIGINAL COMMUNICATIONS.

### I.—*Contributions to the Pathology and Therapeutics of Typhus Fever.* By JOSEPH BELL, M.D., one of the Physicians to the Glasgow Infirmary, &c., &c.

(*Read before the Glasgow Medical Society.*)

PRELIMINARY OBSERVATIONS.—At the present day typhus is considered to be a blood disease, depending on the presence of a poison in the blood; therefore, a true Septicæmia.

On this doctrine, and also from the eruption* which very generally appears, the disease is usually classed with the exanthemata. But variola, scarlatina, and rubeola have a series of local lesions special to each; whilst, on the other hand, many modern authorities maintain that typhus has no such peculiar local manifestation. If this opinion be correct, then typhus is not only essentially different from the exanthemata, but also from every other epidemic disease. The plague and other epidemics of bygone times had their specific local lesions. The cholera has likewise its peculiar manifestation. It is admitted that the form of fever denominated gastro-entérite by Broussais, fievre entero-mésentérique by Serres, fievre typhoïde by Louis, dothinenterite by Bretonneau, and typhoid by British and American writers, has assigned to it a peculiar affection of the elliptical patches of the ileum usually known as Peyer's glands; this lesion forming the basis of the modern controversy regarding the *non-identity* of typhus and typhoid fevers. In the present contribution it is not my intention to enter upon the consideration either of the doctrine of the blood

* The late Dr. Perry of this city was, if not the first, certainly one of the earliest writers, who directed attention to this important point.

poison, or the very questionable zymotic theory of Liebig regarding its *modus operandi.* It is my wish to avoid the seductive temptations of theory, as well as the fanciful attractions of speculation, and to confine myself to the humbler task of placing before the profession the facts which I have observed, in the examination of the bodies of those who have fallen victims to typhus—facts which will justify the conclusion that typhus, instead of standing isolated from every other malady, has "*a local habitation*" in which it specially manifests itself, and also that (in accordance with the laws which regulate the sequences in the phenomena of other exanthemata), secondary local diseases occur in typhus—affections which prove more fatal than the primary one.

It will not be denied but that typhus owes its fatality to local disturbance in one or more of the important organs; probably, in some instances, the derangement may be merely functional, but, in the great majority, it will be found to be structural. We observe the same circumstance in all the exanthemata. Few cases, if any, terminate fatally, without leaving evident traces of disease in some organ. Be it observed, that even in those instances in which no such alteration can be detected, we are not entitled to conclude that it does not exist; its presence may escape observation. Numerous pathological states, which we can easily detect at the present day, were overlooked by our predecessors. Our followers may in their turn arrive at much greater attainments.

But imperfect as the means of investigation at our disposal may be, I venture to say that very few instances of death occur, without affording evidences of a sufficient amount of local manifestation to account for the result. For example, in the most rapidly fatal cases of scarlatina, we find on dissection either effusion into the brain, or severe abdominal or other local disease. Doubtless, the inference usually deduced is, that these local effects result from the action of the morbid poison. This conclusion, probably, is the correct one; at all events, I do not mean to contest it, but simply to observe that the local disorder—no matter how produced—*deprived the patient of life.* So is it in typhus; death can be traced generally to some local disease. Whether or not it can be proved that this affection arises from the specific action of the supposed poison, the *fact* remains unaltered.

Judging from the results of my reading, combined with nearly twenty-five years' personal observation of the disease, I think I may affirm, that the great majority of deaths from typhus takes place either in consequence of cerebral, cardiac, pulmonary, or intestinal disease.

It becomes, therefore, a matter of the highest importance to have a well-defined knowledge of the symptoms, by which these respective affections can be detected. It is also of the greatest moment to understand correctly the relations which exist between these diseases, not only as to causation, but also as to their order

of sequence—to ascertain which is the primary, and which the secondary lesion.

Being convinced that post-mortem examinations afford the only correct means by which these questions can be solved—during the last twenty-three years I have omitted no opportunity which I could command, to inspect the bodies of those who died under my observation from typhus. To the numerous examinations which were made in private I do not intend to refer (further than that I may merely make a passing allusion or so), simply because it might be alleged by those whose opinions are not in accordance with my experience, that preconceived notions may have modified the description of the results of my observations.

Whilst I had charge of the fever hospital in the years 1852 and 1853, the patients were too numerous to permit time to make many inspections. The few cases that were then examined I do not intend to quote at length, but shall bring the principal post-mortem appearances under notice, at the proper part of this paper. Since my present appointment in November last year, fever cases have not been very numerous; I have therefore endeavoured to obtain an inspection in every fatal case. It is my wish to bring the results of these examinations under the notice of the readers of this Journal. Be it observed that these cases during the progress of the disease, and at the subsequent post-mortem examinations, were witnessed by the clerks and a very large number of students. (During the summer months, the clinical class consisted of upwards of 130 pupils.)

In the present paper, though I will confine my remarks to the pathological changes which were found in the muscular tissue of the heart, yet the morbid alterations which were detected in the intestines will be fully described. I intend to embrace an early opportunity of discussing these latter lesions. In the meantime, I beg to solicit special attention to the important facts, that the cases which I am about to narrate were undoubted instances of typhus, and that the abdominal lesions which were found, were those which Dr. Jenner and the other advocates of non-identity contend are never present in that disease, but, on the contrary, form the local pathological condition in the so-called typhoid.

## REPORT OF CASES.

*Case* I.—*Maculated Typhus Fever—Failure of Heart's Impulse and Loss of First Sound—Abdominal Symptoms—Death on the ninth day—Autopsy.*—(Reported by Mr. Shaw.)

P. M'C——, aged 9 years, admitted on the 19th May, 1859; pyrexial symptoms of four days' duration. On admission eyes suffused; tongue furred and dry; skin hot, but no eruption. Pulse 120. Pressure over abdomen gave rise to pain. Heart's sounds normal. Bowels confined.

On the 21st, an extensive eruption appeared over trunk and extremities; it was confluent and of a dark mulberry hue, and did not disappear on pressure. Abdominal tenderness increased. Sinapisms were ordered to abdomen.

On the 23rd, action of heart became feeble and exceedingly rapid; impulse indistinctly felt. He was ordered four ounces of wine.

On the 24th, the symptoms of exhaustion were much increased; he was ordered six ounces of brandy. He died at four o'clock p.m.

*Autopsy—Heart.*—A great portion of the walls of left ventricle near the base was softened, and had a dirty yellow hue, pitting on pressure. Under the microscope no normal muscular tissue could be detected in the softened portion; the fibres were broken down; their striæ destroyed and infiltrated with yellow granular matter, mixed with oil globules. Portions taken from the margins of the softened structure, exhibited the gradual disintegration of the striæ and fibres.

*Abdomen.* — The peritoneal coat of the small intestines at several places was found in a state of intense congestion; of a bright red hue, especially that of ileum and cœcum. Among the convolutions of the former numerous patches of lymph were attached, and some serum was effused. On cutting open the small intestines, the mucous membrane was found extensively congested, especially that of ileum at its lower portion. The patches of Peyer were much elevated, felt hard and granular to the finger; on opening the sacculi, they were found to be distended with a firm cheesy deposit. Many of these sacs had sloughed, leaving small ulcers—some of which were of the size of a split pea; others that of the head of a pin. These ulcers were found chiefly in the patches situated next to the valve. Numerous solitary follicles were enlarged, and filled with similar deposit. The vessels of the submucous tissue were excessively congested; this condition extended to cœcum, giving the mucous membrane a thickened appearance.

*Remarks.*—Death in this case arose partly from failure of the action of heart, and partly from the extensive abdominal inflammation. That the case was one of well-marked typhus, there could be no doubt. Besides presenting all the characteristic symptoms, there is another important fact; viz., the boy's father, aged 39 years, died ten days before him, on the 13th day, of typhus; and his mother died the week previous in the female ward, on the 11th day, of the same disease.

*Case* II.—*Maculated Typhus—Pericarditis—Friction Sound and Bellows Murmur—Loss of Impulse and First Sound of Heart—Delirium—Death on twelfth day—Autopsy.*—(Reported by Messrs. Shaw and Sloan.)

Peter M'C——, aged 21 years, by trade a baker; admitted May 31, 1859. Pyrexial symptoms of six days' duration; coun-

tenance flushed; eyes suffused; skin hot and dry; tongue furred. Pulse 100. Over abdomen a few red spots were discovered. Pressure over right iliac fossa caused pain. Bowels loose, but dejections not frequent.

On the 1st June, the eruption had increased in amount, and assumed a brick colour.

On the 2nd, a double friction sound was heard over cardiac region uninfluenced by respiration; pulse intermittent and rapid. A blister was applied over heart, and he was ordered small doses of chalk and mercury.

On the 3rd, the abdomen, chest, and extremities were covered with a dark mulberry-coloured eruption, which was confluent, and did not disappear on pressure. Heart's action feeble; sounds heard indistinctly; friction sound less intense. Pulse 130, but regular.

On the 4th, he became delirious; the friction sound disappeared, and was succeeded by a soft systolic bruit, heard loudest over the pulmonary artery and at epigastrium. Percussion detected extended pre-cordial dulness. Pulse 130, weak. He was ordered six ounces of wine.

On the 5th, the delirium had subsided; but as the debility still continued, six ounces of whisky were substituted for the wine.

On the 6th, the quantity was increased to twelve ounces. He died at eight o'clock p.m. on the 7th.

*Autopsy—Heart.*—Pericardium contained about two ounces of reddish fluid; several patches of lymph adhered to serous covering of heart. The left ventricle, at the base, was of a deep purple colour; œdematous on pressure, soft, and easily lacerated. This softened condition extended to near apex, gradually tapering to a point. The inner portion of the ventricular wall, throughout its whole course, was healthy in appearance; the softened part extended to about one-twelfth of an inch in thickness. A microscopic examination of this softened portion exhibited the same disintegrated condition of the muscular structure as was found in the first case.

*Abdomen.*—The serous coat of small intestines at several places was much congested, and was of a bright-red colour. On opening the intestines the mucous membrane, from the duodenum to the colon, presented a congested appearance. It was swollen and softened; numerous solitary follicles were filled with deposit. Some had sloughed. The inflammatory condition was much more intense in the ileum, cœcum, and upper part of colon. In the former, Peyer's elliptical groups were much elevated, and the follicles distended with pulpy matter. Near the cœcal valve, two of these patches were almost entirely destroyed. In the cœcum several small ulcers detected. The follicles of upper portion of colon were also prominent, and distended with soft

material, and several small patches of the entire mucous membrane destroyed.

*Remarks.*—Doubtless, in this case, the disease of heart and pericardium caused death. The occurrence of pericarditis may be considered uncommon. I have met with five other cases in the course of my practice. Louis mentions only one instance, and Dr. Stokes seems to have never seen a case. The softened condition of the muscular tissue was extensive. The destruction of the fibres was as complete as we found in the other instance.

The friction sound heard on the eighth day of the illness arose, no doubt, from the effusion of lymph. As soon as this became covered with fluid, the bellows murmur supplanted the friction sound.

With regard to the abdominal lesion, the serous coat of intestines did not present such an extensive and severe amount of inflammatory action as in Case 1. But the disease of the mucous coat was much more intense. Peyer's glands were much more severely involved, and contained a much softer product; and many had sloughed, and not a few ulcerated. It must, however, be remembered, that the patient lived to the twelfth day, the other only to the ninth. This circumstance accounts satisfactorily for the difference.

The few scattered red spots which were discovered on the day of admission (the 6th of the disease) might lead to the impression that the case was one of typhoid. But, in the first place, these red spots became vesicular in a few days; and, secondly, a most extensive dark-coloured confluent eruption appeared on the seventh day, and ultimately became petechial; that is, uninfluenced by pressure—thus establishing beyond doubt the typhus character of the attack.

*Case* III.—*Maculated and Petechial Typhus—Rapid Loss of Impulse, and First Sound of Heart over whole Cardiac Region—Death on the Eighth day—Autopsy.*—(Reported by Mr. Gilland.)

T. M'G., aged 18 years; admitted June 25, 1859—Pyrexial symptoms of three days' duration; face flushed, eyes congested, tongue furred, thirst urgent, skin hot and dry. Pulse 108, soft. Heart's sounds normal. Pressure over abdomen caused uneasiness. Bowels regular.

On the 27th, the impulse and first sound of heart being feeble, he was ordered six ounces of wine.

On the 28th, an extensive eruption of mulberry-coloured spots appeared over body and extremities. Pressure over right iliac fossa caused pain, and elicited slight gurgling. The impulse of heart was still feebly felt, and the first sound nearly inaudible. A blister was applied over right iliac fossa, and the wine continued.

On the 29th, at left apex of heart no impulse could be felt, and the first sound was quite inaudible. Over right apex, impulse and sounds were weakened. Eruption much darker in colour, and did

not disappear on pressure. Pulse 120. Eight ounces of whisky were substituted for the wine.

On the 30th, impulse of heart feeble over right apex, and first sound quite inaudible. Pulse 130, feeble. The whisky was increased to sixteen ounces, and he was ordered three grains of camphor every four hours. He died at 1.30 p.m.

*Autopsy—Heart.*—The walls of both ventricles softened, especially that of the left, which was divided into two nearly equal portions by a layer of coagulated fibrine, which extended from the base to apex. The outside portion was of a dirty-yellow colour, easily reduced to a pulp by pressure between the fingers. The inner half was nearly of normal consistence. Under the microscope, the outside portion exhibited the same destruction of muscular fibre as has been described in the other cases. But in this instance the disorganization was far more extensive and severe, involving a large portion of the outside of the right ventricular wall.

*Abdomen.*—Peyer's glands were found enlarged, and filled with softish deposit. The patches situated next to the cœcum were most diseased, several of the follicles being destroyed. The serous coat of ileum congested. The whole mucous membrane much congested; that of cœcum less so.

*Remarks.*—In this case the failure of heart's action was both rapid and fatal, the patient dying on the eighth day; the softening of both ventricles was most extensive. Taking into consideration the age of the patient and the extensive cardiac lesion, I have not met with a more severe attack since the epidemic of 1846–47, when such cases were very numerous. The pathological condition of Peyer's glands was not far advanced, but it must be recollected that the patient died on the eighth day. I may mention that the patient's mother and a younger brother were admitted into the fever wards on the 3rd and 5th July respectively, in consequence of well-marked typhus. Both recovered.

*Case* IV.—*Maculated Typhus—Loss of Impulse, and First Sound of Heart—Partial Amendment—Death on the eighteenth day—Autopsy.*—(Reported by Mr. Gilland.)

R. M'L., aged 20 years, admitted 6th August, 1859. Eight days previously was seized with rigors, followed by headache and pains throughout body, accompanied by debility and nausea. On admission he was in a drowsy condition, face flushed, conjunctiva injected; skin hot, pungent, and covered with an extensive and well-marked typhus eruption. Tongue coated in the centre, red at tip and margins. Pressure over abdomen caused considerable pain. Pulse 80, full. Bowels regular.

On the 8th, the eruption was much darker in colour. Pulse 120, feeble. Impulse of heart scarcely perceptible over left apex, and first sound feebly heard. Impulse and sounds normal at right apex. He was ordered an ounce of whisky every four hours.

On the 9th, no change of importance.

On the 10th, pulse was 130, face flushed; impulse and first sound of heart imperceptible at left apex, and weak at right. The whisky was continued, and he was ordered a grain and half of musk and two grains of camphor every four hours, and a blister to be applied over heart for four hours.

On the 11th and 12th no alteration; the pulse still quick, and the condition of heart not improved.

On the 13th, the impulse and sounds of heart were better; tongue cleaner, skin cooler; but pulse counted 135. Tenderness over right iliac fossa increased. The whisky was omitted, and a blister applied over lower part of abdomen.

On the 14th the abdominal tenderness was rather increased. Pulse 130, feeble. Tongue dry. Bowels confined. The musk and camphor were discontinued. An enema ordered, and an ounce of whisky every four hours. The debility increasing towards evening, he was ordered two ounces of brandy.

On the 16th he was much more exhausted; bowels had been opened by enema. Impulse and first sounds much weaker, and he complained of pain in abdomen. The wine was discontinued, and six ounces of whisky and four ounces of brandy substituted.

He died in the morning of the 17th, being the nineteenth day of the disease.

*Autopsy—Heart.*—The pericardium contained two ounces of serous fluid. Right and left cavities of heart filled with clotted blood. Endocardium of a deep-red colour, and a deposit of lymph lying over the mitral valve. Muscular tissue of both ventricles softened, especially that of left side. Near the base, at two points, the structure was almost pulpy, and of a dirty yellowish-brown colour. Under the microscope this mass exhibited no remains of muscular structure, but consisted of yellowish-coloured granules, nucleated cells, and fat globules. Around these pulpy masses, some of the muscular fibres were entirely disintegrated; others were only partially so, the striæ being destroyed, and a large quantity of molecular matter and yellowish-looking corpuscles existed between the fibres.

*Abdomen.*—There was a considerable quantity of serum effused into abdominal cavity, and a large quantity of lymph deposited among the convolutions of intestines and mesentery, the glands of which were enlarged and filled with a soft cheesy matter. The serous coat of small intestines at several places was much congested. On opening intestines the mucous membrane of the whole of the small bowels, and of a portion of the colon, was in a state of congestion, the vessels of the submucous tissue being enormously injected. Both the solitary and elliptical follicles of ileum were much diseased; some infiltrated with a soft dark-coloured deposit; several had sloughed, leaving ulcers of various sizes, from that of the head of a pin to that of a halfpenny. The

largest ulcers were situated nearest to the ileo-cœcal valve, and occupied the site of Peyer's glands. In three of these ulcers, not only were the whole follicles of the patch destroyed, but also the submucous tissue—the muscular tunic forming the bottom of the ulcer. In a fourth the muscular coat was also destroyed, the peritoneal covering only remaining; it was of a scarlet-red colour for the space of several inches, and had several patches of lymph adhering to it. These large ulcers had thickened elevated edges. In the cœcum several small ulcers existed. In the upper part of colon the mucous coat was dark, and the vessels injected with blood. The follicles were very prominent, in consequence of being filled with a dark-coloured deposit.

*Remarks.*—In this case the abdominal lesion had proceeded to a much greater extent than in any of the others; but the disease had existed longer, the patient dying on the nineteenth day. The case during life had all the characteristic symptoms of typhus. I have do doubt but that the rapid extension of the ulcerative process to the peritoneum was the cause of death. No doubt the softening of the heart contributed essentially to the fatal result.

Case V. *Maculated Typhus—Failure of Impulse and First Sound of Heart—Second Sound becoming feeble—Death on the Fifteenth Day—Autopsy.* (Reported by Mr. Gilland.)

W. H., aged 36 years, admitted 17th September, 1859. Was seized, eight days previously, with rigors, followed by febrile symptoms. Face flushed; eyes suffused; breathing hurried. Complains of pains of body. Has slight cough with little expectoration. Percussion of chest elicits nothing abnormal. Auscultation detects slight sonorous rales over the large bronchii. Skin hot and dry, and extensively covered with a distinct typhus eruption. Pulse 120; feeble. Tongue furred. Bowels costive. He was ordered an ounce of castor oil.

On the 19th the first sound of heart became feeble and the impulse impaired. He was ordered four ounces of whisky.

On the 20th this was increased to eight ounces.

On the 21st impulse of heart was very feeble, and both sounds faintly heard. The eruption had become very dark-coloured. The whisky was increased to twelve ounces, and a blister ordered to be placed over heart for four hours, and then to be applied to epigastrium for the same period.

On the 22nd and 23rd there was no change of note, except a tendency to coma, for which a blister was applied to the nape of neck.

He died on the 24th.

*Autopsy — Heart.*— The pericardium contained about two ounces of red-coloured fluid; the heart was hypertrophied; the walls of both ventricles softened, especially that of left side. The microscope revealed the same kind of disintegration previously described.

*Abdomen.*—The peritoneal coat of small intestines, and also of posterior part of colon, congested at several places. On opening ileum, the mucous membrane was found swollen, the submucous vessels much congested, Peyer's patches elevated, and the sacculi filled with pulpy matter. Several of these sacculi had sloughed, leaving small ulcerated spots. This was especially the case near the cœcum. At other parts of the ileum the mucous membrane was abraded. In the cœcum the mucous membrane was of a deep-red colour, and effusion existed in the submucous tissue and in the follicles, several of which formed elevated projections of a dark-orange colour. The follicles in the upper part of colon were also diseased; some filled with deposit, and others ulcerated.

*Remarks.*—In this case the cardiac softening was extensive. In the intestines the disease had proceeded to ulceration of many of the follicles, especially those of Peyer. This was not so far advanced, however, as in Case IV.; but the disease had not lasted so long, the patient dying on the fifteenth day. The case was a well-marked example of typhus.

Commentary.—In commenting on these five cases, as I have already premised, I will divide my observations into two heads:—

First, The condition of the heart.

Secondly, The state of the intestinal mucous membrane.

In this paper I intend to confine my remarks to the first subject; deferring the consideration of the second till next number of the Journal.

I. The Softening of the Heart.—Laennec, in his work on "Diseases of the Chest," directed attention to the occurrence of this condition in cases of idiopathic fever. He considered, however, that the heart suffered merely in common with the other muscles of the body. He observes, "The variety of softening which accompanies idiopathic fevers, does not in general present any change of colour in the heart, or it is attended with a deeper colour than natural, approaching purple; sometimes, however, it is yellowish. I think it may be compared to that adhesive softness of the other muscles often observed in these cases, and which is also accompanied by a degree of redness greater than natural. This softening of the heart, as well as the analogous gluey or fishy state of the muscles, is particularly observable in putrid fevers, more especially when these exhibit the phenomena formerly considered as marks of putridity; viz., livid intumescence of the face, softening of the lips, gums, and internal membrane of the mouth, black coating on the tongue and gums, earthy aspect of the skin, distended abdomen, and very fetid dejections. I cannot assert that this softening of the heart exists in all kinds of continued fevers; but I have met with it constantly in such cases as I have attended to, and I have always thought it more marked in proportion as the signs of an alteration in the

fluids were more evident.—(Translation by Forbes, 4th edition, pp. 569–70.) Louis, in his excellent treatise on "Gastro-enterite," has shown that the opinion of Laennec regarding the softening of the heart (being nothing more than that which was common to "the other muscles in fever") is erroneous. He remarks that no similar lesion was found in any muscular organ; as all the muscles which preside over voluntary motion preserved amid the general disorder their natural colour and consistence. "Un autre fait, qui ne me semble pas moins remarquable que la rapidité du ramollissement du cœur; c'est qu'on ne trouvait de lesion semblable dans aucun autre organe musculaire; tous les muscles qui président aux mouvements voluntaires conservant, au milieu du disordre general la consistance et la couleur qu'ils offrent dans l'etat naturel."—(Op. cit., tom. i., p. 333.)

Dr. Stokes is the next great authority on this interesting pathological condition. He quite agrees with Louis in considering the lesion peculiar to the muscular tissue of the heart, and looks upon it as one of the secondary morbid processes peculiar to the fever. It will afford me much pleasure to point out in the sequel, how much we are indebted to Dr. Stokes for furnishing us with a correct knowledge of the symptoms, by which this softening can be detected during life, and also for placing its treatment on proper principles.

In discussing these important matters, I beg to divide the subject into the following heads:—

1. The Physical and Microscopic Appearances of the Softened tissue.
2. The Situation and Extent of the Softening.
3. The Pathological Nature of the Affection.
4. The Symptoms.
5. The Prognosis.
6. The Diagnosis.
7. The Stage of Typhus at which the Symptoms become developed.
8. Frequency and Fatality.
9. The Treatment.
10. The Diagnosis and Treatment of Cerebral and Pulmonary Complications.

1. The Appearances of the Softened Structure:—

*a. Colour* is variable; sometimes livid, at others yellowish; again of a deep-crimson or violet-purple.

*b. Consistence*—The change in this respect is very remarkable. The muscular tissue becomes exceedingly soft and friable, easily lacerated by the fingers. Sometimes very slight pressure reduces it to a pulp. At others a thick gluey fluid is easily pressed out. The microscope reveals still more striking changes. The muscular fasciculi are found completely disintegrated; some of the fibres being entirely destroyed, others having the striæ only

annihilated, and a quantity of cells, nuclei, yellow granular matter, and oil globules substituted. When a thin section is taken from the margins of the softened portions, the muscular fibres are seen undergoing the process of this degeneration, the striæ disappearing and the fibres breaking up, and having infiltrated between them large quantities of yellowish granular matter and cellular formations. Histologically speaking, therefore, the softening consists of two parts—1st. Infiltration between the muscular fibres of a fluid containing granular matter, nuclei, cells, and oil globules. 2nd. The ultimate conversion of the muscular fibrils and their striæ into a yellow granular substance abounding in oil globules.

2. The Situation and Extent of the Softening.—The left ventricle is much more frequently affected than the right. This has been found to be the case by Louis, Stokes, and myself. In three of the cases which I have narrated, the disease was entirely confined to the left ventricle, and especially to the outside portion of the wall near the base. In the other two cases the wall of the right ventricle was also softened—in one a small portion only at the base; in the other the greater portion of the wall: but this patient had laboured under hypertrophy of the heart for some years.

Another important feature in these cases was, that the softening did not penetrate through the whole thickness of the ventricular wall, but extended only to a certain depth, sometimes one-eighth, at others one-fourth, and in one case one-half, of its thickness. The naked eye could easily recognize the line of demarcation between the softened and healthy structure. In Case III. this line of separation was very obvious, being formed by a layer of coagulated fibrine, on the outer side of which the disintegration of tissue was most complete, and in its inside the fibres were perfectly healthy. Similar appearances have been described by Louis and Stokes.

We perceive, therefore, that the softening is situated principally at the outside of the base of the left ventricular wall, and only occasionally extends to the right ventricle.

3. The Pathological Nature of the Softening:—

(*a*) *Decomposition.*—The facts which I have already quoted from Louis are sufficient to refute the opinion of Laennec, viz., that the softened condition arises from simple putrefaction, which was common to all muscles in fever. Dr. Stokes justly remarks—

"On these latter points it is important to dwell, as some have held that the condition of the heart observed in these cases was the result of putrefactive decomposition; but such a view cannot be admitted. In the cases observed by Louis, the alteration was confined to the left side of the heart, and our observations of the signs during life, and from dissection, establish that the left ventricle is the portion of the heart first and most prominently engaged. We have, it is true, observed the phenomena of softening of the entire heart, but all our

results are confirmatory of those of Louis; and it would be a strange sort of putrefaction which would stop short at the left and not affect the right ventricle. But further, in many of our cases dissection was performed before putrefaction of the body had commenced, and, as we have before remarked, the voluntary muscles were unaltered. Again, we often observed that the change of the muscular structure only penetrated to a certain depth, and was found to terminate by a well-defined line. Thus, we could see the external portion of the ventricle affected to the depth of from one-eighth to the fifth of a line, and in such cases the musculi pectinati and columnæ carneæ remain unaffected. But the great argument against this argument [of putrefaction] is the fact of our having been able to connect the important physical phenomena during life with the softened condition as observed after death. We have traced the rise, progress, and retrocession of the weakened condition during life, in cases which, from their close similarity with those that have presented this special alteration of the heart after death, furnish the strongest possible proof that the patients had laboured under, and afterwards recovered from, this peculiar alteration of the heart."—*Disease of the Heart*, p. 371.

Such facts prove most satisfactorily that the softening of the heart is not the result of putrefactive decomposition.

(*b*) *Inflammation.*—Both Louis and Stokes regard the disease as a secondary effect of typhus poison, but contend that the *change* is of a *non*-inflammatory nature. Whilst the first part of this proposition may be readily admitted, yet the correctness of the last clause may be questioned. Let us examine the grounds upon which Louis and Stokes have founded their conclusions.

Louis states that he found "in nearly all the cases of softening, the walls of the ventricles were evidently much less thick than usual, those of the left especially, which were often only three lines thick.* He also informs us that he found the parts diseased, of a pale colour, and devoid of moisture, and without either purulent secretion, or effusion into pericardium :—

"If these facts," he observes "are insufficient to enable us to discover the cause of the softening of the heart, at least they exclude the idea of one of those affections which usually induce a great number of diseases, viz., inflammation. For how can we allow that inflammation is the cause of an acute softening, accompanied by a diminution of thickness, paleness of colour, and a kind of dryness of texture which is the seat of it? . . . If we knew any cause of disease exactly the reverse of inflammation, it would be proper to refer this softening to it. . . . The walls of the heart, although more or less softened, showed no purulent secretion, nor was there any inflammation of the pericardium, which would have been the case had this softening been caused by inflammation.†

---

* Un autre fait qu'il importe de signaler, c'est que dans presque tous les cas de ramollissement dont il s'agit, les parois des ventricules, celles du gauche surtout, étaient manifestement moins èpaisses, et de beaucoup, que dans l'était naturel (souvent trois lignes pour le ventricule aortique). Et comme cette diminution d'èpaisseur était bornée aux cas de ramollissement, il faut la considerer ce me semble, comme une disposition morbide.—(Op. cit., tom. i. p. 531.)

†Si ces faits sont encore insuffisants pour découvrir la cause du ramollissement du cœur, au moins sont-ils exclusifs de l'une de celles qui president a un grand nombre des lesions, je veux dire l'inflammation. Car comment l'admettre dans un ramollisement aigu, accompagné de l'amincissement, de la décoloration et d'une

In estimating the value of these statements we must take into account that Louis and the French pathologists of his time, regarded "rubor" as the essential phenomenon of inflammation; they could not conceive it possible that the inflammatory process could take place without the presence of *hyperæmia* and *stasis*. But at the present day we know that hyperæmia is by no means an essential condition of inflammation; consequently the absence of redness and tumefaction noticed by Louis will not authorize the conclusion, that the softening of the heart is not the result of inflammatory action.

But another important question arises, namely; Are the conditions mentioned by Louis, viz., the thinness, dryness, and paleness, uniformly present? Dr. Stokes states that in his experience he never was able to detect any such appearances. He observes—"It will be seen that in many particulars there is a coincidence between our results and those of Louis; we have not however, observed the *thinning* of the parietes of the heart, nor the dry and unpolished aspect of the cut surface." He also informs us that he has found the softened part of the ventricle of a "livid hue," "generally dark, and it resembles the cortical structure of the kidney."

The appearances found in the cases which have been narrated in this paper, exactly coincide with this description. The colour was often of a deep red, the part affected was œdematous, and the substance when incised instead of presenting a dry surface, was infiltrated with a dirty-yellowish, gummy exudation. Hence we perceive that the very facts on which Louis founded his opinion are not of uniform occurrence, consequently his reasoning becomes inconclusive.

Dr. Stokes places great consideration on the absence of all traces of inflammation in the pericardium, and also on the circumstance, that "out of many hundreds of cases of weak and softened hearts, observed during the last twelve years, we cannot adduce a single instance of organic disease of the heart which could be traced to any injury done pending the typhus affection."

I regret to state that my experience has not been so free from instances of permanent organic lesion. One patient, in whom all the symptoms indicative of softened heart existed during an attack of typhus, two years after recovery was seized suddenly with symptoms of acute pericarditis; he died within forty-eight hours. On inspection it was ascertained that an abscess had existed in the wall of left ventricle, and had burst into the peri-

sorte d'aridité du tissu qui en est le siege. . . Si l'on connaissait une cause de lésions opposée a l'inflammation, il serait naturel de lui rapporter ramollissement qui nous occupe. . . . Les parois du cœur, plus un moins ramollies, n'effraient de trace de pas dans aucun cas: dans aucun, il n'y avait inflammation du pericarde; inflammation qui devrait être assez frequente dans le ramollisement du cœur, s'il était de nature inflammatoire. (Op. cit. tom. i., pp. 331, 332.)

cardium. In another instance of the same class of cases, the patient died from phthisis, eighteen months after recovery from an attack of typhus, during which he exhibited the symptoms of softened heart. On inspection after death, an abscess about the size of a hazel nut was found in the substance of the left ventricle, near its base. I have no doubt but that these purulent collections resulted from the disintegration which had occurred during the attack of fever. In Cases 3 and 4, I feel convinced that the pulpy masses which were detected would have ultimately formed abscesses, had the patients recovered from the attack of typhus.

The history of such patients should be carefully watched for years. Even if abscesses should not result, very probably the foundation of chronic fatty degeneration may be laid. The subject is a highly important and interesting one, and demands further exact observation.

With regard to the arguments founded on the absence of the signs of pericarditis, with all due deference to Dr. Stokes, I venture to remark, that surely it would be a much more correct method of determining the question, to compare the condition of the softened part, with the effects of inflammatory action on other muscles, than to seek for information in the condition of the pericardium. It must not be overlooked, that it is the muscular structure of the heart which is involved, and not its covering. If we turn to any trustworthy authority on pathology, we will find that the state produced by inflammation on the muscular fibres is exactly similar to that which we find in the softened heart. For example, Rokitansky tells us, that—

"At first some redness and injection are perceived; in a little, infiltration of the interstitial cellular tissue. No change is discernible at this period in the muscular fibre itself, but as soon as an actual exudation appears, the muscular fibre becomes discoloured; it changes to a pale red, or reddish-yellow or fawn colour. If the inflammation continue, and the exudation be not absorbed, an important change soon takes place in the texture of the muscle. The fibres lose their transverse striæ, and the fibrils degenerate into a granular mass."

He also remarks, that the peculiar characters of the change are most evident when the inflammation is confined to isolated spots, such as we find in the case of the heart under consideration. "The muscular fibres are then found in the midst of the exudation discoloured, disintegrated, and forming dull, yellowish-red, or fawn-coloured, here and there interrupted stripes. The exudation in which they lie may be a gelatinous, greyish, or greyish-yellow substance; or firmer and reddish or yellowish, or purulent and yellow, or tubercular and cheese-like, or red, filled with blood, and half-coagulated."*

It will be seen that this description exactly represents the condition of the softened portion of the heart found in the cases which

* Path. Anat., vol. iii., p. 308, Syden. Soc. edit.

I have narrated, and that it also completely coincides with the description given by Dr. Stokes himself. Again, if we turn to Wedl's account of the effects of inflammation on muscular tissue, we find a similar description. He says that "the fasciculi adhere more closely together, and in the aggregate appear of a paler colour. In the muscular parenchyma, in this state of gelatinous metamorphosis, opaque spots may be noticed inclosing primitive fasciculi, transformed into a fine molecular substance. Their consistence is thus so far reduced that in some places the limits of the individual fasciculi can no longer be distinguished. The sarcolemma is so much softened by the exudation as to be dissolved, leaving nothing but a fine molecular substance, in which not a single primitive fasciculus can be discerned."—(Path. Histol., p. 241, Syden. Soc. ed.)

Lastly, Virchow has described the condition in almost the same terms, denominating the pathological change "inflammatory softening."

Whether or not we can prove the softening of the heart to be the result of inflammation, it cannot be questioned but that the condition of the muscular fibres is identical with that which is produced by inflammatory action in muscular tissue.

Again, though Dr. Stokes has never met with a case in which the results of pericarditis were detected, yet I have found unmistakable evidence of such disease in three instances, besides the two narrated in this paper. I am certain that I have met with the same condition on other occasions; but as recovery fortunately took place, I was not able to verify my opinion by inspection. In these cases I have heard a friction sound, succeeded by a bellows murmur, which disappeared in a few days. Dr. Stokes states that he has also detected such bellows murmurs, but he ascribes the sound to "a nervous or ænemic condition."

The foregoing considerations induce me to doubt the correctness of the doctrine of the non-inflammatory nature of the softened heart in typhus. On the other hand, I am inclined to view the condition as a secondary effect of the typhus poison on the structure of the heart, and that this pathological change is similar in its nature to those morbid alterations which occasionally arise during fever in the brain and lungs, and which conditions are considered to be of an inflammatory nature, modified no doubt in the results by the typhus influence.

I have entered on this point at some length, as it is one of great practical importance in reference both to the therapeutics and symptoms of the disease. For example, we know that inflammation of a muscle gives rise to a greater or less amount of paralysis, and this is precisely the effect on the heart's action which the symptoms during life denote, viz., loss of impulse and first sound, especially at left side of heart, the portion which we find after death chiefly diseased.

4. Symptoms.—Though we are indebted to Laennec and Louis for having directed attention to this morbid condition, yet this knowledge was of no practical importance, so long as we remained in ignorance of the symptoms by which its occurrence could be detected during the life of the patient. To Dr. Stokes we are entirely indebted for having pointed out the symptoms upon which a correct diagnosis of the disease can be easily made. Of the many important advantages which the labours of this distinguished physician have conferred on the profession, none excel his contributions to our knowledge of this subject. He was the first to direct attention to the physical phenomena connected with the impulse and sounds of the heart as diagnostic of this softened state, and subsequent observers have confirmed his conclusions.

The earliest symptom is failure of the impulse of heart: this is first detected at the left side of apex, and gradually extends to the right. The next indication is feebleness and loss of the first sound of heart: this is also first discernible at the left, but soon extends to right apex. As the disease proceeds, both impulse and first sound become imperceptible over the whole cardiac region, the second sound only being audible. If the case be a very bad one, the second sound also becomes feeble. The symptoms are therefore progressive:—1st, Diminished impulse at left apex; 2nd, Cessation of first sound; 3rd, Impulse becoming imperceptible over right apex (the first sound being sometimes audible at this point); 4th, Loss of first sound over whole cardiac region; 5th, Diminution of the intensity of second sound.

5. Prognosis.—Dr. Stokes has not only shown that the physical phenomena now described exhibit a progression up to a maximum point, but that they also manifest a regular retrocession as the patient recovers. On these facts we are enabled, without any dubiety, to form our prognosis at each visit. During the period of retrogression, the symptoms generally follow an inverse order, the impulse being restored prior to the sound, and that at the right side of the heart before the left. The sound generally returns first at the right, and, secondly, at the left side, and at the base before the apex.

Occasionally we find considerable deviation from this order,—sometimes the impulse and sound return simultaneously; at others the sound precedes the impulse. It is also worthy of notice, that in many slight cases the impulse and sounds are only lost over left side of heart, and in some instances the impulse only is imperceptible, the first sound remaining feebly heard. In all these instances, however, the restoration is generally in the reverse order that marks the downward progress of disease. By attending to this series of physical phenomena, we can have no difficulty in arriving at a correct prognosis. Before leaving this part of the subject, there is a point to which I would beg

especial attention. If under the use of stimulants the action of the heart becomes more rapid, and the impulse restored, without the speedy return of first sound, an unfavourable prognosis may be pronounced. I have found such a combination almost always followed by a fatal result.

6. Diagnosis.—Simple debility of the action of the heart must not be confounded with *softening*. Weakness of the power of the heart occurs to some extent in every case of typhus. The organ suffers in common with the whole system. It is of importance to be able to distinguish between this common debility and the peculiar lesion on which we bestow the name of *softening*. The diagnostics of simple debility and softening depend more, as Dr. Stokes has remarked, on the progressive character of the physical phenomena than on any very essential difference between the symptoms. 1. In simple debility we have diminution of impulse and sounds occurring simultaneously, and the restoration is marked by the same character. But in softening the loss of impulse and sound is progressive, and their return equally so. 2. In simple debility the weakened impulse and sound are detected at the same period over the whole cardiac region; but in softening the loss is detected first at the left side, and gradually extends to right. Dr. Stokes remarks, "that in simple debility we have no predominance of morbid signs on the left, as compared with the right side, nor that of the second over the first sound, but both are diminished in loudness and become of a nearly similar character."—(Diseases of Heart, p. 379.)

It happens, however, that in some cases the symptoms of simple debility of the heart become supplanted by those indicative of softening; or, as the same author remarks, "both sets of phenomena occur at different periods, and as it were run one into the other." The symptoms of simple debility, however, always precede those of softening, so that we can have no difficulty, even under such circumstances, of arriving at a correct diagnosis.

7. The Stage of Typhus at which the Symptoms of Softening become developed.—Dr. Stokes states, that he has found the first indications presenting themselves about the fifth day of the fever. In my experience, though I have met with cases in which the symptoms appeared about the fifth day, yet in the majority of instances they did not appear till about the seventh day, oftentimes the eighth or ninth day, and in some instances as late as the tenth day. I may observe, however, that when the symptoms were late in being developed, the cases were mild, and recovery generally took place.

8. Frequency and Fatality.—I regret that I have no statistical information to give on these points. Dr. Stokes informs us that he has found the disease of frequent occurrence, characterizing the largest class of the cases of typhus, as it has appeared in

Dublin. Dr. Huss states, that out of 250 patients admitted into the Seraphim Hospital of Stockholm between the years 1840–1852, 170 had the systolic sound of the heart altered or lost.* In my own experience, I have found the symptoms of very frequent occurrence, especially in patients above twenty-five years of age. During the last thirteen years I have taken notes of a very large number of cases; but these are so scattered through my papers that it would require much more time than I can at present command, to collect and arrange them in any useful form. I may mention, however, that my excellent clerk, Mr. Gilland, has carefully examined the journals of the male fever ward for the past year, and he informs me that out of 40 admissions above 25 years of age, we had no fewer than 24 instances in which the peculiar symptoms of softening were present; and out of these 24 cases we had 13 deaths. It also appears that we had only 5 additional deaths from all other lesions. This shows how fatal the affection of the heart proves. I have, however, found, that the fatality varies in different epidemics. In that of 1846–47 it was very great, many patients dying from failure of heart's action about the seventh or eighth day of the disease, in spite of the most active stimulation. Stokes has also pointed out the fatal character of this lesion; and Louis states, that nearly the half of those patients who die between the eighth and the twentieth day of fever had softened hearts, and that it was present in a third of those who died afterwards. It must also be remembered, that death does not always arise from the condition of the heart itself, but from its secondary effects on other important organs, such as cerebral and pulmonary congestions and effusions. We therefore perceive, that softening of the heart is not only of frequent occurrence in typhus, but that it is the principal cause of the high mortality which takes place among patients who are above 30 years of age.

9. Treatment.—It is proper to premise our observations on this part of the subject by observing, that the contractile power of the ventricle is from the first impaired, and soon becomes paralysed. It is therefore the object of treatment to ward off this fatal catastrophe, and to *support* the action of the heart until the disease exhausts itself. The free exhibition of stimulants is the only treatment on which we can place reliance. Stimulants may be justly denominated our sheet-anchor. To succeed, they must be administered with no sparing hand. The maintenance of the action of the heart is the only limit to the quantity prescribed. The power of the heart must be restored. It is the loss of this power which demands the use of stimulants in typhus. We do not give this class of remedies for the cure of any of the other

* Statistics and Treatment of Typhus and Typhoid Fever. By M. Huss, M.D. Translated by Aberg.

local manifestations of the disease. It is the state of the heart alone which demands their employment. The weakened condition of the central organ of the circulation, is the principle on which stimulation in typhus should be solely conducted.

The daily careful examination of the heart by palpation and auscultation becomes the imperative duty of the physician. The pulse will not afford the necessary information—often it feels feeble when no abnormal physical phenomena exist, and as often feels full and comparatively strong when neither impulse can be felt, nor systolic sound heard. The pulse, therefore, is a most fallible guide as to the condition of the heart. We must examine the organ itself in order to obtain the requisite knowledge. As soon as a diminution of the impulse is detected, we must commence to give stimulants. If the quantity ordered be not sufficient to prevent farther weakening of impulse, or loss of systolic sound, we must increase the amount, and go on doing so, until the physical phenomena begin to recede, or in other words, until the normal sounds commence to be restored.

(1.) *The quantity of the stimulant* must vary according to the severity of the case. In some a few ounces of wine or whisky will be sufficient; in others pints may be demanded. It matters not how much, it must be administered until the downward progress of the symptoms be suspended. Fortunately we can never be at a loss to regulate the quantity of stimulants. The physical signs connected with the impulse and sounds of the heart form a most unerring guide—a monitor whose assistance we must constantly employ. The pulse will not avail us—we must go to the heart itself for trustworthy information, and by the instruction thence obtained regulate day by day the amount of stimulant to be administered.

(2.) *The kind of Stimulant.*—This must be determined partly by the age, sex, and habits of the patients, and partly by the severity of the case. To females, to young persons under twenty, and to patients of temperate habits, I prefer port wine. To adult males, especially those who are admitted into the fever hospital, I order whisky. The majority of such patients have been too long accustomed to the use of ardent spirits, to derive much stimulating effect from wine. Among the poorer classes of society, I have also preferred whisky to wine, because the latter can rarely if ever be obtained by them in a state of purity. Musk, camphor, and ammonia have been used by some practitioners as stimulants in fever. The two former have been much employed and strongly recommended by Dr. Huss of Stockholm. The camphor he employs in doses of from half a grain to one grain every other hour. The formula he uses is as follows :—

| | |
|---|---|
| Camphor,..... | eight grains |
| Powdered gum-arabic,..... | one drachm |
| Almond emulsion,..... | eight ounces. |

Of this mixture one or two table spoonfuls are ordered every other hour.

The musk he uses in doses of five grains every second hour, for five or six successive times. He also recommends, in severe cases, its combination with camphor. I have often tried these remedies, both singly and combined, in cases in which either whisky or wine produced excitement, or, in other words, when the frequency of the heart's action was increased without the restoration of the first sound. In the present contribution it will be found that musk and camphor were substituted for whisky, in consequence of the great rapidity of the heart's action which followed its use. From my experience of these agents, I can speak favourably of their influence.

Sesqui-carbonate of ammonia I have never found of any use. Patients dislike its taste. And from the fluid condition of the blood in typhus (if any reliance can be placed on the experiments of Dr. Richardson), we must conclude that the salts of ammonia might be productive of much evil.

(3.) *Blisters and Dry Cupping.*—Internal stimulants do not embrace all the agencies which we have at our command in cases of softened heart. Blisters I regard of the highest value, when applied for a few hours over and about the cardiac region—flying blisters, as they have been denominated by Dr. Graves, whose testimony to their beneficial influence is very strong. Others have expressed the same favourable opinions, which I can fully corroborate. Dr. Graves considered that they act as stimulants to the nervous system; but I am inclined to adopt the notion that they act as counter-irritants, and thereby relieve internal congestion.

Dry cupping I have employed in several cases with marked benefit. In two most severe cases which were recently in the hospital, this remedy, along with flying blisters, proved of signal service in conjunction with large quantities of stimulants; the latter alone proving inadequate to suspend the alarming symptoms of failure of the heart's power.

*Case* VI.—*Maculated Typhus—Failure and Loss of Impulse and First Sound of Heart on the Seventh Day—Recovery.*—(Reported by Mr. Gilland.)

P. C., aged 18, admitted October 18. Febrile symptoms of six days' duration. A profuse dark-coloured mulberry eruption over whole body. Pulse 120, rather feeble; but heart's impulse and sounds normal. On the 20th the eruption was darker in colour, the impulse of the heart was imperceptible, and the first sound feebly heard. He was ordered an ounce of port wine every six hours. Next day the first sound of heart could not be detected. The second sound was feeble. He was ordered an ounce of whisky every two hours, and the wine to be continued. Dry cupping to be employed half an hour over cardiac region, and

afterwards flying blisters to different parts of chest, each to be kept applied for the space of three hours.

On the 22nd, no change. The cupping was repeated, and the whisky increased to an ounce and a half every two hours. On the next day, the impulse of heart was much improved. The whisky was reduced to an ounce every two hours.

On the 24th, the first sound of heart was heard at right apex. The whisky was reduced to an ounce every four hours.

On the 25th, the first sound of heart was audible at left apex, and impulse restored. The whisky was suspended, and the wine continued. He gradually recovered, and was dismissed well on the 12th November.

In this case, the symptoms of weakened heart set in rapidly and severely. In consequence of this debilitated action of the heart, the stimulant seemed not to be sufficient to ward of congestion of the pulmonary organs. This case, I conceive, was very essentially benefited by the cupping and blistering. In the following case, the same marked advantage followed the use of these means.

*Case* VII.—*Maculated Typhus—Failure of Sounds and Impulse of Heart—Pericarditis—Congestion of Lungs, Pleura, &c.—Treatment by Stimulants—Dry Cupping and Blisters—Recovery.*—(Reported by Mr. Gilland.)

R. B., aged 47 years, admitted 28th October; typhus of eight days' duration; the eruption was extensive, confluent, and dark-coloured. Pulse full, 110; tongue and gums covered with sordes. Sonorous and sibilant rales extensively heard over chest. Impulse of heart feeble, and first sound faint.

On the next day, these symptoms were aggravated. He was ordered an ounce of wine every four hours; to have his chest dry cupped for an hour.

On the 30th his breathing was much oppressed; face much congested; lips livid. Impulse and first sound of heart imperceptible. The cupping was repeated. Additional to the wine, he was ordered an ounce of whisky every six hours.

On the next day, no improvement; the cupping was repeated, and flying blisters applied to chest; and the whisky increased to an ounce every three hours.

On 2nd November he was nearly comatose; the cupping repeated, and the whisky increased to an ounce every two hours.

On the 3rd he was much improved; impulse of heart restored. The whisky was reduced to an ounce every four hours.

On the 4th the first sound of heart was detected over base of heart; the whisky was further reduced to an ounce every eight hours.

On the 5th the stethoscope detected a double friction sound over heart, uninfluenced by respiration. Pulse was quick, and he complained of oppression at chest. He was ordered to have a

blister applied over heart, and to have the abraded surface afterwards dressed with mercurial ointment.

On the 6th, great oppression of breathing; action of heart feeble; friction sound still heard over heart. Mucous and sibilant rales extensively heard over both sides of chest; a friction sound over right mammary region; countenance anxious; extremities cold. The whisky was increased to two ounces every two hours.

On the next day he was rather better, but action of heart was still feeble; friction sounds gone, and less mucous rales heard over lungs. Pulse very small and feeble; surface covered with cold perspiration; low muttering delirium. The stimulant was continued.

On the 9th, action of heart was slightly improved; and, on the 10th, so very much so, that the whisky was reduced to an ounce every four hours.

On the 12th he was so much better that the stimulants were discontinued. He was dismissed well on the 19th November.

In this case, I feel assured that the counter-irritation and dry cupping were of essential assistance to the stimulants. The occurrence of the symptoms of pericarditis in this case is a point of some interest. These symptoms did not make their appearance till after the crisis; and the effect of the effusion on the action of the heart was most injurious, and would have proved fatal had it not been for the liberal use of stimulants, which were promptly ordered by Mr. Gilland in my absence.

In several cases similar to the two now quoted, I consider I have derived much advantage from the use of flying blisters and dry cupping. Instances in which the patient refused to take either whisky or wine, blistering and dry cupping have proved most valuable agents. In other cases in which the action of the heart became excited, and the face so flushed under the use of stimulants as to demand their discontinuance, I have found great benefit resulting from the application of blisters placed for the space of three or four hours over different parts of the chest. From these circumstances, I can strongly recommend the employment of these remedies as powerful adjuvants to the use of stimulants, in the treatment of the condition of the heart under consideration.

10. Treatment of Cerebral and Pulmonary Complications.—When either delirium or severe congestion of the lungs is associated with symptoms of softening of the heart, our treatment must be stimulating.

In Case II., the delirium was very considerable, but it yielded readily to the use of stimulants. In many instances of weakened heart, I have found not only symptoms of extensive congestion of the pulmonary organs, but even of effusion into air vesicles, bronchii, and pleural cavity, completely removed under the use

of stimulants. In fact, it matters not which organ or organs may become congested or inflamed; provided the impulse and systolic sound be impaired, we must employ stimulants. The action of the heart must be maintained at all hazards. We know that a patient may be insensible for several days, and yet recover. Another may have a large extent of the lungs congested and inflamed during a considerable period, without a fatal result. Again, the mucous membrane of intestines may be very extensively inflamed and ulcerated, yet life may be preserved. But, on the other hand, the action of the heart cannot be suspended for a very few minutes, without death supervening. Its power, therefore, must be maintained by every effort which we can make, without reference to any other lesion which may become manifested during the course of typhus.

These views were very ably advocated seventeen years ago by Dr. Hudson, physician to the Navan hospital;* and more recently by Dr. Corrigan in his excellent treatise on fever. It will be found that their experience fully corroborates the remarks which I have made on the subject.

I beg, however, to observe that two classes of cerebral affections arise during the course of typhus. Only one of these is connected with the symptoms indicative of cardiac weakness. In the other the impulse and sounds of heart are unimpaired; in such cases, stimulants would prove injurious; the proper treatment consists in the use of tartar emetic and other mild antiphlogistics. Pulmonary affections also occur in the course of fever, without any symptoms of weakened heart; to these cases the same remarks are applicable.

Hence we see that the labours of Dr. Stokes have not only resulted in important advantages to diagnosis, but also to practical therapeutics. In the sthenic and asthenic varieties of these cases, the general symptoms are almost identical. On the one hand, the same violent delirium, followed by coma; on the other, the same difficulty of breathing, followed by extensive pulmonary infiltration. It is the physical phenomena alone by which we can diagnose the cases connected with cardiac softening, from those in which its action is unimpaired; and consequently, in this way, we are enabled to adopt the plan of treatment which is best calculated to assist our patients, in their contest with such fatal complications.

It is obvious, therefore, that the examination of the state of the impulse and sounds of the heart in typhus, is not only of importance in reference to the state of the organ itself, but also becomes of immense utility, in enabling us to distinguish with certainty sthenic from asthenic congestion of other vital organs.

* Observations on the Connexion between Delirium and Certain States of the Heart in Fever, with Cases. By A. Hudson, M.B., T.C.D., Physician to the Navan Fever Hospital.—Dublin Journal of Medical Science, vol. xx., 1842.

I cannot lay down my pen without again alluding to the heavy obligation which we owe to Dr. Stokes for his invaluable researches on this subject. (1.) He has placed before us unmistakable and easily-detected symptoms, by which the presence and progress of the cardiac disease can be detected. (2.) He has placed the administration of stimulants in fever on the most certain and well-understood principles, by the application of which we can determine when to give, how much to give, and when to cease to give. (3.) He has furnished us with a key by the use of which we can easily distinguish between active and passive inflammatory conditions of the brain and lungs. To those who have not read either his original memoirs in the *Dublin Journal of Medical Science*, or the chapter—"On the Condition of the Heart in Typhus Fever;" in his classical work "On Diseases of the Heart and Aorta," I beg most respectfully to recommend a careful perusal.

In conclusion, I venture to offer the following propositions:—

1. That in numerous cases of typhus, about the fifth, sixth, or seventh day of the attack, the impulse and systolic sound of the heart become feeble and ultimately imperceptible.

2. That these symptoms indicate a morbid alteration in the structure of the muscular tissue of the heart, especially in the walls of the left ventricle.

3. That this alteration resembles the usual changes which result from congestion and inflammation of muscular structure.

4. That the nature of this pathological change requires further examination and research, because the evidences on which the doctrine of its non-inflammatory origin rest, are not conclusive; the circumstances on which Louis and Stokes have placed reliance being not uniformly present.

5. That the beneficial influence of stimulants does not prove the non-inflammatory nature of the morbid change, because, in asthenic inflammation, a stimulating treatment is always necessary.

6. That whether or not the pathological alteration be owing to inflammation, the softening must be regarded as one of the special secondary effects of typhus.

7. That the proper treatment is to maintain the action of the heart by stimulants.

8. That in cases of cerebral and pulmonary disturbance arising in connection with the symptoms of cardiac softening, a stimulating plan of treatment is indicated.

9. That the presence or absence of the physical symptoms diagnostic of softened heart, may be relied on as affording trustworthy evidence, by which the sthenic or asthenic nature of these cerebral and pulmonary affections can be determined.

From these propositions it follows as a *corollary*, that it is the duty of the physician, to devote the strictest attention to the action of the heart, especially as regards its impulse and sounds, throughout the course of every case of typhus.

## II. *On Laceration of the Perineum in Labour.*

By William Leishman, M.D., Fellow of the Faculty of Physicians and Surgeons, Surgeon to the University Lying-in Hospital.

(*Read before the Glasgow Medical Society.*)

I purpose, Mr. President, in bringing the subject of laceration of the perineum under the notice of the Society, to direct the attention of its members chiefly to the various means which are or have been adopted for its prevention. At the same time I shall submit certain conclusions at which I have arrived, in regard to which, while I lay no claim to originality, I think it right to state that they are the result of some study and considerable personal observation. Writers on midwifery differ very considerably in the methods which they recommend for the management of labour towards the end of its second stage. For my own information on this point, I have consulted many authors, both ancient and modern; and that with results so interesting to myself, that I trust the Society will pardon me, if I introduce the subject by directing attention to what I may call the history of perineal laceration, adhering as much as possible to chronological order in referring to individual authors.

At the outset of this inquiry I find the somewhat strange fact, that the medical writers among the ancients, prolix as they are on other points, have taken no notice of this, either as regards prevention or treatment. In the works of Eros, who lived in the thirteenth century, is the first indication that I can find of any attempt at prevention, which consists, in this case, of baths prepared with emollient herbs, and the external application of the oil of violets or of roses; while another accoucheur—Haly Abbas by name—in addition to similar topical medications, prescribes for internal administration an infusion of swallows' nests. This, we may presume, he prescribed empirically; at least, he does not condescend on any explanation of its probable action on the animal economy. The obstetricians of the seventeenth century, of whom the most distinguished are Guillemeau and Mauriceau, do not seem to have deviated from the path marked out by their predecessors. We have the same hot bathing and lubrication, the latter being varied only by the addition of such substances as dragon's-blood, oil of Saint John's-wort, and yolk of eggs. It was not, indeed, till about the middle of the eighteenth century that the subject came to be noticed in all systematic treatises on midwifery, and other methods suggested for its prevention. The first method, which differs in any essential point from those so long practised, is that of Pugh, which, although it is not a system likely to be adopted in these days, is interesting as indicating the fact which I have mentioned, with regard to the time at which the

THE

# GLASGOW MEDICAL JOURNAL.

VOL. VIII.] APRIL, 1860. [No. 29.

## ORIGINAL COMMUNICATIONS.

### I.—*Contributions to the Pathology and Therapeutics of Typhus.*

By JOSEPH BELL, M.D., one of the Physicians and Clinical Lecturers, Glasgow Royal Infirmary.

No. II.

#### LESIONS OF THE INTESTINAL MUCOUS MEMBRANE.

IN my last communication I solicited attention to two facts—1st, that the cases which I narrated were undoubted examples of typhus; and 2nd, that the abdominal lesions found to be present were those which the advocates of the "non-identity" theory contend are never present in that form of fever, but, on the contrary, constitute the pathological condition peculiar to the so-called typhoid. Before entering on the consideration of this important question, I deem it necessary to say a few words on the following points:—

1st. The nature of the abdominal lesion.

2nd. Its relation to the fever.

I. THE NATURE OF THE ABDOMINAL LESION.—The diseased state of the intestinal mucous membrane in fever is a discovery of modern times. Though glimmerings of a knowledge of this local affection are to be found in the works of Bonetius, and also in the writings of older authors, yet there is nothing definite to be discovered prior to the middle of the last century.

In the year 1750, Sir John Pringle published the result of the post-mortem examination of ten cases of fever, and stated that he found "the intestines particularly disposed to mortify."

Ten years afterwards, Rœdererus and Waglerus made numerous examinations of the patients who died in the epidemic at Gottingen, during the occupation of that city by the French. These

authors state that they found intense inflammation of the alimentary canal, and also often gangrene and sphacelus in the tract of the large intestines, and that the mucous follicles of the small intestines were enlarged with similar spots of gangrene.

In 1803, M. Prost, in a work entitled "Le Medicine eclairé par l'Observation et l'Ouverture des Corps," states that he opened the bodies of 150 persons who had died of "fievres ataxiques," and that he always found inflammation of the mucous membrane of the intestines, with or without excoriation. (Tome i. p. 19.)

In 1813 MM. Petit and Serres published their work on "La Fievre Entero-mesenterique." Though these authors viewed the disease as being of a peculiar character, yet no one can read their description of it without coming to the conclusion that it was undoubtedly typhus. They inform us, however, that the pathological phenomena of the affection consisted in inflammatory alterations of the mucous follicles of the ileo-cœcal valve, and of the parts immediately above and below it, together with alterations of the mesenteric glands, corresponding to the inflamed portion of the intestinal canal, which they found to be in a state of greater or less disorganization in every case.

In 1827 Dr. Bright published his admirable work, entitled "Reports of Medical Cases," &c. In the first volume he gives a most graphic description of the post-mortem appearances which he found in the intestines during the progress of fever. I beg to quote at length this admirable sketch, because, in the first place, Dr. Bright entertained no theoretic opinions on the subject—he considered the matter entirely in reference to the therapeutics of the disease; secondly, his account of the lesion exactly corresponds with that given by Louis, with Rokitansky, and also in the cases which I have inspected; and thirdly, Dr. Bright's observations were confined to fever as it occurred in London. He remarks:—

"The appearances which are most marked in the mucous membrane of the intestines are those of increased action, vascularity sometimes occurring in patches of greater or less extent, without any obvious dependence on inflammation of the mucous glands, and occasionally extending, under some form or other, through the whole tract, from the pylorus to the rectum; but this vascularity is more generally connected with inflammation of the mucous glands, which often appear like the small-pox on the second or third day of the eruption, elevated and almost transparent, and covered with minute vessels, which dip into them from the lining membrane of the intestines; they scarcely seem to go into a state of true suppuration, but become distended with a yellow, cheesy matter, and slough off; or sometimes ulceration takes place upon their points externally, without any collection of yellow matter being perceptible. The same process, or nearly so, takes place in the solitary and in the congregate glands (Peyer's); except that in the latter the appearances become much more formidable and the mischief more extensive. The masses or clusters of congregate glands are chiefly placed along that part of the intestine which is farthest from the insertion of the mesentery; and when the parts are irritated from disease, three, four, or five considerable branches of vessels are seen passing on the mucous membrane, from the mesentery on each side, towards the cluster of

congregate glands; these divide and subdivide before they reach the glands, and running in part over the surface of the cluster till their distribution is lost to the eye, enter apparently into the thickened mass of glandular structure beneath. The glands themselves seem first to enlarge, becoming distinctly visible to the eye, and after some time, form a thick, flat mass, of a lighter colour than the surrounding intestines; this sometimes increases to the thickness of a halfcrown piece, and occasionally even spreads on the top, so that the surface overhangs the base nearly the sixth part of an inch. Sometimes a dark-coloured matter like grumous blood is deposited amongst the glands; so that the whole mass, instead of being lighter than the intestine, is of a brown colour, elevated evenly above the surface; but in either case the mucous membrane is at first only raised, and not broken. In a little time, fissures are formed with ulceration on this mass, and ulcers more or less deep occupy the surface of the whole. Where the irritation is little, the ulceration is often mild and merely superficial; but when anything has occurred to irritate the ulcer, it becomes deep and ragged, with an uneven bottom, caused apparently by the projecting remnants of the enlarged glands, or it is filled by a dense slough, stained of a yellow colour by the bile and fæces. As the inflammation subsides, the depth of the ulcer diminishes; and the greater part of the glandular structure being apparently removed by ulceration and sloughing, the edges fall down, and the ulcer becomes shallow, sometimes leaving the muscular fibres nicely displayed, or often exposing the internal surface of the peritoneum for the space of a quarter or half an inch square. This excavation is filled up by a process of granulation, which may be seen very beautifully by suspending the intestine cut open before a lamp or bright sunshine, and examining it with a common lens; the granulations are then seen, sometimes arising in broken lines in the direction of the mascular fibres, at other times arranged in radiated lines round a central point; and when the whole is healed a scar remains visible for some time, not unlike a superficial scar from small-pox, and generally interspersed with slight elevations of a greyish colour. This scar appears to be covered with a true mucous membrane, the surface being quite continuous with the membrane lining the rest of the canal; indeed, when inspecting the ulcer in the process of healing, we perceive the vessels of the mucous membrane running over the surface to be repaired. The whole process of the ulceration and the healing is quite analogous to those painful and irritating sores which frequently take place within the lips, or on the mucous membrane lining the cheeks, where obstruction in the follicles, enlargement, ulceration, sloughing, and perfect repair, are all most distinctly and easily traced.

"The space occupied by the ulcers in the intestines is usually about two feet at the lower end of the ileum, and frequently the valve of the colon on the side next the ileum is the part where the disease is furthest advanced.

"A few ulcers are likewise often found in the cœcum, and some are occasionally dispersed along the colon, depending on a process very similar to that which I have described as taking place in the small intestine; but the glandular distribution being in this part more simple, the ulcers usually commence by small rounded elevations, and not in spreading masses.

"The peritoneal covering of the intestines at the back of the ulcers is generally discoloured and vascular, but seldom appears actually inflamed; and the distribution of the vessels is somewhat different from that of the vessels which may be seen through the peritoneum on the mucous membrane, and is perhaps chiefly derived from vessels belonging to the muscular structure; for instead of forming numerous branches, they arrange themselves in parallel lines, with vessels crossing nearly at right angles. Occasionally, however, the mischief is not confined to the mucous, or even the muscular covering, but the peritoneum becomes decidedly inflamed; in which case the symptoms are always aggravated, and the tenderness of the abdomen is much more marked, and after death a a sero-purulent effusion is found, and shreds of coagulable matter glue the convolutions together. In a few rare cases the ulceration finds its way completely through the peritoneum, and a portion of the contents of the intestines actually

passes into the cavity of the abdomen, when general inflammation is excited, and death follows. With these appearances of the intestines we usually find some considerable derangement in the structure of the mesenteric glands; they are almost always enlarged and vascular, often exceeding the size of a pigeon's egg, and appearing quite covered with turgid vessels. They are in general most affected immediately opposite to the ulcers of the intestines, and occasionally go into a state of complete suppuration, so that I have seen them apparently on the point of discharging themselves through the peritoneum into the cavity of the abdomen; but I believe that not unfrequently the pus, even after it has been formed, is absorbed, and quietly subsides."—P. 180, &c.

The excellent descriptions given subsequently by Louis, Chomel Rokitansky, &c. &c., do not surpass either in accuracy or vividness the one now quoted. In proof of this, I beg to transcribe the most recent account of these intestinal lesions given by Dr. Wilks in his "Pathological Anatomy."

"In typhoid fever," he tells us that "the Peyerian and solitary glands are affected in a peculiar manner, by a material deposited within them, and which subsequently is absorbed or sloughs out. It has generally reached its greatest amount about the tenth day, at which time the most favourable opportunity is presented for observing its nature, although it is not often that death occurs at this time. The deposit is soft, and of a brown colour, and is met with in Peyer's patches in the lower part of the ileum, and in the solitary glands of the same part, as well as of the cœcum, and sometimes in ascending colon. Since Peyer's glands are larger and more numerous at the very end of the ileum, and are smaller as we proceed upward, so necessarily is the deposit greater at the termination; here there are often three of these patches near together, and these being occupied by the adventitious matter, the intestine is almost closed, as you may see in this example. Not only is there more disease below, but it has further advanced, showing that the deposit first occurred in the larger patches, at the inferior portion, and then proceeded upwards; we thus find, for a distance of about two feet up the ileum, smaller masses of the same deposit or exudation. If this be examined by the microscope you see merely cells, some of them with three or four nuclei; but, as I before said, I do not know that these are characteristic; at least I do not think they could be recognised as typhoid. The usual time for examining the disease when death has occurred, is after the third week, when it has arrived at its height. In these cases, although we may still find the deposit in the upper glands, it is almost removed from the lower, where a roughened or ulcerated spot is left. The ulcer, if there be one, has sufficient of the material about it to render it capable of our recognition as of the typhoid character, and assistance is also afforded by the simultaneous affection of the mesenteric glands. It is still a question what becomes of this adventitious material—whether it is absorbed, or sloughs away, leaving an ulceration. We are so in the habit of finding ulceration in fatal cases of fever, and speaking of typhoid ulceration, that we have generally looked upon it as the natural process; but this, I think, is not proved. That ulceration often deeply affects the coats and leads to perforation, we know constantly occurs, but it does not follow that ulcers are always left in the bowels in cases of recovery; the material is got rid of partly by sloughing, and possibly partly by absorption, for we may actually see loose, detached pieces; and where, indeed, a mass has diminished in size without any detachment of substance, we suppose absorption has occurred, as we know must have been the case in the mesenteric glands. That a great change must be apparent in the mucous surface after fever, is apparent from the deposition and absorption of the material. It is about the tenth or twelfth day that you would see to perfection the glands thus affected—Peyer's patches presenting a large, raised, oval mass, projecting into the intestines, and amongst these round masses like peas, corresponding to the solitary

glands, which also are generally seen in the commencement of the colon; at this time the mesenteric glands are also found enlarged. At the usual period of death in fever the glands are found covered with a small amount of this material, forming irregular projections, and often the mucous membrane is ulcerated, leaving the muscular coats bare beneath; and sometimes, if death occurred from perforation, a small hole is found at the bottom of the ulcer, through which fæcal matter has passed, producing fatal peritonitis. In some cases this occurs from several minute pin-hole perforations, which have allowed some exudation through them, and thus set up the final inflammation."—P. 298.

These observations fully accord with the descriptions given by Louis,* Bretonneau,† Chomel,‡ Forget,§ Leuret, Gendron,‖ Putégnat,¶ Letanelet, Lombard, Fauconnet, Mayer,** Delarroque,†† Jacquot,‡‡ E. Boudet,§§ Valleix,‖‖ Jenner,¶¶ Stewart,*** Robert Williams,††† Huss, Bartlett, Stokes, Ritchie, and numerous other well-known authorities.

From the immense mass of observations which have been collected by these different writers, it becomes indisputable, 1st. that the abdominal lesion consists in inflammation of the mucous membrane of the small intestine, especially of the lower portion of ileum; 2nd, that the follicles, particularly those known by the name of Peyer's glands, are tumified by the presence, in one class of cases, of nucleated cells and granular matter, and in another of a gelatinous fluid along with these cells and granules; 3rd, that these follicles slough and ulcerate; and 4th, that either cicatrization or progressive ulceration occurs, in the latter case involving not only a large portion of the mucous membrane, but extending to the submucous, muscular, and peritoneal coats, and not unfrequently ending in perforation of the intestinal canal and consequent peritonitis. In a few words, the abdominal lesion in fever may be defined to be inflammation of the mucous membrane of the small intestine, especially that of the ileum and of the corresponding mesenteric glands. I beg to repeat, that this inflammation goes through the various stages characteristic of the inflammatory process, viz.:—

1. Congestion of the submucous vessels, sometimes extending to those of peritoneum.

* Rech. Anat. Path. et Therap., sur la maladie connue sous les noms gastro-enterite, &c. Paris.

† Arch. de Med., t. x. pp. 67, 169.

‡ Lecons sur la Fievre Typhoide.

§ Traité de l'enterite folliculeuse.

‖ Journal de Conn. Med.-Chir. 1834.

¶ Bull. d'l Acad. de Med., t. ii. p. 853; and Gaz. Med., Novemb. 1838, p. 710.

** Bull. de la Soc. de Med. de Besançon, No. 2, 1847.

†† Trait. de la Fievre Typhoide. 1847.

‡‡ Rech. sur la Fievre Typhoide. 1845.

§§ Arch. Gen. de Med., 4e senè, 1846, t. xi. p. 161.

‖‖ Consid. sur la Fievre Typhoide, Arch. Gen. de Med., 3e senè, 1839, t. v. p. 75.

¶¶ On Typhoid and Typhus Fever.

*** Ed. Med. Journal. 1840. ††† El. of Med.

2. Effusion into the follicles of Peyer, and not unfrequently into the submucous tissue.

3. The effusion becomes more or less consolidated, after which increased fullness of the vessels of the submucous and peritoneal tissues ensues.

4. The effusion again undergoes a process of softening.

5. The follicles and softened matter slough, leaving a small ulcerated spot, the bottom of which is formed by the submucous tissue.

6. Cicatrization of the ulcer takes place, leaving a simple depression, which can only be detected by carefully holding the intestines to the light, when the thinness will be detected. A lens is often necessary to enable the observer to see these cicatrices.

7. When cicatrization does not occur, the ulcer extends (sometimes slowly, at others with great rapidity), the submucous coat becomes destroyed, and a deposit takes place round the margin of ulcer, giving it an elevated edge—this destructive process sometimes becomes suspended, and cicatrization takes place, but at others no such reparative process occurs, and the ulcerative action goes on, until not only the muscular coat becomes destroyed, but also the peritoneal covering. Let it be particularly observed, that *this chronic ulcer is not to be regarded as the anatomical lesion of the fever. The primary ulcer and the pathological processes by which it is preceded constitute the anatomical lesion.* This fact is too often overlooked by many writers on the subject. They seem to regard the chronic ulceration as the peculiar anatomical condition, but the slightest reflection must satisfy every one that such views are erroneous. The anatomical lesion, strictly speaking, consists of inflammation and its effects. The chronic ulcer is an abnormal result from these effects—the healthy termination being cicatrization.

It must not be overlooked that even primary ulceration may not necessarily result from the pathological process. The inflammatory action may become suspended in any of its stages.

1. The congestive stage may subside without effusion.

2. The effusion stage may be removed by absorption.

3. After consolidation and the subsequent softening, the contents of the follicles may also become absorbed, and consequently we will have neither sloughing nor ulceration.

Let us next procced to the consideration of the second point, viz.:—

II. The Relation of the Abdominal Lesion to the Fever.—Dr. Bright does not seem to have entered upon this inquiry; he was content to point out the therapeutical indication which the condition of the mucous membrane afforded. He refers to the frequency with which diseases of the brain and lungs occur in fever, but adds that there is decidedly no class of morbid

appearances so frequent, and none more important, than those which involve the structure of the intestines, "for they lead us at once to inquire into the most secure mode of treatment, where we can ascertain the existence of mischief which adds so materially to the risk of life." He offers no opinion regarding the relation which the morbid changes holds to the disease; he simply points to the means by which the lesion may be cured.

Broussais was the first to attract general attention to the subject. He regarded the local intestinal disease as the *cause* of the fever, which he styled a genuine *gastro-enterite.* Though this was a very erroneous conclusion, yet I beg to say that this great man does not deserve the censure which has been so very unsparingly bestowed upon him, and that, sometimes by those who are entirely ignorant of his writings, their knowledge consisting at most either of quotations, or allegations made by the opponents of his doctrines.

Broussais prosecuted the study of morbid anatomy with a devotion and zeal unsurpassed; he attempted, so far as post-mortem examinations enabled him, to connect the anatomical lesions with disordered physiological action. To him physiology and pathological anatomy appeared to form the basis of medical science, both as regards the symptomatology and the therapeutics of disease. It should also be remembered, that the morbid conditions found after death were regarded in his day as the cause of the disease, or, in other words, that morbid anatomy constituted pathology. At that period pathology, in the present acceptation of the term, was unknown. Hence, when Broussais found traces of inflammation of the intestinal mucous membrane invariably present in fever, he was led to the conclusion that the local lesion was the cause of the general disturbance of the system. The morbid condition of the mucous membrane was a *fact;* the inference regarding its relation to the disease was an error. And probably the energy with which he advocated his theory may have had a much greater influence in leading Louis and others to an earnest examination of the subject, than the mere enunciation of the *fact* itself would have secured.

Though we are principally indebted to Louis for the elucidation of the true relation which the disease of the mucous membrane holds to fever, yet other writers, prior to the publication of his admirable treatise on the subject, had alluded to this relation. For example, Dr. Armstrong of London, in his excellent work on the "Morbid Anatomy of the Stomach and Liver," &c. (published a year prior to that of Louis), makes the following remarks:—

"Now if putrid matter be so intermixed with the blood as to give rise to a distinctly-marked fever of a specific sort (typhus), the brain and its membranes, the bronchial lining and that of *the small intestines*, are invariably, as far as my dissections have extended, affected by the appearances of increased determina-

tion or inflammation, when the disorder runs its definite course of about three weeks; and as these appearances are found in the male and the female, in the young and old, whatever may have been their temperaments or habits, so is it reasonable to conclude that the contamination of the blood *operated specifically* on the *forementioned textures.*"—Pp. 9, 10.

From this extract it is evident that the author regarded the local manifestations in fever as the specific effect of the fever poison, and not the cause—an opinion precisely similar to that of Louis. Again, I may remind the reader that in Dr. Armstrong's "Lectures on the Practice of Physic," published in 1834, he speaks with still greater clearness on the subject. At page 273 he observes, "I have never seen a case of typhus fever in which there were not inflammation of the air passages, and ulceration of the intestines." And at page 333 he states that "inflammation of the mucous membrane most frequently has a certain duration; and when it occurs, you cannot cut it short, as you can inflammation of a serous membrane." At page 564 he affirms that "some traces of inflammation are found invariably in the mucous membrane of the small intestines, and especially of the lower portion of the ileum; this portion of the ileum is invariably found inflamed, either with or without ulceration. When the affection has gone on for a fortnight or three weeks, you will almost invariably have inflammation and ulceration there, and the mesenteric glands will be more or less enlarged." It is worthy of notice that the observations of Armstrong and Louis were made at the same period—the one in London and the other at Paris; and it is quite evident that both had arrived at the same conclusion, viz., that the mucous membrane of the small intestines was invariably inflamed, and that the elliptical glands of the lower part of the ileum ulcerated, and that these local lesions were to be regarded as the specific effect of the fever poison.

It is to Louis, however, that we are indebted for having brought the subject fully before the notice of the profession.* He demonstrated that in the various forms of fever which prevailed in Paris, the condition of the mucous membrane of the small intestines which I have described invariably existed; forming, therefore, the anatomical lesion peculiar to the disease.

The observations of Louis were amply confirmed by Audral and Chomel. The former states that he found the lesion present 98 times out of 100 cases.† The latter asserts that he found the local affection almost invariably present. His words are:—"Les lesions anatomiques qui accompagnent constamment ou presque constamment l'affection typhoide occupent les follicules de l'intestin et les ganlions mesenteriques. Ces lesions sont les seules que l'on rencontre dans presque tous les ces; toutes les autres, quel-

* Recherches Anatomiques, Pathologiques et Therapeutiques sur la maladiè connue sur le nom Gastro-Enterite. 1829.

† Clinique Medical, t. iii. p. 462.

que frequentes qu'elles soient, n'approchent pas sous ce rapport de celles des follicules et des ganglions mesenteriques, et sont rangées parmi les lesions accidentelles."* He afterwards informs us that during five years' experience he only found one case in which no disease of the follicles could be detected.†

I need scarcely remark that this author very earnestly contests the doctrine of Broussais; and shows most satisfactorily that the local disease is not primary, but secondary; that it is one of the phenomena resulting from the disease; a phenomenon, however, so constantly present as to lead to the belief that it forms the characteristic lesion of fever.

After pointing out the analogies which exist between the inflammatory condition of the intestinal mucous membrane and the character of the "phlegmasies disséminées," he remarks:—"Si, a cette consideration, fournie par l'analogie nous joignons ces deux autres circonstances précédemment établies, savoir. 1°, qu'il n'y a pas proportion constante entre la gravité des symptomes et celle de la lesion des follicules; 2°, que cette lesion a manqué completement chez des sujets qui avaient offert pendant la vie tous les symptomes de l'affection typhoide, il deviendra plus évident encore que la maladiè typhoide ne consiste pas essentiellement dans l'inflammation des follicules; que cette inflammation n'est qu'un des phenomenes de cette maladie, qu'elle appartient comme la plupart des inflammations disséminées, aux inflammations secondaires; qu'elle peut étre comparée, quant a *sa valeur pathogenique*, non pas meme aux pustule dans le variole, car est il y a toujours proportion entre le nombre des pustules et la gravite de la maladie, mais plutot au bubon dans la peste d'Orient. Mais apres avoir diminué l'importance de l'inflammation folliculeuse dans la fievre typhoide, nous avons besoin de redirè combien est grande de valeur comme lesion caracteristique de la maladie, de repeter que si elle n'est pas constante, dans la rigoureuse acception de ce mot, il est extremement rare qu'elle manque entierement, et qu'il n'existe pas un seul example authentique de cette lesion chez un sujet qui n'aurait pas offert les symptomes de la fievre typhoide."—*Loc. cit.* p. 537.

The views of Louis and Chomel were adopted by the late Dr. Robert Williams of London, not from a perusal of the writings of these authors, but from his own personal observation of fever, as he witnessed it in London. Indeed, he states the doctrine of the specific character of the local disease of the intestines in much more precise and philosophic language than that adopted by either of the French writers. In describing the pathology of the disease he tells us that—

"The typhoid poison having been absorbed and mingled with the blood, and the period of latency passed, it primarily induces certain derangements of the

* Lecons de Clin. Med. t. i., p. 56, recul. par. J. L. Genest. 1834.

† Loc. cit. p. 528.

functions of the great nervous centres, and consequently of the many organs and systems they supply; derangements which constitute the phenomena of fever; as alterations of temperature, changes in the force and frequency of the pulse, disorders of the alimentary canal, with other concomitant affections. After a certain time, however, not yet accurately determined, the typhoid poison, in addition to the febrile phenomena, induces certain local lesions, or alterations of structure in a limited number of organs or tissues of the body. The alterations of structure are, first, inflammation of the mucous membrane of some portion of the intestinal canal, which membrane is the great and primary, if not constant seat of the action of the poison; secondly, inflammation of the membranes of the brain, which, though not constant, are frequent; thirdly, certain cutaneous eruptions, likewise frequent, but not constant; and lastly, inflammation of the substance or bronchial membrane of the lungs, or both, which occurs in a small number of cases only.

"The law that fever always precedes the great specific actions of the typhoid poison is deduced from the fact that there are many instances in which the patient has fallen in typhus, and yet no alteration in the structure of any organ has been traced; it is consonant, also, with the laws of other morbid poisons. . . . . Is the dose of the typhoid poison in excess, and the disease rapidly fatal, we should naturally expect, as in the case of an excessive dose of arsenic or of oxalic acid, that the morbid appearances would be either trifling or altogether wanting; while, supposing the dose to be milder, we should equally expect much more extensive marks of the specific action of the poison. When we observe, also, the poisons of smallpox or of scarlet fever, producing their specific eruptions, sometimes on the cutis, sometimes on the mucous membranes, and not unfrequently on both, we can hardly feel surprised that the poison of typhoid may sometimes attack one of the constituent parts of the same membrane, sometimes another, and occasionally different combinations of those parts. When we know, also, that poisons which act on two organs, as that of hooping cough, will sometimes affect one organ, and sometimes another, we have most abundant authority for believing there is nothing remarkable in the typhoid poison occasionally affecting one portion of the alimentary canal and occasionally another portion. It is admitted that we cannot determine the inexplicable modification the poison may have undergone to produce these various results; but the differences that have been mentioned in no degree disprove the unity of the efficient cause, nor are greater deviations than are common to the laws of poisons generally. Every pathologist, therefore, will be prepared to admit occasional and limited differences in the seat of the disease, as also of the pathological phenomena affecting those seats in typhus fever." *

With regard to the morbid appearances in the alimentary canal, he says that—

"When the typhoid poison sets up its great specific action on the mucous membrane of the alimentary canal, that of the cœcum and ileo-cœcal valve is in a great majority of cases the exclusive seat of the disease; but in a smaller number of cases, the inflammation extends its ravages both upwards and downwards, from a few lines to many inches, from these points as from a centre. In a few instances, however, the colon, or small intestines, or the stomach, is the exclusive seat of the disease, or the poison may involve any combination of these parts. The inflammation thus excited may attack the free surface, or the adherent surface of the mucous membrane; or it may fall on its most common seat of action, the mucous follicles, and attack these parts either separately or conjointly."—P. 46.

If the reader examine the work, he will find that the post-mortem appearances which Dr. Williams describes exactly correspond with

* Elements of Practical Medicine, Vol. i. pp. 42–45. London, 1836.

the observations of Bright, Louis, Chomel, and the other writers to whom I have referred. It will also be found, that the symptoms which Dr. Williams describes as pertaining to the disease during life, are uniform with those pointed out by the French writers as characteristic of the Parisian fever. We find, therefore, that about the year 1840 it was established by the observations of able physicians belonging both to London and Paris, that in the disease which they called fever the principal local lesion existed in the mucous membrane of the alimentary canal, and that the portion most frequently affected was the mucous membrane of the ileum, and its aggregated follicles. It was likewise established, that the true relation between the local disease and the fever poison was one of cause and effect. Not, however, as Broussais taught. On the contrary, instead of the fever arising from the local lesion, this local disease resulted from the cause of the fever, viz., the introduction into the blood of a specific poison. The febrile disturbance and the local manifestation were merely associated conditions arising from the action of the poison on the system—the abdominal lesion consequently having the same relation to fever that the eruptions of scarlatina, rubeola, and variola hold to the respective morbid poisons from which they originate. It was also established that the local intestinal disease, though generally present, *was not invariably so;* cases occasionally were met with in which no abdominal disease could be detected after death, just in the same way that we now and then observe cases of exanthematous disease in which the specific local manifestations are absent. Again, it was shown that the local intestinal disease went through a series of stages—first, congestion; secondly, effusion; thirdly, consolidation; fourthly, softening; fifthly, ulceration; sixthly, cicatrization; seventhly, degeneration. And it was also pointed out that the febrile disturbance preceded the local manifestation of the abdominal lesion. In this way not only an analogy, but a close resemblance was demonstrated to exist between the modus operandi of the typhoid poison and that of the morbid poisons from which the exanthemata originate.

About 1840 a new doctrine was promulgated in this country and America, from whence it extended to France; namely, that in Great Britain we had two forms of fever, perfectly distinct in their symptoms, etiology, and pathology; that one of these forms corresponded exactly with the fever of France, and should be denominated typhoid; but that the other, which was quite peculiar to Great Britain, was never seen in France: on this form the name Typhus was bestowed. It was contended that in this disease the intestinal mucous membrane was rarely, if ever, involved; and that the symptoms during life were so dissimilar to those seen in the fever of France as to justify the conclusion, that the diseases were non-identical as to origin. Among the advocates of this very modern theory, Dr. A. P. Stewart of London

(a distinguished pupil of the medical school of this city); Dr. Bartlett of Philadelphia; and Dr. Jenner of London—hold the most prominent place. It is useless to remark that we find the names of equally able and experienced physicians who maintain that both typhus and typhoid fever originate from the same poison; and that the dissimilarities which are seen can be satisfactorily accounted for on the same principles on which we explain the difference between confluent and distinct variola, or between the different forms of scarlatina, &c. This important point has not improperly been denominated the question of the day. Considering that the cases which I have narrated in the last number of this Journal, have a most important bearing on the point at issue, and as the results of these cases coincide with my experience of fever during a period of nearly twenty-five years, I feel anxious to enter upon the consideration of the subject with the care and deliberation which it demands.* I therefore beg to defer its discussion to the next number of the Journal, in which I also intend to state the results of my experience regarding the treatment of the abdominal lesions.

---

## II.—*Successful Case of Transfusion.* By John Thomson, Surgeon, Kilmarnock.

On the 27th November, 1859, I was called to visit Mrs. M——, aged 31, of rather delicate constitution, and the mother of four children. After the birth of her third child, flooding took place to such an alarming extent as to place her in circumstances of the most imminent peril. This was accompanied with almost constant retching and vomiting for a period of about three weeks. During the whole of this time life was sustained by enemata of beef tea, with the addition of a fixed amount either of wine or brandy. Convalescence at last was established, and she made a slow but progressive recovery.

At my first visit, on the present occasion, I learned that the menses had been absent for a period of about four months. Her breasts at first began to swell, and she experienced the usual concomitants of pregnancy. At the end of two months she sustained a slight injury. Very soon after this, the swelling and fullness of the breasts gradually subsided, while the other symptoms which characterize that condition ceased to be felt. Being anxious and uncertain as to what her situation really was, I had little hesitation in expressing my conviction that she really was

* The motive by which I have been actuated in quoting so much from the various authors as I have done in this number, is to place the subject before the reader in a manner that will enable me to enter on its discussion in the fullest and most satisfactory manner.

see that it is turned in such a direction that its image produced by the reflector has its greatest diameter at right angles to the axis of the eye observed; the area illuminated will then have the greatest diameter attainable under the circumstances.*

Some eyes, without really having any turbidity of the humours, are so difficult to illuminate, that one might readily suspect it; and this is particularly the case with regard to hyperpresbyopic ones, of which those deprived of the lens are extreme cases. Such eyes are the last in which one would expect, *a priori*, to meet with any difficulty of the sort; and, if the source of light and reflector were both very large, we should probably find none. The difficulty is usually met with when flame, reflector, and eye are very near one another, and the foci of the refracted pencils are far behind the retina. In this case, the circles of dispersion on the retina extend far beyond the points where the axes of the extreme pencils meet it, and the illumination is consequently faint, except in a very small space near the centre. It will often be found that by removing the flame a little further from the reflector when a concave one is used, the intensity of the illumination will be increased to the required degree. The pupils of such eyes, and indeed the eyes themselves, are generally small, if the defect is an original one.

In conclusion, it may be remarked, that in the cases represented in figs. 1, 2, and 3, it is not necessary to illuminate so large an area as in that represented in fig. 4, because the area seen by the observer is smaller in the former ones.

*(To be continued.)*

---

## III.—*Contributions to the Pathology and Therapeutics of Typhus Fever.* By Joseph Bell, M.D., one of the Physicians and Clinical Lecturers, Glasgow Royal Infirmary, &c.

### No. III.

### ON THE IDENTITY OF THE TYPHOID AND TYPHUS FORMS OF FEVER.

The investigation of this point involves three considerations:—1st, Similarity of the symptoms; 2nd, Identity of the pathological lesions; 3rd, Community of origin.

1. *Symptoms.*—It is at once conceded that the symptoms *are not precisely similar* in the two affections; but, at the same time, I contend that the points of dissimilarity are neither so constant, so well defined, nor so important as to authorize the conclusion that the two forms of disease arise from different specific causes.

* See figs. 1, 2, 3, and 4.

Again, the points of resemblance between the symptoms preponderate immensely over the differences which exist; or, in other words, the symptoms which are identical are much more numerous and important than those which are non-identical.

In proof of this, I beg to quote an analysis of the symptoms of the two diseases from Dr. Bartlett, one of the earliest and ablest advocates of the non-identity theory. I prefer his account of the matter to any original one of my own, as it will prevent all cavil as to partiality on my part.

I beg to place his description of the two diseases opposite to each other, so that the reader may trace at a glance the points of resemblance and those of difference :—

| TYPHOID FEVER. | TYPHUS FEVER. |
|---|---|
| Typhoid fever is an acute affection occurring most frequently between the ages of 15 and 30 years, sufficiently often previous to the former period, but rarely after the fortieth year of life, attacking at least in cities and amongst adults in a large majority of instances, persons who are recent residents; occasionally, and under certain conditions, the nature of which is unknown, capable of transmission from one individual to another, rarely occurring twice in the same person, more common in certain countries than in others, but not confined to any geographical localities or regions, prevailing at all seasons of the year and in all climates. Sometimes sudden, and sometimes gradual in its access, attended at its commencement with chills or rigors not commonly very severe, and usually repeated at uncertain periods for the first few days. | Typhus fever is an acute affection occurring *at all ages*, attacking at least in cities, somewhat more frequently persons who are recent, than those who are old or permanent residents. Often transmitted directly from one individual to another; very much more common in the British Islands than elsewhere; also prevailing at times in other countries, generally in the form of circumscribed epidemics; often connected with the crowding of many persons into small, dark, and poorly ventilated apartments, amidst filth and destitution. Sometimes sudden and sometimes gradual in its access, attended at its commencement with chills, usually slight, and in many instances repeated. |
| Then, with more or less feverish heat of the skin, generally with increased quickness of the pulse, somewhat accelerated respiration, slight dry cough, an extensive sonorous or sibilent rhonchus, with pain in head, back, limbs, loss of the vigour, and in some grave cases, perversion of the faculties of the mind, dull expression of the countenance, more or less somnolence or watchfulness, giddiness, or dizziness, ringing, roaring, and buzzing in the ears, occasional epistaxis, great loss of muscular strength; in grave cases with spasmodic twitchings of the muscles, especially those of the forearms and hands, with entire loss of appetite, and with thirst; sometimes with nearly a natural appearance of the tongue, and | Then, with morbid heat of the skin, in many cases very intense and pungent, with increased quickness, with softness and feebleness of the pulse, with accelerated respiration, and in many cases with the physical signs of bronchitis and pulmonary congestion, with pains in head, back, and limbs, dulness or perversion of the powers of the mind, drowsiness or stupor, dizziness, deafness, and ringing or buzzing in the ears; morbid sensibility of the skin and muscles on pressure; extreme prostration of muscular strength, spasmodic twitchings of certain muscles; dull and stupid expression of the countenance, fuliginous flush of the face, suffusion of the eyes, with loss of appetite, and thirst; sometimes with a |

at others with a red, dark, dry, glutenous, cracked, trembling state of this organ.

Sordes upon the teeth and gums, occasional nausea and vomiting, frequent diarrhœa, abdominal pains, and tenderness, these latter not unfrequently most marked in the right iliac region, dullness on percussion over the spleen, meteoric distension or rigidity of the abdomen. The skin, particularly of the front part of the body, being usually the seat in the course of the second and third weeks of the disease, of a peculiar eruption, not commonly abundant, consisting of small circular or oval spots, of a bright red colour, slightly elevated above the surrounding surface, and readily disappearing under pressure.

The blood when drawn from the body having its proportion of fibrine diminished, in a degree closely corresponding to the gravity of the affection.

Which symptoms differ very widely in their duration, in their march, in their severity, and in their combinations in different cases; no one of which is invariably met with, and several of which are frequently wanting, but enough of which are almost always present to characterize the disease. Which symptoms, furthermore, may either gradually diminish in severity, and finally disappear, between the twelfth or the thirtieth day of the disease; or may increase in severity, and terminate in death between the seventh and fortieth day from their access.

The bodies of patients exhibiting, on examination after death, in only a certain proportion of cases, various pathological changes in the brain, head, lungs, stomach, and liver; but in most cases enlargement or softening, or both, of the spleen; and in all cases thickening, or redness, or a morbid transformation or ulceration in all these changes of the elliptical plates of the ilium, with enlargement, redness, and softening of the mesenteric glands, corresponding in their position to the altered intestinal follicles. Which disease, thus characterized and defined, differs essentially

slightly altered tongue; but in grave cases with a dry, red, brown, or black, and fissured state of this organ.

Sordes upon the teeth and gums, occasional nausea and vomiting, frequently with a constipated state of the bowels. Epigastric and abdominal pain and tenderness. The skin of the body and extremities being generally 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 and third week, consisting of small spots, generally somewhat obscurely defined; not unfrequently grouped and confluent, of a dusky red colour, not elevated above the surrounding surface, and disappearing only imperfectly, or not at all, on pressure.

The body of the patient, in grave cases, giving out a pungent offensive and ammoniacal odour.

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 stupors, the suffusion of the eyes, the fuliginous skin, and the dusky cutaneous eruptions. Which symptoms may either gradually diminish in severity, and finally disappear between the seventh and thirtieth day; or may increase in severity, and terminate in death between the third and twentieth day from the access; the liability to a fatal termination being much less in early than late in life.

The bodies of patients, on examination after death, exhibiting no constant pathological changes of any of the organs, but in a considerable proportion of cases engorgement of the vessels of the brain, with moderate subarachnoid, serous effusion, engorgement of the posterior portion of the lungs, redness of the mucous membrane of the bronchii, softening or mamelonation of the mucous membrane of the stomach. The blood being generally of a dark colour, often fluid, soft, and non-fibrinous, and the body in many cases running rapidly into decomposition. Which

from all others in its causes, in its symptoms, and in its lesions.*

disease, thus characterized and defined, constitutes a peculiar individual affection, differing essentially from all others, although related by many analogies to typhoid fever.†

The slightest glance at this description will satisfy the reader that the two diseases have been described, at least by this author, in nearly identical terms. The difficulty experienced in perusing the quotation is to find out any point of dissimilarity. Any difference which exists depends more on the mere change in the terms used by the author in his description, than in any dissimilarity between the symptoms described. It is obvious that on the most numerous and most important points there is a perfect similarity, whilst the differences are exceedingly few and of little value.

The dissimilarities which do exist in the symptoms are merely differences in degree, precisely similar to those which we perceive between either distinct and confluent smallpox, or simple and malignant scarlatina, or mild and severe rubeola. Indeed, the dissimilarities which we witness between the different varieties of these exanthematous diseases are much greater, much better defined, and more characteristic, than those which exist between the typhoid and typhus forms of fever. Let any one contrast the symptoms of a mild case of distinct with those of a severe one of confluent smallpox, and he must admit that the differences are much more decided, both in the general and local symptoms, than those which take place between typhoid and typhus fever. The same remarks will apply to the other forms of epidemic disease.

Perhaps the greatest difference between the symptoms of the typhus and typhoid forms may be said to exist in the greater amount of depression of the general system in the former, than in the latter, and also in the respective eruptions. But both these instances are only differences in degree, not in nature. They are to be explained on the same principle by which we account for the differences that are perceived between the similar phenomena in cases of smallpox and other epidemic maladies; namely, that these dissimilarities arise in consequence of differences of constitutional vigour, or the amount or virulence of the poisonous agency from which the diseases arise. Dr. Bartlett has, indeed, supplied the true explanation, when he states "that the blood when drawn from the body has its proportion of fibrine diminished in a degree closely corresponding to the gravity of the affection; and that in typhus, "it is of a dark colour, and non-fibrinous." This fact was pointed out long ago by Andral, and affords a most satisfactory explanation of all the dissimilarities which exist in the symptoms. For example, when the blood is not much impaired, the eruption will

* Bartlett on Typhoid and Typhus Fever, p. 180. † Ibid, p. 331.

chiefly consist of congested papillæ, or at most of the effusion of a little liquor sanguinis, which may have its fibrine consolidated, forming a little conical spot. But when the blood has been nearly defibrinated the eruption will be darker, softer, and more difficult to efface, as fluid blood has been effused. I need not pursue the subject further, because on the slightest reflection it must become obvious that constitutional causes, combined with differences in the amount or virulence of the exciting poison, must influence the symptoms. These facts afford a most satisfactory explanation of the differences which are found to exist between the two forms of fever. So long as the advocates of the non-identity theory accept of this principle as explanatory of the dissimilarities seen in epidemic diseases, they are bound to show how it will not apply to typhus and typhoid fever. This they have never attempted. Again, they ignore one of the best-established principles in medicine, namely, that poisonous and medicinal substances even in the same doses produce dissimilarities in their effects on different patients; and that differences in the amount of the dose is followed by different effects in the same person. These are medical axioms—indeed, they may be denominated self-evident propositions, and are in daily use to explain the differences which we perceive to exist in the phenomena of disease and the results of remedies. Strange to say, the advocates of the non-identity theory keep these facts entirely out of sight in the question at issue, and rest their conclusions on an entirely opposite foundation, namely, that dissimilarity in symptoms proves a difference in the nature of the exciting cause.

Again, let us refer to syphilis—how many different phenomena have we resulting from this poison? In one patient ulcerated tonsils; in another lepra; in a third impetigo; in a fourth iritis; in a fifth nodes; in a sixth rupia; in a seventh psoriasis; in an eighth lupus, and so on. Surely these dissimilarities are much more striking than those seen in fever, and yet no one doubts of the identity of their origin. Examine the effects of opium or alcohol on different persons in the same or in different doses, and we have the most startling dissimilarities in the phenomena produced. It is, therefore, a most obvious fallacy to argue that a mere dissimilarity in the symptoms of diseases proves the non-identity of the cause. In the question at issue, the differences are so slight (indeed, often so much so that none can be defined by the most acute and careful observer), that it is almost incomprehensible how any person possessed of the most ordinary intelligence, could for a moment entertain the doctrine of the non-identity of their origin. This becomes still more extraordinary when we reflect on the fact that some of the symptoms which are said to be so dissimilar often become convertible. Thus, for example, the eruption of scattered rose-spots occasionally becomes mulberry in colour and extensively diffused. This I have

often witnessed. I have seen a patient labouring under a well-marked attack of typhoid, after a severe epistaxis have the rose-coloured elevated spots converted into dark-coloured non-elevated maculæ, at the same time a large additional number making their appearance, so that in the course of a few hours a case of undoubted typhoid eruption became converted into an unmistakable one of typhus. I have seen the same circumstances occur when the patient's strength became reduced from other causes, such as a severe purge, much undue exertion, &c. Dr. Kennedy of Dublin has lately drawn attention to this circumstance, and it affords me much pleasure to corroborate his observations. The theory of the co-existence of the two poisons, so ingeniously adduced by Dr. Murchison of London, will not explain away this important fact.

We therefore find, in the first place, that the differences which exist in the symptoms of the two forms of fever are merely those of degree. 2nd, That these dissimilarities are not uniform. 3rd, That the symptoms most dissimilar are convertible into each other. The inference is consequently inevitable, that such differences do not authorize the conclusion that the two forms of disease are non-identical in their nature. The differences which exist denote nothing beyond mere varieties in the manifestation of the effects of the exciting cause—varieties arising from peculiarities of constitution and other concomitant circumstances.

2. *Identity of Pathological Lesions:* In other words, Are the typhus and typhoid forms of fever identical in the pathological lesions of the small intestines?—The five cases which I have narrated justify an answer to this question in the affirmative. No one who saw these cases, or who reads the faithful account of their history, that has been recorded by the clinical clerks, can doubt but that the cases would have been classified during life by any of the advocates of the non-identity doctrine, as instances of undoubted typhus, whilst, on the other hand, it must be equally clear that, if the post-mortem examinations had only been witnessed by the same authors, they would have denominated the cases typhoid. The very decided character of the typhus symptoms during life, and the very unquestionable disease of Peyer's glands found at the inspections, completely set aside all cavilling as to error of diagnosis, and dispel every pretence for objections based on the supposition that the cases were typhoid in their character during life. It may be urged, however, that the evidence yielded by so small a number of cases is not entitled to much, if to any, consideration. To such objectors I beg to state, that these five cases may be taken as affording a very correct illustration of my experience of fever during a period of nearly twenty-five years.

I commenced the study of fever in the wards of the Glasgow

Royal Infirmary in the year 1835. During the winter of that year, and also the following one of 1836, I devoted as much attention as I possibly could to the cases in the wards, and witnessed numerous inspections after death. The result of my observations impressed me with the conviction, that disease of the mucous membrane and of its follicles was the invariable concomitant of fever. I had the good fortune to be a pupil of the late Dr. Hannay, who advocated with much force and eloquence the views of Broussais; this circumstance led me to bestow especial attention to the disease at the time, particularly as regarded the morbid appearances. Though I could not concur in the views which Broussais held regarding the relation existing between the local lesion and the disease, yet I could not overlook the fact, of the nearly uniform presence of the local manifestation.

In the summer of 1837 I settled in the neighbouring village of Barrhead, and during the seven years which I resided there, I was seldom without fever cases under my care. Indeed, the disease was often epidemic. I have seen fever in the over-crowded houses of the worst parts of the village, in the cottages attached to the various factories and coal mines, in the farm-house, in the cottar's hut, on the hillside, and in the glen, and from the most careful observation of the disease during life under all these varying circumstances, and from frequent post-mortem examinations, the same conclusion was forced upon me, viz., that disease of the mucous membrane was, to a greater or less extent, the constant attendant of the disease in all its forms, and that all the modifications which were exhibited during the progress of the disease arose from modifying influences of the age and constitutional powers of the patient, combined with the amount of exposure to the poison. Since I removed to Glasgow I have had tolerably extensive opportunities of studying fever, both in private and in hospital practice—1*st.* The great epidemic of 1846–47. The result of my observations during this epidemic was the same as before. 2*nd.* In the years 1852–53 I held the appointment of physician to the fever department of the Infirmary of this city. During 1852, the cases were very numerous, above one hundred patients being always under observation. In 1853 the disease was not so prevalent, the average number being considerably less. 3*rd.* Since my reappointment in 1858, comparatively few cases have been admitted. But close observation during life, and careful examination after death during these latter periods have led to the very same conclusion that had been previously forced on my mind, viz., that the two forms of fever are identical as to local manifestation on the mucous membrane of the intestinal canal.

I have now examined the bodies of *fifty-one patients* who have died from fever—ten of the typhoid, and forty-one of the typhus form—with the following results:—

1. In five who died before the 8th day, more or less congestion

or inflammation of the mucous membrane of the small intestines, and deposit in the aggregated follicles were detected.

2. In eighteen who died after the 8th, but before the 15th day, inflammation of the mucous membrane, softening and ulceration of the aggregated follicles, and enlargement of mesenteric glands were observed.

3. In twelve who died after the 15th, but before the 28th day, there was seen extensive chronic ulceration of the mucous coat—in one of these, perforation and fatal peritonitis.

4. In six who died after the 15th, but before the 28th day, death arising from cerebral and pulmonary disease, complete cicatrization had taken place in four instances, and nearly so in the other two; a few small ulcerations still remained.

5. In six who died after the 28th day, extensive thick-edged ulcers existed in the ileum of all the cases, also in the cœcum of two, and in one case the colon was extensively ulcerated.

6. In four who died after the 28th day, complete cicatrization was detected; two of these patients died from cerebral disease, one from extensive bad sores and cellulitis, and another sunk from erysipelas.

Twelve of these inspections were made whilst I resided in the country; six among private patients during the epidemic of 1846–47; and twenty-one were made in the Clyde Street Fever Hospital, under the charge of the late Mr. Jas. Thomson; six were made during the years 1852–53, at the Royal Infirmary; one last year, in private (at Partick); the remaining five are those narrated in the number of this Journal for last January.

I beg to remark, that in all the cases, placed under the 2nd, 3rd, 4th, and 5th heads, cicatrization of numerous follicles was detected. In many it was evident that only some of the glands had degenerated into chronic ulcers. I have never examined the intestines of a fever patient who died from the effects of intestinal ulceration, without observing numerous cicatrices of primary ulcers. This fact has been altogether overlooked by the advocates of the non-identity theory; I beg most respectfully to direct their attention to the matter in their future inspections.

Taking into consideration the results of my own observation of fever, I am forced to the conclusion that disease of the mucous membrane of the small intestines constitutes the anatomical lesion in typhus as well as in typhoid fever, consequently the two diseases are identical in this essential pathological lesion.

I have read very carefully the works of Louis, Chomel, and of numerous other French writers on fever, and I have been unable to discover a single important point in which the disease which they describe differs from the fever that I have witnessed. Be it observed, that neither Louis nor Chomel contend that the fever of France is different from the fever of this country. On the

contrary, the latter undoubtedly regards the fevers of London, Dublin, Edinburgh, and Glasgow, as narrated by Tweedie, Elliotson, Christison, Marsh, Stokes, and Miller, as identical with the disease which he saw in Paris. In discussing the causes of fever in reference to contagion, he compares the fevers described by the above named writers with those which prevailed in France, and dwells upon the similarity of their etiology, symptomatology, and pathology; and besides this he always designates the British fever by the term "typhoide," "l'affection typhoide."

He also institutes a comparison between the epidemic typhus, or camp fever, and the fever of Paris. He observes—

"Si nous comparons ces deux maladies, et d'après nos souvenirs et d'après la description qu'en a donné Hildenbrand, et dont nous avons été à même en 1814, de constater l'exactitude, nous retrouvons *les mêmes symptoms dans les deux affections;* . . . chez la plupart des sujets, la prostration et la stupeur apparaissent dés le principe, et non pas seulement comme dans les autres affections, après que la maladie a duré longtemps, et a profondement debilité l'organism. Les autres symptoms, tels que le meteorisme, la diarrhée, l'affaiblissement notable des sens, la disposition aux escarres et hemorrhagies sont communs aux deux maladies. La marche est la même dans les deux maladies. . . . . L'une des differences, peu nombreuses, que nous avons observées entre ces deux affections, consiste dans la durée, qui est plus longue dans l'affection typhoide que dans le typhus. . . . . Une autre difference consiste dans la frequence avec laquelle on observe dans le typhus les veritables petechies, ou tachés pourprees, qui comparativement sont rares dans la maladie typhoide.

"Quant à l'exantheme cutane, ou eruption typhoide, il offre les mêmes caractères dans les deux affections; les seules differences sont dans le nombre des taches, et dans l'époque de leur apparition. Au lieu d'être bornées, comme elles le sont le plus frequemment dans la fièvre typhoide, à l'abdomen et à la poitrine, les taches lenticulaires, dans le typhus couvrent, et en plus grand nombre, presque toute la surface du corps. Dans ce dernier l'eruption se developpe ordinairement vers le quatrième jour de la maladie; dans la fièvre typhoide, elle apparait seulement vers le huitième jour, et quelquefois beaucoup plus tard. . . . . La seule difference qu'admettent Hildenbrand et Pringle entre le typhus et la plupart des autres fièvres que nous avons rapportées à la maladie typhoide, c'est que la gravité de la maladie est plus grande dans le typhus, sa marche plus rapide, les phénomènes adynamiques plus prononcées, et l'eruption plus generale; mais ces differences ne suffisent pas pour faire rejeter l'identité de la maladie, car elles peuvent dependre des circonstances plus ou moins fâcheuses dans lesquelles elle se propage. Ces differences peuvent plutôt indiquer des degrés d'intensité divers que des maladies entièrement distinctes."

He next refers to the different accounts which have been published recently regarding the post-mortem appearances in the two diseases, and sums up by saying—

"Ces documens, les plus recens que nous ayons sur cet object, sont trop contradictoires pour que l'on puisse avoir une idée bien arretée sur la nature des lesions de l'intestin dans le typhus. Aussi malgré tous les travaux faits sur l'affection typhoide depuis quelques années, son identité avec le typhus bien que probable," &c.—Pp. 335–38.

So much for the opinion of Chomel. I need not remind the reader that this author has divided fever into five varieties:—

1. Fièvre typhoide inflammatoire.
2. " " bilieuse.
3. " " muqueuse.
4. " " ataxique.
5. " " adynamique.

Let any person unfettered by theory, carefully compare the symptoms which Chomel describes as peculiar to these varieties with the symptoms of the two forms of fever designated typhoid and typhus, and I feel convinced he will arrive at the conclusion that, on the one hand, the symptoms of the first three varieties are similar to those seen in typhoid, and that, on the other, the description of the last two varieties completely coincides with those of the typhus form. It is of great moment to keep in mind that Dr. Williams and many other writers have divided fever into two forms—typhus mitior and typhus gravior; the former corresponding to the first three varieties of Chomel, and the latter with his ataxique and adynamique. It is also worthy of remark that Dr. Williams employs the terms typhoid and typhus synonymously, and it is evident from his work that he was quite conversant with the writings of Louis and Chomel, and it is equally obvious that he regarded the disease which they described as identical in cause, symptoms, and pathology with the disease he witnessed in London—the result of the post-mortem examination in his cases corresponding in every particular with the anatomical lesions found by the French writers in their experience.

We have seen that the result of Dr. Bright's dissections of fever cases exactly coincided also with that of the French physicians. The same remarks will apply to the experience of Dr. Armstrong. I hold that these unquestionable facts furnish the very strongest argument in favour of the identity of the fevers of London and Paris.

It may be objected, in the first place, that Williams may have been influenced in his views by the works of Louis and Chomel, but this objection will not apply either to Bright or Armstrong, as they both had recorded their observations prior to the publication of the opinion of Louis. With regard to Dr. Williams, it is merely necessary to point to the circumstance that, so far as the question at issue is concerned, it is not his opinions that are of importance, but the *facts* which he records regarding the symptoms and anatomical lesions which he witnessed. These are perfectly identical in every particular with those narrated by Louis. But it may be objected, in the second place, that Drs. Williams, Bright, and Armstrong confounded typhus and typhoid cases together—that they were not aware of the distinctions which have been more recently expiscated by Stewart, Jenner, and others. To this objection it is a sufficient reply, to remark

that the description which Bright, Armstrong, and Williams give of the symptoms of the disease, perfectly accords with the disease described by Louis as typhoid. A reference to the cases narrated by these authors will satisfactorily establish the correctness of this statement.

Secondly, all the inspections made by these three distinguished physicians revealed the same anatomical lesions that have been described by Louis. It must, therefore, be admitted that, so far as the post-mortem experience of Bright, Williams, and Armstrong is concerned, all the cases which they inspected were typhoid. Now, it is the very climax of absurdity to suppose that they could possibly have inspected only the typhoid cases, and overlooked those of the typhus form. Hence we perceive the utter worthlessness of the objection urged by some of the most ardent advocates of the non-identity theory, with the view of invalidating the legitimate conclusions deducible from the observations and experience of Bright, Williams, and other writers on the character of the fevers of this country.

Again, it must be conceded on the one hand that Armstrong and Bright describe a disease which would be defined by the advocates of non-identity as typhus, and on the other it cannot be denied but that disease of the mucous membrane was detected on the inspection of the bodies of those who died from the disease. Farther, in the epidemic of typhus described by M. Reveille-Parisse* the glands of Peyer were found inflamed. Forget and Cruveilhier found the same diseased condition constantly present in the inspections which they made during the epidemic of typhus in 1814. Pinel reports the same circumstance as occurring in his experience. Hildenbrand also asserts that he found the glands of Peyer always diseased in typhus. Herzog tells us that in the exanthematous typhus which prevailed in the duchy of Posen in 1829–30, he found inflammatory conditions of the mucous membrane of the intestines present, Peyer's patches being tumefied, livid, and with surrounding congestion. Fleury and Pellicot, in their account of the typhus which prevailed at Toulon in 1832, inform us that the inflammatory condition of the intestinal canal was slight and variable, and that no ulceration of Peyer's glands was detected.

Let us next turn to the experience of physicians to whom the doctrine of non-identity was familiar. I beg to direct attention, in the first place to the experience of the late Dr. Perry of this city. In a paper published in the *Edinburgh Medical and Surgical Journal* for January, 1836, he sums up the results of his careful observation (extending to upwards of four thousand cases and three hundred microscopic inspections) in the form of sixteen propositions; in the last of these he states "that dothinenteritis,

* Bull. de Therap. t. vi. 2 liv.

or enlargement of the mucous follicles of the smaller intestines and enlargement and ulceration of the aggregated glands of the lower third of the ileum, *occur in combination* with contagious typhus, and are to be met with in about *one in six* of those who die *from typhus.*"

I would like very much to know how the advocates of the non-identity theory would attempt to set aside this evidence. I need scarcely remark that Dr. Perry was quite alive to the distinction between the typhus and typhoid forms of the disease—indeed he states in the very proposition from which I have quoted, that dothinenteritis also occurs as a *disease per se*, and proceeds to point out the differences which exist between its symptoms and those of what he styled contagious typhus. Again, it must not be overlooked that Dr. Perry regarded the existence of ulceration as essential to constitute the anatomical lesion; he did not recognize either simple congestion of the submucous vessels, or the elevated follicles as constituting dothinenterite. If these lines attract the notice of any of his pupils, I feel assured that they will bear testimony to the fact that he attached no pathological importance, either to simple congestion or trifling elevations of the follicles, and consequently in all the inspections in which such lesions were seen, the intestinal tract was considered by him as destitute of the anatomical lesion peculiar to typhoid. On several occasions I have taken away portions of such intestines, and found not only the follicles filled with the peculiar typhoid deposit, but often detected small ulcerations. Then (and the fact must not be forgotten), that in the great majority of the deaths from typhus the disease proved fatal before the end of the second week, and hence little or no *ulceration* existed. We could expect to find in such cases only fulness of the submucous vesicles and elevation of the follicles. If we take these facts into consideration it will become obvious that the results of Dr. Perry's observations afford a most decisive proof of the identity of typhus and typhoid fever, so far as the intestinal lesion is concerned. It must not be for a moment supposed, that I intend to ignore the correctness of Dr. Perry's method of observation. On the contrary, I have always regarded him as a most correct observer and acute reasoner. I simply state the facts which are well known to many. Dr. Perry was by no means singular in his idea of the character of the anatomical lesion in fever. Many medical men are still under the impression that ulceration, and ulceration alone, constitutes the pathological condition. Very recently, a medical friend of the highest scientific attainments, on contrasting the result of his experience in the epidemic of 1846–47 with mine, observed that in all inspections of typhus he *never met* with one in which a *loss of substance* could be detected in the follicles of Peyer. It is needless to remark, that such notions of the patho-

logical condition of these glands in fever is quite erroneous, especially when the cases examined had terminated on or before the twelfth day of the disease.

There is another circumstance to be kept in mind, which will exonerate Dr. Perry from any blame in overlooking the minute primary ulceration, especially when this was confined to a few of Peyer's patches. During his time, the hour of inspection was late in the afternoon, often between three and four o'clock—a time at which, during six months of the year, the light was exceedingly bad—and at that period the inspecting-room was by no means well lighted; indeed, unless the ulcerations had been very large, they would have been most readily overlooked. Probably some of my readers may remember a circumstance of this character. One November afternoon, in the year 1836, the body of a fever patient was examined. Dr. Perry was not there, but the gentleman conducting the inspection pronounced to the few students who were present that no ulceration existed. One of these students used the freedom to take away the ileum, and next day the late Dr. Hannay used the intestine to demonstrate to his class the various stages of ulceration which it presented; in truth, it was one of the best examples of the disease I ever witnessed. There is yet another fact which I beg to mention, that in all the inspections which I witnessed at the time, the intestine was slit up along the free side, and not along its mesenteric attachment; consequently, the incision passed completely through the centre of Peyer's patches; and I very often found, on a careful examination of the margins of such intestines, unmistakable ulceration, which had been overlooked in consequence of the margins of the incised intestines becoming reflexed.*

Dr. Stokes of Dublin is the next author to whom I beg to refer. The accuracy of his powers of observation, the extent of his experience, are too well known to permit of any allegations against his ability to discriminate between the symptoms which characterize the two forms of fever. He observes that—

"Many circumstances observable in the epidemic fevers of Ireland, must make us cautious in drawing too strong a distinction between, not only the maculated and the non-maculated cases of fever, but also between typhus—properly so called—and the typhoid disease already specified. It is not to be denied that the characteristic cases of either of these groups of diseases are different; and we have seen that with respect to the condition of the heart, which is the subject more immediately in hand, some remarkable contrasts have been found; yet we cannot help believing that these diseases are but varieties; and that they are results of the same poison or exciting cause acting on individuals in different states; but we need not here enter into lengthened discus-

* I may state that my observations were made at the very period at which Dr. Stewart of London refers in his essay on the Non-identity of Typhus and Typhoid.

sions on this point. Let it suffice to indicate generally the grounds of this opinion; they are as follows:—

"1. That epidemics of fever may occur in which, with well marked petechiæ, the follicular ulcerations of the intestine exist in a large proportion of cases.

"2. That in cases of the best marked maculated typhus we have found occasionally the above anatomical condition, and this at a time when no epidemic disposition to the disease could be supposed to exist.

"3. That we have frequently observed, in cases where a large number of a single family have been successively attacked by contagious fever, that every form of the disease, from the most malignant typhus to the mildest typhoid fever, may be presented by different members of the family.

"Lastly, that a patient who has gone through the typhoid fever in its best marked character may, after an apyrexial period of a few days, be attacked with the most severe form of maculated fever; and conversely, that in certain cases where the first fever was maculated typhus, the symptoms in the second attack or relapse were those of typhoid fever.

"These circumstances, when fairly considered, makes a strong case against the existence in these countries of two essentially different forms of fever; and, if anything was wanting to strengthen this conclusion, it is the great fact that although in the so-called typhoid fevers we are not so often nor so urgently requested to employ stimulating treatment, the general principles of management in both forms of disease are truly the same."

Let me next turn the attention of the reader to the work of Dr. Magnus Huss of Stockholm. This physician, after twelve years' experience of fever in the Seraphim Hospital of Stockholm, visited the principal hospitals on the Continent, America, &c. After giving a very lucid exposition of the points of similarity and dissimilarity which he has observed to exist between the two forms of fever, he states his opinion to be "that typhus and typhoid fever (typhus petechialis et abdominalis), such as they appear in the north, neither can nor ought to be *considered as two distinct* maladies, but only as two varieties of the same morbid process;" and he adds, "I am persuaded such are the opinions of the majority of Swedish physicians. This may consequently be considered as the opinion of the north on the subject." †

Be it observed, that the grounds on which this opinion has been founded are fully given by the author in his introductory chapter. In this chapter the reader will find numerous most important observations bearing on the subject, both as regards the disease in its epidemic and sporadic attacks. But I must refer to the work itself for further information on the subject.

Skoda is the only other author from whom I will at present quote. He thus states the opinion which prevails among the physicians of Germany regarding the identity of typhus and typhoid fevers. "We consider that the disease here is *one*, and ought to be called abdominal typhus; that the typhoid fever of the French is not to be distinguished from what they call typhus; that exanthematic typhus does not constitute a specific affection; and that it is

* Dis. of Heart, &c., p. 450. Dublin, 1854.

† Statis. and Treatment of Typhus and Typhoid Fever, p. 23.

nothing else than the union of their typhus with the accidental appearance of different cutaneous exanthemata." *

The preceding facts, collected from writers of all countries, and from men who wrote both prior and subsequent to the promulgation of the doctrine of non-identity, completely coincide with the result of my experience on the subject, namely, that in typhus fever the *same conditions of Peyer's glands exist as were found in typhoid.*

I beg briefly to repeat the facts of the case. Williams, Armstrong, and Bright, of London, have found ulceration and disease of Peyer's glands in typhus. Parisse, Forget, Cruveilheir, Pinel, Hildenbrand, Herzog, &c., have found the same ulceration in their inspection of patients dying from typhus. Dr. Perry of Glasgow (an advocate of non-identity) found *ulceration* of Peyer's gland in the proportion of about 20 per cent. out of upwards of 300 inspections of *genuine typhus.* Dr. Stokes tells us that he has found disease of Peyer's glands in the best marked cases of genuine maculated typhus. Huss states that both he and his brethren of the North of Europe have found the same morbid condition present in genuine typhus. Skoda tells us that he and other German physicians have found the same occurrence in undoubted typhus. I have placed on record in this journal *five* well observed cases of *typhus*, in which the same morbid lesion existed. I repeat that these *five* cases are fair specimens of many others in which the same conditions were found.†

I hold, therefore, that these undoubted facts, surely justify the conclusion that typhus and typhoid fever are identical as to the pathological conditions of the small intestines; or, in other words, they are identical as to anatomical lesion.

I am at a loss to conceive how any one conversant with the foregoing facts can assert "that disease of Peyer's glands never exists in typhus"—a statement completely inconsistent with fact. It seems to be the usual custom with the advocates of the non-identity theory to take for granted that Peyer's glands are always "diseased in typhoid and never in typhus." They make this assumption the basis of all their investigations, when, in truth, this is the very question in dispute—the subject to be proved.

I certainly was much astonished to find Dr. Tweedie of London adopting this principle in his lectures on Fever in the course of publication in the *Lancet.* In the number of that Journal for 14th of April last, at page 366, he states, as an invari-

* Clin Lect. and *London Med. Times* and *Gazette*, Feb. 21, 1859, p. 175.

† I have quoted the five cases only, because they are recent, and were witnessed during their course and at the inspection by a very large concourse of students, many of the more advanced of whom were not only familiar with the non-identity theory, but were advocates of its correctness, until they witnessed these five instances—the force of which was so striking and convincing as to completely change their views on the subject.

able and settled fact, that "in typhus, with the occasional instances of softening of portions of the mucous membrane of the stomach, the whole tract of the alimentary canal is in a normal state." Again, at page 367, he affirms that in typhus, Peyer's patches and corresponding mesenteric glands are in their natural condition." I repeat that I was greatly astonished to find such an assertion made by Dr. Tweedie, who must be familiar with the observations of Dr. Perry, as he has quoted from this author's papers in his lectures; and Dr. Perry states that he found Peyer's glands diseased in about 20 per cent. of genuine typhus. Dr. Tweedie, in showing that *typhoid* fever existed at a former period in London, alludes to a preparation in the Hunterian museum of disease of *Peyer's glands*, and remarks that Hunter, though he considered the preparation as an example of local disease in typhus, yet was in mistake, as the case must have been one of typhoid. This is an example of the fallacy to which I have referred, viz., taking for granted the very matter which is in dispute.

There is another fact well ascertained in the history of the typhoid form of the disease, which also tends to prove the identity with typhus; namely, the want of any definite relation between the severity of the abdominal lesions found after death and the characteristic symptoms during life.

In my post-mortem experience of the fever, I have found that the local intestinal lesions did not always hold a close relation to the severity of the symptoms during life. In some of the most rapidly fatal cases, the local disease was comparatively slight; in one instance in which death took place after the 28th day, the patient, a girl aged 21 years, had very severe diarrhœa from the effects of which she died; at the inspection only two small ulcers were detected in the ileum, and one in the cœcum. In another case in which death took place from pleuro-pneumonia, extensive ulceration was found to exist in the ileum, yet no diarrhœa existed during life.

This want of relation between the symptoms during life and the disease of the intestinal mucous membrane as found after death, has been pointed out by Louis and Chomel, and admitted recently by Dr. Jenner. I consider that this important fact furnishes a very powerful argument in favour of the identity of typhoid and typhus fever. If it can be shown that differences of a similar nature exist in the lesions of the mucous membrane among patients dying from the purely typhoid form, as are found to exist between instances of typhus and typhoid, then we possess a strong argument in favour of the identity of the two forms of disease.

If the local lesion hold no uniform relation to the symptoms considered as characteristic of either of the two forms; or if in both we have occasionally no local lesion; or if we have the local lesion presenting differences in degree, and undistinguished by

any characteristic symptoms during life—then I contend that it would be most illogical to infer that because, in one form of the disease, the pathological state varies from that of the other, that they are non-identical. The importance of the fact of the non-existence of a relation between the symptoms and local lesions is so very great, in reference to the question at issue, that I beg to refer the reader to the observations of Louis and Chomel on this point. I have only space to give a summary.

In the forty-first case reported by Louis, the patient had no abdominal symptoms until the 23rd day, when *perforation* of the intestine occurred. In the forty-second case, the diarrhœa was slight on the 20th day; "un vingtieme jour diarrhée legere;" but, on the 24th day, perforation occurred. The post-mortem examination detected extensive ulceration of the ileum, with enlargement of the mesenteric glands. The upper part of the ileum was not much affected; but the lower was in a very different condition. He observes:—

"Au dela elles offraient vingt ulcerations irregulières, plus voisines de la forme carrée que de la forme arrondie, abords peu saillants grisâtres, coupés en dedolant, de la largeur de deux lignes, principalement formés par l'epaississement de la tunique sous-muqueuse. Au fond de ces ulcerations se trouvait la tunique musculaire un peu rouge, plus ou moins amincie, d'ailleurs saine. Elle etait detruite, et la tunique peritoneale existait seule, au centre de l'une d'elles. Enfin cette membrane avait disparu là ou etaient les perforations. La plus grande des ulcerations finissait a un pouce de la valvule ileo-cœcal, avait deux pouces de long sur un de large."*

In the forty-third case, though hypogastric pain was the only abdominal symptom noticed during life, yet perforation took place on the 45th day; the inspection of the body discovered extensive ulcerations of Peyer's patches, with enlarged mesenteric glands. In the forty-fourth case, no abdominal symptoms were witnessed, still perforation occurred on the 36th day; on the post-mortem examination most extensive ulceration and mesenteric disease were detected. In the forty-fifth case, the same phenomena are related. In this series of cases, we perceive that during life the general symptoms were slight—those of intestinal lesion almost absent—and yet most extensive disease existed. Louis narrates another series of instances (Obser. 46, 47, 48, 49). In these the general as well as the abdominal symptoms were severe, yet very trifling intestinal disease was found after death. In the forty-sixth case, death took place on the 19th day, and only a few slight ulcers were found in the ileum, yet the patient had diarrhœa, severe abdominal pain, and meteorism. In the forty-seventh case, severe diarrhœa and hemorrhage occurred; but only a few ulcers were found after death. In the forty-eighth case, severe diarrhœa occurred from the 8th till the 13th day; and, on

* Tom. ii. p. 344.

inspection after death, a few of the elliptical patches of the ileum were found, "un peu epaissais, d'un bleu fonce, prés du cœcum." In the forty-ninth case, severe diarrhœa existed with meteorism; after death a few of Peyer's glands were found hardened, "dures gris orange," and slightly ulcerated, "legerement ulcerées."

Again, the same author narrates another series of three fatal cases (Obs. 50, 51, and 52) in which, though diarrhœa and other abdominal symptoms existed, yet after death no intestinal lesion was detected. I again beg the especial attention of the reader to these most important facts.

They authorize us to conclude—1st, that in no individual case, either of typhus or typhoid, can we predicate the existence or non-existence of *ulceration* of the small intestines from the symptoms seen during life. 2nd, That, in order to ascertain the condition of the intestines, a post-mortem examination in all cases is absolutely required. Suppose that the one class of the foregoing cases, in which abdominal symptoms were observed during life, had not been inspected, the inference would have been deduced that severe ulceration existed. Again, suppose that the other class of cases, in which no abdominal symptom was seen during life, had not been examined, and if the patients had presented a dark-coloured eruption of typhus, the inference would have been at once adopted that no ulceration existed.

This is precisely the position in which the five cases that I have published in this Journal for January last, were placed. No marked abdominal symptom existed; but the extensive typhus or mulberry eruption and other symptoms during life would have authorized the advocate of the non-identity theory to have concluded that no abdominal lesion existed. These five cases, with those quoted from Louis, show very forcibly how fallacious and unsound it is to form any opinion as to the state of the intestine from the symptoms; the appeal must be made to a post-mortem examination in every instance.

The cases to which Chomel makes reference in his lectures confirm these observations. He proposes the question—viz., Is there a proportion between the severity of the lesions of the follicles and the severity of the symptoms? He sums up his observations and reasonings on this point as follows:—It is then demonstrated that, in a great number of cases, there is not any proportion between the severity of the malady and that of the anatomical lesions.

As it is obvious that this circumstance has such an important bearing on the theory of the non-identity of fever, I beg the reader to consult Chomel on the subject.

He refers to a class of cases in which the general symptoms were most severe, and accompanied with abdominal symptoms, and yet, on inspection after death, scarcely a trace of disease could be detected either in the mucous membrane or its follicles. He

alludes to another class of cases in which the general symptoms were exceedingly mild, and neither diarrhœa nor any symptoms of abdominal lesion existed; but after death extensive disease of the mucous membrane of the small intestines, embracing congestion, softening, ulceration, and even perforation, was found. He concludes by asserting that it has been demonstrated that, in *a great number* of cases, there exists no relation between the severity of the disease and of its anatomical lesion. His words are—

"Il est donc demontré que dans un grand nombre de cas il n'y a pas proportion entre la gravité de la maladie et celle des lesions anatomiques."—Op. cit.; tom. i. p. 527.

Such facts as these appear to me to obviate the force of the argument deduced by the advocates of non-identity from the comparatively small amount of intestinal lesion which is occasionally found in typhus. Suppose that any of these cases had presented typhus symptoms during life, would not Dr. Jenner have considered the slight lesions which existed as merely accidental, and of too trifling a character to oppose his theory? Assuredly he would, because we find that in many of the cases of genuine typhus which he reports in his essay, he found that intestinal lesions of even greater extent existed; yet he explains this away as a trifling incident, and calls the intestine healthy.

Dr. Jenner admits that there exists no relation between the severity of the fever and the intestinal lesion. In a discussion on this subject at a recent meeting of the Royal Medical and Chirurgical Society, he remarks that "many of the early cases which he published, were designed to show that the fever and the lesion bore no relation in severity to each other."—(*Med. Gazette*, June 2, 1860, p. 561.)

He evidently does not recognize the bearing of this important fact on the identity of the two diseases. I do not mean to say that this fact proves their identity; but I do contend that it sets aside the arguments which are employed to establish their non-identity from any dissimilarity of the intestinal lesions which may exist.

Dr. Jenner, in speaking of the non-relation between the severity of the fever and lesions, justly remarks that "a patient might die of virulent small-pox almost before the eruption appeared; and death might occur from the general disturbance in typhoid fever, with very little intestinal lesion." So it is, I contend, in the typhus form of fever, that patients often die before the development of much intestinal lesion, and without a trace of ulceration. It must be admitted that the general disturbance is much more vehement in typhus than in the typhoid; hence we find death often occurs without much trace of intestinal lesion. But this leads me to the consideration of the question—How is it that in typhus, as a general rule, chronic ulceration seldom results, whilst in the

typhoid form it so frequently occurs? Or, in other words, I come now to the explanation of the differences in the intestinal lesions, which are perceptible in the two forms of fever. I admit that considerable dissimilarities do often exist; but I contend that these are differences in degree, and not in kind.

In my last communication I pointed out that the essential pathological lesion consisted in turgescence of the submucous vessels, chiefly of the ileum, and effusion into the follicles, especially those of Peyer; and that sloughing, ulceration, and cicatrization of these follicles were the series of steps through which the morbid process marched, and that the chronic ulcer (which is erroneously described by the majority of the advocates of the non-identity theory as the lesion peculiar to typhoid) is in reality the degenerated primary ulcer, and not the pathological lesion. This proposition cannot be controverted. Drs. Tweedie and Jenner tacitly acknowledge it, as they admit the correctness of Rokitansky's history of the anatomical lesion. I beg to remark in passing, that Dr. Tweedie leads his readers to infer that Rokitansky's description applies to typhoid fever only, and not to typhus; and in this way Rokitansky is made to appear as a supporter of the non-identity theory. A reference to his work on Pathological Anatomy will show the very reverse. He describes the appearances which he found in all forms of fever, but employs exclusively the term typhus. The essential primary lesion is therefore congestion and effusion into the aggregated follicles of the small intestines. This, I contend, exists both in typhus and typhoid fever, the only difference being that in the former the effusion is softer than in the latter—a fact pointed out by Rokitansky and others. This then is the essential difference between the two forms of the disease—a difference depending on the greater defibrination of the blood in the one than in the other. I have already remarked, that it is not questioned but that the severity of typhoid cases holds an exact ratio to the amount of defibrination, and that in typhus this loss of fibrin is very much greater than in typhoid, consequently the effusion remains softer in the former, and often does not consolidate.

So much for the difference in the primary stages of the pathological condition of the intestinal mucous membrane. I maintain that with this exception the lesion will be found as invariably present in typhus as in typhoid. I am prepared to stake the issue of the whole question upon the result of careful post-mortem examination instituted by any of my readers. If the glands of Peyer be carefully examined, they will be found always more or less affected. But it must be borne in mind that this morbid condition will not be found at every period of the attack. It will seldom be detected before the 8th or 9th day. It should also be recollected that the intestine must be opened carefully, not cut up in the centre as is usually done, but at the

insertion of the mesentery. If the opposite and usual practice be adopted, Peyer's patches will be destroyed by the incision passing through their centre. I feel convinced that if these precautions be adopted, the morbid condition will be found present to a greater or less extent. In patients who die before the 10th day, it is unreasonable to expect to find any amount of ulceration of the follicles: nothing will be detected but turgescence of the vessels. It will be admitted that in very virulent attacks of typhus, when it is epidemic, many patients die on or prior to the 10th day, and in such cases no ulceration can be expected to be found. Even at the 14th day, if the intestine be not carefully opened, very little disease will be found, because the incision will divide Peyer's patches; and as the edges of the cut intestine almost instantly become reflexed, the diseased condition is overlooked. From the 12th to the 15th day in the great majority of cases, however, often very great differences will be found to exist between cases of the typhoid and typhus. In the typhus form the follicles slough about the 12th day, the cicatrization commences, and is often complete in a couple of days; but numerous exceptions are found, especially in the young, and when the disease is sporadic. In many cases of the typhoid form we have sloughing and cicatrization occurring about the same period as in typhus; but in a larger proportion of cases we have, instead of cicatrization, degeneration of the primary ulcer into the chronic form, which goes on extending in every direction. This degenerated ulcer, the character of which I have pointed out in the last number, has been well described by Rokitansky. Around the primary ulcer a quantity of fibrin is effused. This becomes first consolidated, next softens, and then becomes degenerated into pus. A new deposit of fibrin takes place around the margin of the first; this secondary deposit in its turn becomes degenerated and surrounded by a new layer of deposit, until the elevated, irregular-edged, and deep ulcer is formed. Now, it is at once conceded that this ulcer is much more frequently found in typhoid than in typhus cases; but surely it must appear self-evident to every one that mere differences in the effects of any pathological lesion will afford no grounds to justify the doctrine of the non-identity of the lesion. For example, in pleuritis we have an effusion into the pleural cavity: this effusion becomes absorbed in one case, in another a layer of organized fibrin remains, and in a third purulent matter is formed. Now these differences in the secondary effects of the inflammatory action would as fully justify the conclusion that these effects arose from non-identical pathological causes, as the differences found in typhus and typhoid justify the doctrine of their non-identity. These differences in the ultimate course of the intestinal lesion in fever, arise from a variety of causes, the most important of which is the virulence of the attack. In typhus the blood is too much

defibrinated, and its fibrine is too soft to become consolidated. But this is not the case in typhoid, especially after the fourteenth day, when the poison has nearly exhausted itself, the irritation of the primary ulcer leads to congestion, effusion, and so on until the chronic ulcer is formed. Hence the origin of the differences which we find to exist in the course of the two forms of fever; hence why in epidemics we have the abdominal lesion less marked than in sporadic cases; hence also why it seldom happens that the chronic ulcer is found in the weak, debilitated, and old patients, or those who are half-starved, and live in ill-ventilated apartments, whilst in the young, the inhabitants of the country, and in sporadic cases, the chronic ulcer is more usually found.

It has been urged that if typhus and typhoid fever be identical, and if typhus be produced by the more intense dose of the poison, then we should find the pathological lesion more severe. To this objection it is only necessary to point to a few facts. 1st, In the most virulent cases of exanthemata, patients often die before the local lesion becomes developed. 2nd, Typhus patients generally die before the period at which the chronic ulcer is usually formed. 3rd, The objection is founded on the erroneous notion that the *chronic ulcer* constitutes the anatomical lesion.

It has also been argued that if typhus arise from a larger amount of the same poison as that from which typhoid originates, we would expect, in those cases of typhus which die after the 28th day, much more extensive ulceration than we find in typhoid. This objection arises simply from the fact being kept out of view that cicatrization of the primary ulcers may have occurred, this cicatrization having been completely overlooked at the post-mortem inspection. I have seen this mistake committed more than once by those very persons who used the argument. The intestine was slit up through the very centre of Peyer's glands, and the existence of cicatrization never once looked after, the non-appearance of ulceration being considered quite satisfactory.

We therefore perceive that we have abundant evidence of the highest character to authorize the *conclusion* that typhoid and typhus fever are identical as to pathological lesion; and that the differences which take place among the effects of these lesions, arise from the influence of well-known concomitant circumstances.

But it may be asked, how can the experience of Dr. Jenner and others be reconciled with this conclusion? Take Jenner, for example. He informs us that he examined the bodies of 43 patients who died from typhus, and that "recent disease of Peyer's patches was absent in every one of the 43." I think there can exist no difficulty in accounting for Dr. Jenner's experience. In summing up the results of his observations on the condition of the "small intestines and mesenteric glands," he states that "*the presence or absence of lesion of these organs was the ground on which the cases of typhoid and typhus fever here analysed were divided from each*

*other ;* consequently, they *were invariably diseased in the one, and normal in the other.*"*

This plan explains very satisfactorily the result of Dr. Jenner's investigation. How could it be otherwise? If any one proceeds to make an examination upon this principle, he will undoubtedly arrive at conclusions similar to those of Dr. Jenner. I ask the reader to pay particular attention to the passage quoted. Is it not obvious that Dr. Jenner's experience is of little or no value regarding the point in dispute? He takes for granted the very question to be proved, and divides the cases he inspected into typhus and typhoid, on the " ground of the presence or absence" of the intestinal lesion. Or, in other words, he fixes beforehand that he will classify all those cases typhoid in which intestinal lesion may be found, and all those typhus in which no such disease can be detected. " Consequently," as he remarks, the intestines " were invariably diseased in the one, and normal in the other." Thus, it must be obvious to every one that the whole fabric of Dr. Jenner's theory is founded on the well-known sophism of *petitio principii.* He commences by begging the question, and then proceeds by another well-known fallacy—namely, *reasoning in a circle*—to support his false position. I have no wish to throw aside such a large amount of post-mortem experience as Dr. Jenner has furnished. That he should have examined 43 cases of fever, without finding any disease either of the mucous membrane or of the aggregated glands, I confess is sufficient to stagger confidence in the opinions which I have ventured to advocate. Let me, therefore, briefly analyse the information which he has given regarding the condition of the intestines in these 43 cases. I may remark, however, that it is not clearly stated in how many of the cases Dr. Jenner noticed the condition of the *small intestines throughout their whole extent.*

In the first place, he tells us that the colour of the mucous membrane of the jejunum and ileum was healthy in 34 *out of* 39 *cases, of which notes on this point were made.* This would give only the result on this point in 39 cases, not 43. But out of the 34 healthy-coloured cases, he tells us there existed ramiform injection of the large vessels. This reduces the healthy number to 30. Again, he states that in two cases " hæmorrhagic spots existed beneath the mucous membrane." In one of the two, these spots about twenty in number, were scattered over the jejunum. They varied in size from a pin's-head to a line and half in diameter, some of a deep purple colour, and others red ; and all were hard to the touch and distinctly elevated, and vessels containing fluid blood could be traced into them. In the other case, injection of the mucous membrane existed. Some of the large veins were filled with dark blood, and in their vicinity were small spots of ecchymosis. This

* Edinburgh Monthly Journal, vol. x., p. 312. The italics are ours.

reduces the number of healthy cases to 28. He tells us that in another case the jejunum was finely injected, and in another the fine injection was limited to the lower part of ileum. Thus we find, that instead of the colour being healthy in all the 43 cases, he only examined 39 cases, and of these the colour was abnormal in at least one-third.

We come next to the *consistence.* He tells us that this "was normal, *or nearly normal*, in twenty-nine cases." Now, by this *nearly* normal what are we to understand? This very indefinite term involves the whole of his experience in doubt and uncertainty. Again, are we to infer that he examined the whole forty-three cases and found undoubted evidence of disease in the other twelve cases? He mentions six cases in which softening existed, but does not tell us whether or not these cases are included in the twenty-nine. He refers to three other cases of softening in the same indefinite manner. But if these nine cases are included in the twenty-nine, then his normal or nearly normal cases are reduced to twenty instances. Again, we are not informed how many of his cases presented this softened consistence, and how many change of colour, or the number of cases in which these diseased conditions were both present. These are most important points, because if he found congestion in thirteen cases and softening in twenty-one cases, this would leave some nine cases only in which the mucous membrane could be said to be in a healthy condition.

Let us next turn to the state of Peyer's glands. He commences by announcing that, with three exceptions, these organs were perfectly healthy; he then modifies this by explaining, "that is, neither elevated, reddened, softened, nor ulcerated." I beg to remark in passing, that the absence of these three circumstances does not prove the absence of disease. The question is, did they contain typhus deposit or not? or was all trace of cicatrization absent? He further states, that in fourteen cases they (Peyer's glands) were scarcely visible; but he does not inform us whether this invisibility arose from cicatrization, or in consequence of their healthy state. Did he use a lens? If not, he then was not in a position to pronounce whether their being out of sight arose from a healthy state or from their actual destruction. In sixteen they were noted to be *visible and normal in appearance.* Now, taking for granted that though *visible* (I presume to the naked eye), yet they were healthy, this would only give us sixteen healthy cases out of forty-three inspections. Again, he informs us that "I have not considered that appearance, likened by French pathologists to the newly-shaven beard, as a deviation from health; this condition was observed in four of the above cases." I need not remark that other pathologists do regard this as not only a deviation from health, but as characteristic of the earliest stages of the typhus deposit. In fact, the French patho-

logists would consider such cases as examples of typhoid fever. However, these four instances reduce the number to twelve cases of "normal appearance." In the other "ten cases, the whole intestinal canal was carefully examined, and recorded as *perfectly healthy; but no note was made as to whether the patches were visible.*" Therefore, according to Dr. Jenner's own statement, only ten cases out of the forty-one were *recorded* perfectly healthy, —"*recorded*," let it be recollected, without any notice being taken of the condition of the patches. Let the reader reflect on these data:—1st, We are told, that with three exceptions, in forty-three inspections the glands were healthy. Then we are told they were *scarcely* visible in fourteen—*visible* in sixteen, but *normal* in appearance!!!—in four of these, elevated to the state denominated pathological by French authors. Then in ten cases the glands are *recorded* as healthy, but no note is taken of their actual condition. In the three exceptional cases ulceration of the patches, and also disease of mesenteric glands existed, but these cases he endeavours to explain away, by the existence of the tubercular diathesis. Without further remark, I leave the reader to judge how much weight he should attach to the conclusion adopted by Dr. Jenner; viz., "that recent disease of Peyer's patches *was absent in every one of the forty-three* cases of typhus fever!!!"

Thus we find, that a careful analysis of the experience of Dr. Jenner affords no substantial evidence that can invalidate the conclusion which I have deduced.

I need not pursue the discussion further. In conclusion, I earnestly appeal to my professional brethren, whatever their views may be on the subject at issue, to avail themselves of every opportunity of inspecting the bodies of those who die from fever.

Let the characteristic symptoms be carefully noted during life, and let every precaution be taken at the inspection. Let the intestine be carefully opened at its mesenteric attachment; let it be carefully sponged and minutely examined, *not* simply for ulceration, but for congestion of the submucous vessels, and infiltration of the follicles with semi-solid or solid matter—(a condition which will be found in the first stages only of fever, generally from the beginning to the middle of the second week.) Let the observer note the condition of the elevated patches of Peyer, especially if his patients die between the eleventh and thirteenth day, he will then generally find many of the sacculi ulcerated. If the patient die from the typhus form during the third week, or afterwards, let a careful examination be instituted, in order to ascertain if any cicatrices exist in the site of Peyer's patches, especially at the lower portion of the ileum, near the *ileo-cœcal* valve.

If these precautions be adopted, I feel convinced that the experience of every observer will confirm the result of my investiga-

tion. The same conclusions will be forced upon him, namely, that the typhoid and typhus forms of fever present similar pathological conditions of the mucous membrane of the small intestine—conditions, identical as to nature, differing merely as to extent and secondary effects.

The length to which this paper extends causes me to defer the consideration of the third topic, viz., "Community of Origin," to the next number of this Journal; in which I intend to consider also the treatment of the intestinal lesions.

---

## IV. *Statistics of the Glasgow University Lying-in Hospital, from 1st November, 1852, till 1st January, 1860; with Remarks by* John M. Pagan, M.D., Professor of Midwifery in the University of Glasgow.

These statistics of the Glasgow University Lying-in Hospital have been prepared by Mr. James Christie, A.M., from the journals, and have at least the merit of perfect fidelity. When the class and circumstances of the patients are taken into consideration, it is matter of surprise that the general results are so favourable. This remark applies in an especial manner to the cases of operation where, for the most part, the difficulties are increased, in no slight degree, by the length of time that the labour has lasted before assistance from the hospital is sought for. To this cause, also, may be ascribed part of the maternal and infantile mortality in cases of operation. In several of the cases of craniotomy, for example, the infant was known to be dead before the operation was had recourse to.

| | Single. | Twins. | Triplets. | Hydatids. | |
|---|---|---|---|---|---|
| Number of Deliveries, | 2586 | 44 | 2 | 1 | |
| Total number of females delivered, | | | | | 2633 |

Twin births 1 in 59·8.

| | Males. | Females. | Abortions. | |
|---|---|---|---|---|
| Number of children born, | 1431 | 1224 | 25 | —2680 |

| | Alive. | Still-born. | Died. | |
|---|---|---|---|---|
| Total number of births, | 2472 | 188 | 20 | —2680 |

PRESENTATIONS.

| | | | |
|---|---|---|---|
| Cranial, | 2555 | | |
| Breech, | 40 | 1 in | 67 |
| Footling, | 28 | 1 in | 95·7 |
| Shoulder, | 2 | 1 in | 1340 |
| Arm, | 2 | 1 in | 1340 |
| Compound, | 18 | 1 in | 148·8 |
| Facial, | 6 | 1 in | 446·6 |
| Placental, | 4 | 1 in | 670 |
| | 2655 | | |
| Unknown, | 25 | | —2680 |

when seen by ordinary daylight, especially if the direct method is employed.

The principles upon which we may determine the state of focal adjustment of the observed eye have been already discussed. The importance of the ophthalmoscope used for this purpose in military and medico-legal practice, has been pointed out by Professor E. Jäger, of Vienna.* He refers to a circumstance, which may rather embarrass the observer in examining hyperpresbyopic eyes in the direct manner, viz., that in extreme cases the apparent magnitude of the image of an object, such as the papilla, may not exceed a third or a fifth part of what it appears in a normal eye. A much larger area is then seen, and this circumstance may add to the difficulty of illuminating hyperpresbyopic eyes, referred to in Part II. The illumination may or may not be fainter than it is in a normal eye, according to the mode in which it is accomplished, and according to the cause of the hyperpresbyopia, whether dependent on absence of the lens, &c., or on shortening of the axis of the eye; but the area illuminated will always appear smaller than one of equal dimensions in a normal eye, and it will seldom occupy the whole of the observer's field of vision.

Apparent motions of objects situated in different planes within the eye, are often very difficult to distinguish from real ones. They may depend on motions of the eye observed, or on motions of the observer, or the biconvex lens held in his hand. The apparent changes in the position of objects caused in this way may be regarded as an exaggeration of those observed in looking at objects from different points of view under ordinary circumstances; except that in the direct mode of examination, it is the most distant objects which appear to move most rapidly, instead of the nearer ones, as is always observed in nature, and generally in the inverted image.

---

## IV.— *Contributions to the Pathology and Therapeutics of Typhus Fever.* By Joseph Bell, M.D., one of the Physicians and Clinical Lecturers, Royal Infirmary, Glasgow.

### No. IV.

### ON THE IDENTITY OF TYPHUS AND TYPHOID FEVER.

### COMMUNITY OF ORIGIN.

If we find the same phenomena accompanying the two forms of fever during life, and the same pathological lesions after death, and if we can trace the two affections to a common origin, then

* Der Augenspiegel als Optometer, österr. Zeitschrift für practische Heilkunde, March, 1856.

it must be granted that a complete proof of their identity has been furnished.

In the previous numbers of this Journal the first two points were as satisfactorily established as the nature of the case will admit. We have now to examine the last topic.

In the course of my own observations, I have witnessed numerous instances of both forms of fever occurring in the same family, and at the same time. For example, a young farmer, aged twenty-five years, was seized with the typhoid form; he had a few elevated, rose-coloured spots on abdomen and chest; his tongue was chopped and red. About the end of the second week, diarrhœa and other abdominal symptoms supervened; these conditions existed during the space of two weeks, after which he slowly recovered. At the end of the fourth week of the illness his father, aged fifty-eight years, was attacked with febrile symptoms. On the sixth day of the disease an extensive typhus eruption made its appearance on chest, abdomen, and extremities, along with other symptoms of typhus, such as stupor and depression; he required the free use of stimulants, but had neither diarrhœa nor any other abdominal symptom. On the fourteenth day improvement commenced, and his recovery was rapid. During convalescence his daughter, aged twenty-two years, was seized with fever; on the seventh day her whole body was covered with the dark mulberry rush peculiar to the typhus form; she died on the eleventh day. On the day of her funeral, another brother, aged thirty years, was attacked; the disease assumed the typhoid form, under which he lingered upwards of a month, having suffered severely from diarrhœa. This family lived in an elevated situation about a mile from the village of Barrhead. It was considered that the son, who was first attacked, caught the infection from a labourer who had recently recovered from fever.

On another occasion I attended a boy, aged fourteen years, during an attack of well-marked typhoid; he suffered much from diarrhœa; became greatly emaciated; his illness lasted upwards of one month. He had three sisters, aged respectively seventeen, twenty-two, and twenty-four years; two brothers, the one aged nineteen, and the other twenty-six. His father was aged fifty-nine, and his mother fifty-three. The two youngest sisters and the youngest brother were attacked, though not simultaneously, yet very nearly so (in the course of the third week of the lad's illness), with well-marked typhoid symptoms, viz., scattered rose-coloured papular eruption, diarrhœa, &c. Next week the eldest sister and brother were seized with the disease in the genuine typhus form, and had extensive mulberry eruptions, stupor, &c. The father was next attacked with typhus; the mother was ultimately seized, and died on the ninth day, having presented true typhus symptoms. The daughter, aged twenty-two, also died, but from extensive ulceration of the ileum. The others recovered.

Ten weeks elapsed from the first seizure till the death of the mother. The family lived in an elevated situation in one of the cottages attached to Ferneze spinning-mill.

I might quote from my note-books many similar instances, showing, that in the same family at the same time, and in the same bed, I have had some members labouring under well-marked typhoid, and others presenting equally distinctive typhus symptoms.

In glancing over some of these old dusty pages, my eye has fallen upon a memorandum of a very striking instance of this community of origin. In the year 1841 a young woman came from Glasgow to Barrhead; she obtained lodgings in one of the worst ranges of buildings in the village, the houses being both greatly overcrowded and badly ventilated. I was called to see her on the third day after her arrival; she had all the symptoms of fever. I immediately communicated with the Inspector of Poor, and recommended her removal to the Glasgow Royal Infirmary, being convinced, that if she were allowed to remain, the disease would spread extensively among the inhabitants.

In these days parochial relief was administered on exceedingly economical principles. The inspector very kindly visited the patient, but he did not participate in my apprehensions; however, after a little importunity on my part, he granted a line of admission to the Infirmary, but would allow half-a-crown only to pay a cart to convey her there (a distance of seven miles). Of course no person would accept of such an insufficient remuneration, consequently she was allowed to remain. The case turned out typhoid; on the ninth day a few rose-coloured spots (about a dozen) appeared over chest and abdomen; about the twelfth day diarrhœa accompanied with pain and gurgling on pressure in the right iliac fossa. The illness lasted nearly six weeks. Mark the consequences—a girl, aged twenty years, a factory-worker (who lodged in the same room), was seized about the beginning of the third week; a few days later the mistress of the house was attacked, the one with typhoid, and the other with typhus. Next, a young man who lived in the opposite room was seized, then his sister, and next his father; the children had the typhoid, but the father the typhus form. The disease went on spreading, until no fewer than thirty cases occurred in this range of buildings, some presenting typhoid and others typhus symptoms. Let it be observed that there was not a single case of fever in the locality when the girl arrived from Glasgow. After the disease had existed some weeks in the buildings to which she came, it next spread, not only to other parts of the village, but extended to Neilston. So much, on the one hand, for the effects of parochial economy, and on the other, for that which is of more importance, our immediate object, viz., the community of the origin of the two forms of fever.

In Glasgow I have not had the same opportunities of tracing

cases of fever as I had in the country; but in the wards of the Royal Infirmary, I have seen on several occasions some members of a family labouring under typhoid, and others under typhus. For example, five members of a family were admitted in August, 1853; three had the disease in the typhoid, and two in the typhus form. Again, on the 23rd July, 1859, a girl, aged twenty-three, was admitted on the 5th day of fever; on the 7th day the mulberry eruption of typhus covered almost the entire surface of her body. Convalescence commenced on the 2nd of August, and she was dismissed on the 16th. A cousin, who lived in the same house with her, was seized with febrile symptoms on the 23rd; that is, seven days after the girl returned home; he was admitted on the 30th; he had a well-marked attack of typhoid, a few rose-coloured spots, which appeared in successive crops and abdominal tenderness and diarrhœa. He was a month under treatment.

Another girl, aged nineteen years, was admitted on the 4th of August, labouring under fever of nine days' duration; her body was covered with a very distinct mulberry rash; bowels constipated. The other symptoms, which occurred in the course of her illness were distinctly typhus. Her two brothers, aged respectively seven and fourteen years, were admitted on the 10th and 14th of August, both having the disease in the typhoid form.

The few cases to which I have now alluded by no means exhaust my experience on the subject—an experience which is far from being unique, or unprecedented in the practice of other physicians. In the last number of this Journal I quoted the result of Dr. Stokes' observations on the point. He tells us "that we have frequently observed in cases where a large number of a single family have been successively attacked by contagious fever, that every form of the disease, from the most malignant typhus to the mildest typhoid fever, may be presented by different members of the family."*

Dr. Huss of Stockholm, to whose excellent work on fever I have already referred, states that "At the barracks of the gensdarmes, out of 250 men, 64 were taken ill with fever in the course of six weeks. Although the men lived under exactly the same circumstances, were exposed to the same etiological agents, were all between 20 and 40 years of age, the disease assumed the distinct form of typhus in one part of the cases, of typhoid in another, and in a third an intermediate form. In the private house of a cabinetmaker I saw 17 cases within a fortnight, of which 10 were typhus and 7 typhoid fever, although here also the dwelling and other circumstances, with the exception of sex and age, were the same for all.

"The following incident may also merit notice:—A man had died, it was stated, of typhus. The brother and his wife went to

* Disease of Heart, p. 450.

live in the house of the deceased, and used his clothes without previous airing and cleaning. They were soon taken ill and brought to the hospital, where they both died. The husband had violent delirium and a profuse petechial eruption, the post-mortem examination showing no change of the intestinal glands; the wife had milder cerebral symptoms, and a very scarce crop of eruption; but on examination, swollen mesenteric glands and ulcerated Peyer's patches were found in the abdomen.

"The same experience has been made during these years, when typhus has occurred only sporadic; some cases have taken the characters of typhus, others of typhoid fever, though the latter were, under these circumstances, always more numerous. The season also has in this matter shown a decided influence; sporadic typhus occurring during autumn and winter, while spring and summer have introduced the typhoid cases.

"Although these facts are founded on observations made in Stockholm, I believe I may state with probability, if not with absolute certainty, that the same may be said of the rest of Sweden, to judge from the communications received from physicians of different parts of the country." In a foot-note he relates a most important occurrence which completely confirms the statement made in the text. "Amongst other information, which has been communicated by my colleague in the country, the following from Dr. F. Lang in Gottenburg merits special notice, as it proves that the typhus process in other parts of the country agrees, in the respect in question, with that of the metropolis. A traveller came to a small island situated on the western coast of Sweden. He was sick when he arrived, and was the same day laid up with a fever. The disease showed all the marks peculiar to typhus petechialis, viz., clearly marked alteration of the blood, and a very copious typhus eruption (ecchymotic petechiæ), and ended on the ninth day fatally. Seven persons were successively taken ill on the island; only one of these presented the marks characterizing typhus; the remaining six cases, of which one was fatal, were all clearly distinct typhoid. The course of this island-epidemic made it evident that it was produced by infection from the first diseased person, who came ill; before whose arrival no case of typhus or typhoid had been seen for several years either on the island or in the neighbourhood, and that the same contagion produced both typhus and typhoid fever."

I do not see how we could possibly have more powerful or conclusive proof of the identity of the two forms of fever than that which is furnished by the series of facts that I have now brought forward, not only from my own observation, but also from the experience of Stokes, Huss, and others. If such facts fail to convince the reader that typhus and typhoid fever have a common origin from the same morbid poison, then, I frankly confess, that

I am unable to present him with anything of a more decisive character.

The advocates of non-identity have produced much ingenious and very plausible writing, with the view of either weakening the force of these facts, or of withdrawing the attention of the reader from the due consideration of the real question at issue.

For example, such cases are said to be "rare," and hence it is argued that they are of no moment in the solution of a question already settled by other evidence. "They are, however, of such rarity, while at the same time all other evidence tends to establish the non-identity of the poisons of the two diseases, that it seems more probable that they admit of another explanation."* In this passage we have, in the first place, the "rarity" simply asserted. In the second place, the non-identity taken for granted, and in the third place, the conclusion deduced, that because they are non-identical, the reader must enter upon the region of probabilities to seek for an explanation of such cases, as they cannot possibly arise from the same poison—a process of reasoning so very characteristic as to preclude comment.

Another writer, who has lately joined the ranks of non-identity, criticises the statement of Dr. Huss in the following terms:—"In the elaborate work of Dr. Magnus Huss of Stockholm, one of the latest defenders of the theory of identity, much is made of a single instance, in which a man and his wife, having gone to inhabit an infected house, were seized, one with typhus, and the other with enteric fever." The writer first suggests, "the possibility of error and of oversight in an isolated observation of this kind." He next adverts to the experience of Dr. Huss regarding epidemics of both diseases occurring simultaneously, and then sums up by observing, "but, that under these circumstances, the coincidence of typhus and enteric fever in the same family should be so rare as to demand special notice, seems to me to be a far more serious objection to the views of Dr. Huss than can be overcome by any of his arguments upon the other side."†

I beg to request especial attention to the quotation which I have made from Dr. Huss. It will be seen that he does not try to make much of this single instance; he merely calls it an incident worthy of attention—an incident by no means an "isolated" one, as the writer is pleased to style it, but one of frequent occurrence. After relating the circumstances of this so-called isolated case, Dr. Huss remarks that the same experience has been made during those years when typhus has occurred only sporadic, some cases taking the characters of typhus, others of typhoid, though the latter were, under these circumstances, always more numerous. Then he alludes to such cases as being common in the practice of other Swedish practitioners. How the case alluded to can be

* Dr. Murchison, Med. Chir. Trans., vol. xli., p. 277.

† Ed. Med. Journal, Sept. 1859, p. 245.

called a "single instance," I leave the reader to judge. It is singular that the writer of the criticism should have neglected to mention the instances of the barracks, and of the cabinetmaker, to which Dr. Huss alludes, especially when they were detailed in the very same page as the "incident;" and is still more remarkable that no notice is taken of Dr. F. Lang's experience, which I have quoted, and which is given in a foot-note on the page opposite to that in which the so-called "isolated case" is noted.

But is the experience of such an extensive, painstaking, and careful observer as Dr. Stokes to be overlooked? A man who has seen probably more cases in a few months than some of the writers who favour non-identity have seen throughout their lives. Does he speak of such cases as being rare? In the quotation which I have made from his work on Diseases of the Heart, it is stated "that we have frequently observed such instances. Rare, indeed! It reminds us of the adage, "there is none so blind as those who will not see." I think that I need add no more in order to settle the argument founded on "rarity."

I believe "that it may not have occurred to some of those who make use of this argument," to have observed the simultaneous progress of typhus and enteric fever in one house, or even in one "land." Still, however, this limitation of their experience does not disprove the numerous observations of many other competent observers; very probably a greater familiarity with fever may afford them, not merely isolated examples, but numerous cases of the kind in question. I need not remind them of the story of the Indian monarch who would not believe in the existence of ice because it was inconsistent with his experience.

The following advice is given by a modern facetious writer:— "When any person brings an accusation against you, if it is difficult to rebut, the best plan to follow is to urge a counter charge against the accuser; this being always much more easily done than to disprove his allegation."

Some of the advocates of the non-identity theory seem to act very much on this principle.

When they are unable to controvert such facts as I have now quoted, facts which irresistibly point to a community of origin, they announce quite a new theory, viz., that typhus and typhoid fever have "different foci of infection." That the former arises in over-crowded buildings which have a deficient ventilation, and whose inhabitants are in a state of more or less destitution; and that the latter prevails among people who are exposed to emanations arising from cesspools, churchyards, gully-holes, and choked up drains in which decomposing organic matters exist. To the reflective reader it must appear obvious that the real question at issue is entirely overlooked in this argument. It is not the identity of the foci, but the identity of the *poison* that constitutes the matter

in dispute. The "foci" may be merely predisposing causes (which I readily grant they are), and consequently possess no exciting influence. The poison may be identical, and have its manifestations merely modified by the agencies which these writers denominate "foci;" therefore, though a difference of "foci" could be established, yet that would not prove a non-identity of the poison. An examination of the facts and circumstances brought forward by those who employ this argument, will satisfy the reader of the correctness of the statement which I have now made, and will show further that the argument itself is quite untenable, being founded on mere assumptions, imperfect observations, and supported by most fallacious reasoning.

In the thirty-third volume of the Medico-Chirurgical Transactions, Dr. Jenner has published an essay, in which he attempts to show that typhus and typhoid fever arise from different foci, that each has its own specific cause, or morbid poison, as essentially distinct as that of measles is from that of small-pox, &c. What are the proofs or evidence on which he forms this conclusion? They are twofold. 1st. He affirms that the poison of small-pox will not produce measles, or the poison of the latter will not originate scarlatina, or *vice versa*. No person doubts these facts, but how do they bear upon the identity or non-identity of typhus and typhoid? In fact they have no reference to the question at issue. The manner in which Dr. Jenner uses the facts, as an argument in favour of his views, is perfectly illogical. He takes for granted the very subject to be proved, and assumes that as typhus and typhoid are different diseases, they therefore must require for their production the application of distinct specific causes.

Besides this serious fallacy, Dr. Jenner conceals a premiss which excludes a fact that furnishes a powerful argument in favour of the identity of the two forms of fever. He entirely keeps out of view the circumstance that the specific causes of small-pox, measles, and scarlatina *do not* uniformly produce the same symptoms. In his concealed premiss he assumes that an invariable uniformity marks the phenomena of these diseases. He thus silently ignores the important fact, that from the same poison we either may have a most terrible case of confluent small-pox, or we may have death produced, without the appearance of a single pustule, or we may have the disease so mild, and the eruption so trifling, that only a few spots can be detected over the body. How different then are the general and local manifestations of the same poison? What immense disparity do we not witness between the simple, innocent-looking vaccine pustule on the child's arm, and the purulent mass which covers the face and extremities of a patient labouring under confluent small-pox? Yet in both cases the poison is essentially identical. In scarlatina the same poison often destroys a patient in a few hours, without

any cutaneous eruption. In another the skin is extensively affected, and the tonsils nearly destroyed; whilst, in a third, a mere efflorescence and slight febrile disturbance are only produced; and in a fourth, a slight congestion of the fauces may be the only result. Similar circumstances are remarked among the different manifestations witnessed during epidemics of rubeola and cholera. I consider that the man must have his perceptive faculties greatly impaired who cannot see greater differences between the phenomena of the above-named diseases than that which appears between cases of typhus and typhoid fever. The proper argument furnished from a consideration of the history of exanthematous diseases, is the very opposite to that deduced by Dr. Jenner. We see that a poison essentially the same produces in different individuals non-identical symptoms, and different pathological lesions, and therefore the differences which we see between cases of fever should be considered as mere dissimilarities in the manifestations of the same poison or specific cause. It is as absurd as it is illogical to argue from the fact that the specific poison of one exanthematous disease will not produce the peculiar manifestation of another; that, therefore, typhus and typhoid fever must each have its own specific cause. The diseases, as viewed in the argument by Dr. Jenner, are not parallel; and, besides this, there does not exist the slightest logical connection between the premises and the conclusion. It would not be more absurd to attempt to prove a difference between Benlomond and Snowdon from a comparison between London and Glasgow Bridges, than to try to prove that typhus and typhoid fever must arise from different foci of affection, by appealing to the impossibility of originating measles from the specific poison of small-pox.

Dr. Jenner's argument is, therefore, nothing more than a series of fallacies. He takes for granted the very question at issue, and argues that because typhus and typhoid fever present different phenomena, therefore each must have a different specific poison from which it originates. He thus begs the question, and, then secondly, by the "circular" fallacy, argues that as each has its own specific poison, they are consequently non-idential diseases. Thirdly, the argument founded on exanthematous affections exhibits the well-known fallacy of a "non tali pro tali." In the suppressed premiss he affirms a parallelism which does not exist, namely, that the phenomena of these specific affections are always uniform in every case—a proposition totally inconsistent with fact. It is by carefully concealing this premiss that he is enabled to attach importance to his reasoning and conclusions.

His second argument is equally fallacious; it is founded on the circumstance that during three years, viz., 1847, 1848, 1849, he ascertained among the patients admitted into the London fever hospital, that only two instances occurred in which typhus and

typhoid cases came from the same house and family. He affirms that in every other case the typhoid and typhus patients came from different localities. He therefore infers that the two diseases must arise from different specific poisons.

The fallacy of this argument is very obvious. In the 1st place, it is a well known fact that during the prevalence of any epidemic, the cases which occur among the destitute and overcrowded localities are much more malignant than those which appear in other places of the same district. 2nd. The period of three years is by far too limited to authorize the general, indeed, I may say the universal conclusion deduced by Dr. Jenner. 3rd. The two exceptions upset his conclusion. These two cases are *positive* facts, and are of immensely greater value in reference to the question at issue than many negative instances.

It is quite illogical to argue, that because he found a series of typhoid cases in certain localities, and typhus cases in others, that these forms of disease must necessarily arise from different morbid agencies. The character of the people, as well as that of the locality in which they dwell, must powerfully influence the activity of any specific poison, and thus modify its phenomena.

But if, on the other hand, we find typhus and typhoid cases existing simultaneously, or nearly so, in the same house, or in the same family (not rarely, as Dr. Jenner's observations would lead us to believe), but very frequently, according to the experience of Stokes, Huss, Kennedy, myself, and others, the conclusion that the two diseases do arise from the same poison cannot be set aside by any inference, however ingenious, deduced from such negative considerations as Dr. Jenner has produced. I repeat that he has committed two grave fallacies in this argument. 1st. He infers from his own limited experience of three years, the same of the collective experience of all others. 2nd. His experience will not justify the conclusion, viz., that the two diseases must arise from different poisons.

The views of Dr. Jenner, regarding the origin of typhoid and typhus fever from different foci of infection, have received the support of several writers, among whom Dr. Murchison deserves, perhaps, to be considered the most distinguished. Before examining his contributions to the subject, I beg to inform the reader that he differs essentially with Dr. Jenner regarding relapsing fever. Dr. Jenner considers this a totally distinct affection, both from typhus and typhoid fever. Dr. Murchison, on the other hand, contends that it is *identical* with typhus.

To those practitioners who witnessed the epidemic of 1843 and 1844, it must appear obvious that Dr. Murchison cannot have formed his opinions on this subject from clinical experience. The closet and the bedside—the book and the patient—are very different data upon which correct opinions are to be founded. But we may leave the two learned physicians to settle this important

point, and proceed to investigate the additional evidence which Dr. Murchison has furnished to show that typhus and typhoid fever arise from different specific poisons, or as he styles it, "from different foci of infection."

His views and arguments will be found in an essay "On Continued Fevers," published in the 41st volume of the *Medico-Chirurgical Transactions*, and in an article on the "Causes of Continued Fevers," published in the *Edinburgh Medical Journal* for August, 1859.

He contends that typhus fever is caused by destitution and the concentrated exhalations from living bodies, and that typhoid is caused by putrid emanations from sewers, &c. These propositions embrace three questions—1. Do these alleged causes originate either of the two forms of fever? 2. Does either of these forms so invariably appear in the localities which Dr. Murchison assigns, as to authorize the conclusion which he deduces? 3. What is the essential difference between emanations arising from the decomposition of the contents of sewers, cesspools, &c., and those exhaled in the overcrowded dwellings of the destitute?

The consideration of these questions may appear to be a digression from the main subject of this paper, but an investigation into their correctness becomes necessary, because they constitute the premises from which Dr. Murchison deduces the conclusion of "two different foci of infection," and consequently infers the non-identity of the two forms of fever.

1. *Does Typhoid Fever arise from the putrid Emanations of Cesspools, &c.?*—Dr. Murchison, and those who answer this question in the affirmative, bring forward numerous instances in which fever of the typhoid form seems to have occurred in localities where the drainage was defective, and where emanations proceeded from the contents of cesspools and the like. On the other hand, those who take the opposite view adduce numerous examples, in which the conditions of putrid emanations and overcrowding prevailed to a much greater extent than that quoted by Dr. Murchison, and yet no fever resulted. They also point to another series of facts which show that both forms of fever have occurred where no such conditions as either foul drains or overcrowding existed.

I beg now to examine (with as much brevity as possible) a few of the examples which Dr. Murchison brings forward in support of his views. He refers to an epidemic at Croydon in 1852, and refers to Mr. Carpenter's article, published in the *Association Journal*, vol. ii., p. 900. "Mr. Carpenter," Dr. Murchison affirms, "has adduced facts which render it highly probable that in many cases the fever was owing to the contamination of the drinking water in the wells from the disturbance of the drains." If the reader refers to the paper of Mr. Carpenter, he will find that this gentleman merely states that "the surface sewerage found its way into the wells, and that this complication

gave rise to a fever of a typhoid type, differing in degree from the generality of cases which occurred indiscriminately in houses connected, as well as unconnected, with the Board of Works; these I believe were entirely due to a foul supply of water, assisted by the undoubted epidemic tendency of the season."

This is a very different statement from the version given by Dr. Murchison; in fact, it appears that fever was epidemic at Croydon, and that those parties seized by it who had to drink the contaminated water, presented abdominal symptoms, namely, diarrhœa. The facts only show at most that the drinking of putrid water acted as a modifying influence, not as an exciting cause. Without further comment, I leave the reader to judge how much confirmation the fever of Croydon yields to Dr. Murchison's views. He next refers to Dr. Beadle's account of a fever which broke out at Tewkesbury in August, 1853. He states that Dr. Beadle's description "leaves no doubt but that it was typhoid." Let the reader turn up the *Association Journal* for 1853, and at page 733 he will find Dr. Beadle's paper, in which article the term typhoid never occurs. The fever is described as an epidemic of "typhus." He narrates ten cases; the first he calls mild typhus, without any abdominal symptoms, and on the seventh day the patient was in a state of approaching convalescence. The greater part of the other nine cases had pulmonary complications, three only had diarrhœa, and in all the fatal cases delirium, prostration, the alvine discharges being passed involuntarily, with other adynamic symptoms. No post-mortem examinations are reported. I presume none were made. No doubt Dr. Beadle states that he ascribes this epidemic of typhus partly to want of drainage and ventilation, human effluvia, concentrated and damp soil, and partly to infection; but on what principle Dr. Murchison affirms that Dr. Beadle's "description leaves no doubt that the epidemic was typhoid," I am at a loss to conceive. In fact the description proves the very contrary. The disease had all the characters of genuine typhus, and the author of the paper does not ascribe it solely to sewerage or cesspools, but to a multiplicity of causes, among which we find infection. Dr. Murchison alludes to Mr. Taylor's account of an epidemic of fever which broke out at Old and New Lenton, in the year 1846, and refers to the 15th volume of the *Medical Times* for a description of the disease. At page 159 of this volume the reader will find a report of a lecture delivered by Mr. Taylor on the subject. On reading this it will be found that Mr. Taylor considers filth and decomposing emanations principally as "a predisposing cause, and rarely as an exciting one." Again he mentions the important fact that the cases were most numerous at New Lenton, whilst the abominable contaminations were most intense at Old Lenton. He asks the question—"Why did not the disease ravage more particularly the locality where the poison sprung from?" He

answers this by stating that the inhabitants of New Lenton were exposed to *additional predisposing causes*, "there being destitution and over-crowding." From this it is evident that Mr. Taylor regarded the emanations as *predisposing* causes. If the reader will refer to page 727 of the *Medical Gazette*, vol. iii., new series for 1846, he will find that fever was epidemic at the time, not only in Old and New Lenton, but in Bradford, Nottingham, and the country district around; and that in accordance with general experience of all epidemic visitations, those places suffered most in which filthiness, poverty, and over-crowding existed. This, however, is a very different matter from the origin of the exciting cause.

Dr. Murchison next refers to Mr. Turner's account of fever at Minchinhampton in 1846–47, recorded in the *London Medical Gazette*, vol. xlii., p. 157 (May, 1848). Dr. Murchison asserts that Mr. Turner states in his paper that "the privies at the back of the houses in which many of the fever cases occurred, exhaled an intolerable stench"—Ergo, fever arose from the stench. On reading the paper it will be found that Mr. Turner furnishes us with a very different account. After some preliminary remarks, he tells us that "no fever had been at Minchinhampton for nearly half a century, till some few cases made their appearance here at the end of 1845, and that without any *satisfactory* cause. The fever of 1845 was of the simple, or continued form. . . No case of fever presented itself in this place until the end of June, 1846, and that case happened in the person of a little girl who came from Reading upon a visit to a gentleman's house in the town. The second case was that of the lady at whose house the child was visiting—this was about three weeks from the child's illness. The third case which occurred was the gardener. It then appeared at the other end of the town, just where you might expect, namely, at the house of the person who washed the linen; one or two washerwomen became ill, and from that time cases broke out here and there, not only in town, but surrounding it. Isolated cases occurred here and there in the country, clearly traceable to local causes. Indeed, one of my worst cases evidently arose from the miasma arising from drains and privies at the back of my patient's house." Here we have the epidemic clearly traced, not to exhalations, but to infection; and we have only one case ascribed to privies instead of the "many" asserted by Dr. Murchison. I think the reader will agree with me in ascribing this one case, that of a druggist, let it be recollected, to contagion, and not to the intolerable stench. Mr. Turner mentions several other instances in which the disease arose from, and was propagated among families by infection alone; hence we perceive the doctrine of the origin of fever from putrid emanations receives no support from the epidemic which prevailed in 1846, not only at Minchinhampton, but at other places in the same district. We

find that the disease arose from infection, and not from the effluvia of drains, cesspools, &c.

Dr. Murchison alludes to a formidable outbreak of fever in 1848 among the boys attending the Westminster School. This he ascribes to the disagreeable stench which proceeded from a foul and neglected sewer, in which "fecal matter had accumulated for years without any exit, and which communicated by direct openings with the drains of all the houses in which it occurred."

He admits that several boys were also attacked who lived in a house at a little distance, "but who were in the habit of playing every day in a yard in which were gully-holes opening into the foul drain." It seems strange how it happened that no fever resulted during the series of years throughout which their fecal accumulations existed prior to 1848. The reason is obvious, namely, that some other cause, either additional to or independent of the stench, must have been in operation to produce the disease. I may also mention that Dr. Watson of London did not consider the affection to be continued fever at all, but an affection of the gastro-intestinal mucous membrane.

I will not stop to analyze the evidence of a clergyman who was present at an inspection of one of these drains, and who exclaimed that it "breathed out typhus!!!" But we cannot pass over a celebrated "dancing proof" without some little comment.

"Two balls had been held at a hotel in Cowbridge, Wales, in November, 1853. About 140 persons were present from all parts of the surrounding country. Shortly after, many of those persons were seized with fever, "presenting," according to Dr. Murchison, "all the symptoms of typhoid, and about eight died. This fever was not prevalent at the time, and it only attacked those who had attended the balls, some of whom were not taken ill till after their return to their homes in Devon and Somerset. An inspection of the hotel was made by order of the local authorities, and it turned out that the supper-room was merely a temporary transformation of a *loft over a seven-stalled stable*, and that the passage between it and the ball-room was built over a large tank which collected the water from the roof of the house. In this case, also, there were suspicions of poisoning."—*Med. Chir. Trans.*, vol. xli. p. 270.

I ask, can any man, unblinded by theory, believe for a single moment that an extensive and very fatal typhoid fever could originate among the people who passed an hour or so in a stable loft, and merely crossed a few times a lobby built over a rain-water tank!!! The notion is perfectly preposterous. If such an intense poison emanated from these sources, why did the stable boys, the waiters, the landlord, and his family, who were constantly exposed to them, escape? Is it not much more likely that the viands were the cause of the disease—a disease

essentially dissimilar in most important points from typhoid fever. If the reader will refer to page 476 of the 31st volume of the *Medical Times and Gazette*, he will find a report of Dr. Camps' paper as it was read at the meeting of the Epidemiological Society of London, April 2, 1855. He will be astonished to find that Dr. Camps drew up his paper from hearsay information, derived mostly from non-professional persons more than a year after the occurrence of the attack; but his astonishment will be still greater when he finds that instead of the disease "presenting all the symptoms of typhoid," as represented by Dr. Murchison, it is stated by Dr. Camps "that all authorities from which he had been able to procure trustworthy information on the subject, agreed in representing the disorder as *typhus fever of a very low and malignant type*, characterized by *extreme debility and prostration* of strength, and in *some cases attended* with hemorrhage from the intestinal canal." It is also worthy of note, that none of the members of the Society who took part in the discussion which followed the reading of the paper, ascribed the disease to the stable or tank, but some to sudden exposure to cold, and others to some morbid poison taken in the food. The "ball proof" is therefore a complete failure.

Dr. Murchison has made much of an outbreak of fever which took place recently at Windsor. On a perusal of his article in the *Edinburgh Medical Journal* for 1859, it will be found that no trustworthy information is given of this epidemic. 1st, That Dr. Murchison shows in his paper that grave mistakes in diagnosis were made during its prevalence, so much so, that he found many of the cases reported as fever, were instances of scarlatina, which also prevailed at the time. 2nd, Diarrhœa and dysentery seem also to have been very common prior to, and co-existent with, the fever. I hold it much more likely that acute dysentery would be mistaken for typhoid fever than scarlatina; and as dysentery often results from morbid effluvia, this circumstance tends very much to diminish our confidence in the relation which Dr. Murchison and others attempt to establish between typhoid and putrid emanations, because many of the cases may not have been fever at all, but simply autumnal dysentery.

Dr. Murchison gives us a table prepared from the register of deaths. This table he heads, "Deaths from Pythogenic (Typhoid) Fever in the Windsor district in the year 1858, from the Register of Deaths (official)." He tells us that "the number of deaths referable to pythogenic fever (although entered under different names) in the district register was 34. . . . . The cases exhibited all the characters and history of the fever which has been described as typhoid fever, and for which I have proposed the term Pythogenic, including a red fissured tongue, abdominal pain, tympanitis, diarrhœa, hemorrhages, an eruption of successive crops of rose-coloured papules, and a protracted

duration. I had abundant opportunities of verifying these observations for myself." (P. 503.)

The best way to allow the reader to estimate the value of the above statements is to place before him this table, as quoted by Dr. Murchison:—

"DEATHS FROM PYTHOGENIC (TYPHOID) FEVER IN THE WINDSOR DISTRICT IN THE YEAR 1858.

*From Register of Deaths (official).*

| No. | Date | Age and Sex. M. | F | Disease as entered in the Register. | Residence. | Remarks, Name, etc. |
|---|---|---|---|---|---|---|
| 1 | May 5 | ... | 15 | Brain Fever | Clewer Green | Removed ill from Trinity Place, Windsor. |
| 2 | Aug. 7 | 5 | ... | Brain Fever, 14 days | 71 Bexley Street. | |
| 3 | " 9 | 26 | ... | Typhus Fever, 3 weeks | 89 Peascod Street | Came ill from London. |
| 4 | Sept. 2 | 10 mos. | | Gastric Fever, 10 days | Spital, Borough side | Minell. |
| 5 | " 27 | 24 | ... | Fever and Congestion of Brain | Infantry Hospital | Came ill from Ascot Heath. |
| 6 | " 27 | 21 | ... | Do. and Ulcern. of Bowels | Do. | |
| 7 | Oct. 18 | ... | 37 | Low Fever | 8 Brunswick Terrace | |
| 8 | " 21 | ... | 41 | Typhoid Fever, 28 days | Victoria Street | Rider. |
| 9 | " 22 | ... | 18 | Typhus Fever, 9 days | 27 Park Street | Brought home ill. |
| 10 | Nov. 3 | ... | 23 | Fever, 7 days | William Street | Taylor. |
| 11 | " 4 | 1 | ... | Remittent Fever, 14 days Bronchitis, 3 days | Russell Street | Simms. |
| 12 | " 12 | 15 | ... | Low Fever with Ulcern. of Bowels, 14 weeks | Royal Mews | Clark. |
| 13 | " 14 | 21 | ... | Typhoid Fever, 4 weeks, Renal hemorrhage | Victoria Street | Shirley. |
| 14 | " 15 | 12 | ... | Typhoid Fever | Victoria Street | Rider. Same house as No. 8. |
| 15 | " 15 | ... | 2 | Fever and Diarrhœa | 81 Bexley Street | |
| 16 | " 16 | ... | 38 | Typhus Fever, 21 days | Old Windsor | Wife of a gardener taken ill there. |
| 17 | " 17 | 59 | ... | Fever, Exhaustion | Sheet Street | Rangecroft. |
| 18 | " 18 | ... | 28 | Typhoid Fever, 14 days | 4 Adelaide Terrace, Sheet Street | |
| 19 | " 20 | ... | ? | Nervous Fever, 3 weeks Congestion of Brain | 11 Park Street | |
| 20 | " 20 | ... | 26 | Typhoid Fever, 14 days | Lower Ward of Castle | Gosset. |
| 21 | " 21 | 69 | ... | Typhoid Fever, 17 days | Do. Do. | Harris, Lay Clerk St. Geo.'s Chapel. |
| 22 | " 24 | 36 | ... | Low Fever, 3 weeks | 5 Adelaide Square | |
| 23 | " 24 | 5 | ... | Typhus Gravior | 1 Gloucester Place | Youngman. |
| 24 | " 24 | 9 | ... | Gastritis | Victoria Street | |
| 25 | " 25 | ... | 4 | Fever, 3 weeks | Sun Pas. Peascod St. | Snell. |
| 26 | " 26 | ... | 16 | Scarlatina Maligna, 14 days, with mortification of Bowels | 3 Goswell Place | Originated in Park Street. |
| 27 | " 27 | 18 | ... | Typhus Fever, 3 weeks | Sheet Street | Second death in this house. |
| 28 | " 28 | .. | 19 | Sloughing Sores after Fever | Union Workhouse, Old Windsor | Removed from Windsor. |
| 29 | " 29 | ... | 30 | Typhus Fever, 14 days | 3 West Row Street | Removed from 4 Brunswick Ter. |
| 30 | " 30 | ... | 21 | Typhus Gravior | Royal Mews | Sayer. |
| 31 | Dec. 3 | ... | 6 | Exhaustion following fever, Ulcern. of Bowels | 9 Gloucester Place | |
| 32 | " 5 | ... | 3 | Convulsions after Low Fever. | 2 Brunswick Terrace, Sheet Street | |
| 33 | " 15 | 29 | ... | Exhaustion following Fever | 1 Gloucester Place | Youngman. |
| 34 | " 23 | 8 | ... | Typhus, Hydrocephalus | Royal Mews | Long. |

*Note.*—In addition to the above, at least 5 persons contracted the fever in Windsor, and died after removal to their own homes in other districts."—*Edin. Month. Jour., Aug.*, 1859.

A careful examination of this table will show that it does not give the slightest ground for the assertions made by Dr. Murchison; on the contrary it positively contradicts his most important statements. "No doubt," he observes, "that typhoid has been registered under different names." But what evidence does he adduce for this very grave charge against the correctness of the returns, which must have been made by the medical men in attendance on the cases. The table itself, however, bears internal evidence of the incorrectness of Dr. Murchison's opinion. We have "brain fever;" next, "brain fever of fourteen days." *Protraction* did not characterize these cases, as Dr. Murchison alleges. The medical men seem to have been quite alive to the distinction between typhus and typhoid, and have reported eight cases of the former, and one of typhoid. I repeat that the register contains internal evidence of the correctness of the diagnosis; the duration in the typhus cases being short, from 9 days to 21 days. Then two of the typhus cases are designated typhus gravior, which renders their true nature unmistakable. Then the typhoid cases seem to have had a much more protracted duration from 14 days to 4 weeks. Again, we have a case of gastric fever of 10 days' duration—how protracted! Another case is entered as fever and congestion of brain—a rare occurrence in typhoid fever!! Another case is entered as low fever; another as fever of 7 days—how characteristic of typhoid!! Then we find a case of remittent fever of 14 days' duration, terminated by a 3 days' bronchitis. Again, we find another case of nervous fever with congestion of brain. One case is entered as gastritis; another is recorded as "scarlatina maligna of 14 days' duration, with mortification of bowels." Still Dr. Murchison calls all these instances "characteristic cases of typhoid fever." Let the reader recollect that Dr. Murchison regards all these 34 cases as examples of typhoid fever, notwithstanding the most forcible internal evidence furnished by the table to the contrary, and he describes them, as I have already quoted from his paper, as exhibiting "all the characters and history of the fever, which has been described as typhoid fever," &c., &c.

Without further comment I will leave the reader to form his own judgment of the amount of confidence which he should place on the allegations made by this historian of the Windsor epidemic of 1858, and how much proof this epidemic affords as to the origin of fever from putrid emanations. These no doubt existed to a very great degree, but then they had long done so to a similar extent, without the presence of fever, a fact which should at once suggest that they had no effect in originating the disease, however much they may have contributed to its extension among the inhabitants. It is somewhat remarkable that Dr. Murchison and others completely overlook the fact, that packed up drains, offensive sewers, fæcal accumulations, overpowering

stenches, with all the other species of putrid decompositions and fetid exhalations, existed for years without originating fever of any description—an important circumstance, the existence of which in every instance brought forward by Dr. Murchison cannot be questioned.

I deem it unnecessary to examine any more of these "beggarly elements," which constitute "the array of evidence which has been brought forward," and which Dr. Murchison considers "demonstrates as clearly as can be, that typhoid fever is often if not always generated by the putrid emanations from drains and other sources, or by decomposing organic matter in drinking water."

With regard to the origin of typhus, "the array of facts" will be found to be of a similar character. They consist of mere coincident associations. No one doubts but overcrowding and destitution are frequent, if not universal concomitants of typhus; but there have been very few unexceptional instances, if any, adduced to show that typhus has originated from such alleged causes.

I need scarcely remind the reader that those members of the profession who have had the most extensive experience of fever, and who have directed especial attention to its origin and the laws which govern its propagation, have been most decided in stating that the causes alleged by Dr. Murchison are quite incapable of originating the disease. I could easily fill more than a number of this journal with such opinions; I will, however, only refer to one or two. The late Professor Alison states in his evidence before the Poor Law Commission, "I do not maintain that destitution has power to engender fever." The late Dr. Perry of Glasgow states in answer to the question, "Are there any local circumstances in Glasgow that encourage epidemics in those quarters?" Ans.—"When epidemic fever is prevailing, there are circumstances which increase its prevalence, but *never* in any case do these originate contagious diseases."

Query.—"But are there local circumstances which encourage disease?" Ans.—"I know nothing that does so, but crowding of people together in small dwellings, and these probably filthy lodging-houses, and places of that kind. I am satisfied this never gives origin to fever, though it spreads it when it occurs as an epidemic."

Query.—"Do you think the want of proper sewerage contributes?" Ans.—"I believe that it has no effect whatever. I have paid particular attention to that subject."*

Such is the experience of two eminent men, who had an opportunity of observing fever of the most extensive character during a long series of years. There are no places in Great Britain in which fever prevailed to the same extent and frequency as in Edinburgh and Glasgow, and no men had better opportunities

* Report of Poor Law Inquiry, p. 58.

of forming an opinion as to its origin and diffusion than Alison and Perry.

In Dundee, fever also has been a most frequent visitor. Dr. Arrol, physician to the infirmary, gives similar evidence to the same effect, viz., that whatever influence destitution, overcrowding, filthy emanations, may have had in increasing the spread of fever, they had no influence in originating the disease *de novo*. The history of fever in Glasgow since Dr. Perry's time amply confirms his views. I am sorry to say that overcrowding, deficient ventilation, bad sewerage, fæcal accumulations, and putrid emanations exist in the city to an extent as great as that which occurred in his experience; but how stands it with fever? Then the deaths from fever was nearly 12 per cent. on an average of seven years, sometimes as high as 20 per cent., but now the deaths have fallen to a mere fraction. If putrid emanations produce typhoid, and if overcrowding originates typhus, whence then our fortunate immunity from both forms of the disease for upwards of an entire decade. Both of these alleged causes exist at the present period to an extent and with an intensity unsurpassed at any period of the history of the city, and yet neither typhus nor typhoid prevail. For example, let me direct the attention of the reader to the condition of the Camlachie Burn. The sluggish stream at Camlachie receives all kinds of impurities in the shape of decaying organic matters, including the drainage from a manure manufactory. Afterwards, as it passes through Mile-end, it receives the contents of the privies attached to a number of the factories which are thickly situated along its course, and also the surface drainage from the densely-populated streets; after it reaches Calton, it still continues to receive the contents of privies and drains; in fact, it becomes a large open sewer, a common receptacle for every species of filth, both animal and vegetable.

But this is not all; its water is pumped into the engine-rooms of the different factories, and used as condensing water. It is then returned to its bed reeking hot, sending up volumes of stinking vapours along its densely-populated margins. In order to secure a sufficient supply for condensing purposes, the stream is dammed up at several places in its course, so that it becomes a stagnant, fetid pool, a filthy cauldron from which clouds of effluvia are constantly arising, and when the eye can penetrate through the thick mist, it rests upon a most disgusting dark-coloured, thickish fluid, in which the decaying carcasses of cats, rats, and dogs, and human ordure are commingled. I have the privilege of crossing this stream several times each week, and I have often to shut both nose and eyes, whilst passing over the hissing, bubbling, steaming cesspool of impurities, in its appearance disgusting beyond all description. The factories built along its course employ about fifteen thousand individuals, a great number of whom pass and repass the stream several times each day;

besides this a considerable portion live in the densely-populated and much overcrowded dwellings immediately adjacent (indeed sometimes these houses overhang the stream); and during the night its disgusting emanations are sent into the very sleeping apartments. I have already mentioned that in order to secure a large quantity of water for the engine-room, the stream is dammed up, especially during the night; this causes the water to rise above the level of the mouths of the drains which open into it, consequently the water regurgitates into these drains, sending the effluvia up through the gratings in the streets, the gully-holes in the entries, and the jaw-boxes in the staircases. Can the stench from a seven-stalled stable, or the effluvia from a drain, or the emanations from a covered tank of rain water, be compared for a moment to the intense and copious exhalations which arise out of this stream from it leaves Camlachie till it reaches William Street, Calton, where it is lost to sight, being covered over as it passes through the Green, till it reaches the Clyde. This burn, let it be recollected, runs through one of the most densely-populated districts of Glasgow, and yet fever is not endemic. This is by no means a solitary instance of the existence of most intense putrid emanations, without the presence of typhoid fever. We have also the classic Molindinar. This slow, rolling stream, before it reaches the Cathedral, has received many contaminations; after this it is enriched by the drainage from the Cathedral burying-ground on the one side, and that of the Necropolis on the other; it then runs through a steep ravine till it reaches Drygate, receiving at every inch additional effete animal and vegetable matters. After it passes under Drygate, it remains uncovered except by lanes and houses till it reaches Duke Street, constantly receiving at every point large quantities of impurities of every kind. At Duke Street factories the water is used for condensing purposes, and sent back at a high temperature. The stream next winds its slow course along Burnside Street, a most fetid and unseemly, thick, dark-coloured fluid, till it reaches the College Park, its course through which is covered over. After it passes under Blackfriars' Street, it remains open unless when passing beneath either lanes or houses, till it reaches the Spoutmouth at Gallowgate. After this it is covered over till it arrives at the Clyde opposite the jail, where it forms a most conspicuous object in consequence of the disgusting matters which it pours into the river.* From it leaves the Necropolis till it reaches the Gallowgate, with the exception of the College Park, its banks are surrounded with some of the most closely built, the worst ventilated, the most overcrowded hovels in the city. To such an extent is this the case, that the buildings, or kennels, as they

* The authorities are now adopting, ostrich-like, measures to *hide* this offensive disgorgement, by causing it to pass into the river through pipes, placed below the level of the water.

have been called, have often been the subject of serious consideration to the local authorities. The Drygate, Burnside, the Vennels, Havannah, Dovehills, and Spoutmouth, are localities notorious for their wretched condition to every one acquainted with Glasgow. I ask, then, if typhoid fever has been produced among the members of a household by emanations from a bad drain at the bottom of a garden, or has resulted to a "dancing party" who supped over a "seven-stalled stable," and walked over a "tank of rain water;" would we not expect to find the disease raging every autumn at least along the course of the Molindinar? yet we find no such occurrence. When fever prevails epidemically, these parts of the city suffer severely from typhus, but we have few if any typhoid cases. Indeed, during the last six or seven years, fever of any kind has been rare.

In like manner I might take my readers to the celebrated Pinkston Burn, and part of the river Kelvin; the stench arising from putrifying matters being so disgustingly intense as to cause the passenger to turn aside. The visitors to the West-end Park are unfortunately too familiar with the fact; yet typhoid fever does not prevail in the densely-populated houses placed along these fetid streams.

These are only a few of the many instances to which I could refer to show that putrid emanations exist in the most intense form, without producing fever of any type. No doubt Dr. Murchison may say, "these are only negative facts—I have adduced positive ones, and one of the latter character is worth a host of the former." So it would if such a positive fact could be adduced; but this I maintain has never been done, and I hold that the very existence of such negative facts as I have brought forward, demands that the alleged positive facts should be divested of all dubiety before we are called upon to admit them. These negative facts demonstrate that the relation which putrid emanations are alleged to hold to fever, is very unlikely to be one of cause and effect. I repeat that, until it can be clearly established that putrid emanations without any other influence will produce fever, these negative facts authorize us to decline acknowledging the circumstances adduced by Dr. Murchison and others as facts at all. His "positive facts" are nothing more than inferences deduced from premises which are by no means settled questions; and though they were fully established, yet they would not justify his conclusions, unless supported by additional evidence of a very different character from any which he has brought forward.

We see, therefore, the unsound nature of his argument. He takes for granted that emanations from cesspools and decomposing matters originate typhoid, and that overcrowding and destitution produce typhus; and then infers that the poison from which the two forms of disease arise must be essentially non-identical in its

nature. Let the reader bear in mind that the circumstances to which Dr. Murchison appeals in support of his conclusion (that typhus and typhoid arise from separate poisons) have reference merely to the correctness of his premises. Though he could establish the origin of fever either from cesspool exhalations or from overcrowding, yet this would not justify his inference that there existed an essential difference in the poisons so originated. The poison might be the same, having its intensity merely modified by the difference in the circumstances under which it was produced. But we have seen that the facts adduced by Dr. Murchison will not even establish his premiss, viz., that fever originates from the alleged causes. We have found that the well-known fallacy of "non causa pro causa," or "post hoc ergo propter hoc," pervades his whole arguments; he mistakes a sign, or a mere accidental relation, for a cause. His reasonings remind me of the story of the rustic who argued that Tenterden Steeple caused Goodwin Sands. This forenoon the weather was fine, but the mercury in the barometer was falling; and this evening it rains and blows with violence. To ascribe this change of weather to the fall of the mercury would not be more illogical nor more absurd, than to ascribe the origin of the outbreaks of fever to the emanations from the cesspools, stables, bad drains, water-tanks, old grease-pots, street-sweepings, &c.*

From the numerous facts which I have adduced, it appears quite conclusive that such agencies, *per se*, do not appear to originate either form of fever, and that therefore their relation to the disease is not one of cause and effect. In the present state of our knowledge on the subject, we are only entitled to conclude that emanations from decomposing animal and vegetable substances, and overcrowding and destitution, exercise a depressing effect on the constitutions of those who are subjected to their influence, whereby fever and other epidemic diseases spread rapidly and extensively among such communities.

11. *Does either form of fever so invariably occur in the localities assigned by Dr. Murchison, as to justify the conclusion that typhoid exists only in the vicinity of emanations from cesspools, &c., and that typhus occurs only among the destitute and overcrowded?*

We have already examined some of the "array of facts" adduced to prove this opinion:—

1. The epidemic of Tewkesbury described by Dr. Beadle. Dr. Murchison states that the description of this outbreak leaves no doubt but that it was typhoid; we find, however, that Dr. Beadle described the disease as an epidemic of typhus, and nar-

* "Why, Sambo, how black you are?" said a gentleman to a negro waiter; "how the world did you get so black?" "Why, look a-here, massa, the reason am dis: de day dis chile was born dar was an eclipse." Sambo's logic is quite identical with that of Dr. Murchison.

rates ten cases under this designation. This fact I have already pointed out, and need not refer to it any further.

2. We have also seen that the fever, which prevailed at New and Old Lenton, as described by Mr. Taylor, and ascribed to cesspool emanations, was not typhoid but typhus. Mr. Taylor affirms that "most of us have witnessed cases, which have run into the worst stages of typhus."

3. Then we have seen, that in the Windsor epidemic of 1858, instead of the thirty-four cases quoted from the register of deaths, presenting all the characteristics of typhoid, "that the majority were genuine cases of typhus." It is obvious that some serious error regarding the character of these cases must have been committed, either by Dr. Murchison or by the medical men who signed the certificates of death. Dr. Murchison states that he had "abundant opportunities of verifying these observations for himself," and he designates the thirty-four instances as well-marked examples of typhoid; but the medical attendants have, on the contrary, denominated six only as typhoid, and the majority of the others, either as simple typhus, or this disease along with its usual complications.

This is a most serious discrepancy, and we have seen that the register affords internal evidence of the correctness of the returns. How can these contradictory statements be explained? It appears to me that Dr. Murchison may have seen some cases of genuine typhoid; and that, in consequence of the detection of stagnant drains, filthy cesspools, &c., he came to the conclusion that all the thirty-four cases of fever, "although entered under different names in the district register, must have been typhoid." This mode of settling the disputed question is more commendable for its originality than its philosophy.

4. It is in this manner, however, that Dr. Murchison proceeds in his "array of evidence." Whenever he finds instances of fever associated with putrid emanations, he at once assumes that the disease was typhoid. For example, he quotes as follows from Dr. Southwood Smith:—"I have been struck with the number of cases of fever in houses opposite gully-holes." Dr. Murchison remarks that "although Dr. Smith says nothing as to the nature of the fever, yet there can be little doubt that the cases he alluded to were typhoid." Let the reader try to imagine the proof which he gives of this assertion; it is neither more nor less than "from what has already appeared in the course of this paper."* This is a very common specimen of the kind of logic used by the other advocates of non-identity, as well as by Dr. Murchison.

5. In the disease which followed the race-ball at Cowthorpe, we have an example of the same peculiar mode of reasoning. Dr. Murchison asserts that the affection was genuine typhoid;

* Medico-Chir. Trans., vol. xli., p. 265.

but we have shown that Dr. Camps stated in his paper, that from the most trustworthy accounts which he received, the disease was "malignant typhus."

In reference to the preceding instances, I beg to observe in passing, that Dr. Murchison commits several well-known fallacies. He takes for granted that the fevers were produced by effluvia, arising under the several circumstances mentioned. Thus he is guilty of "begging the question;" this, however, he dexterously endeavours to keep out of view by suppressing the premiss in which the points in dispute are implied. For very obvious reasons, he uses other propositions in which these assumptions are contained. Thus he conceals the very questions at issue, when he is actually taking them for granted.

In all the instances to which I have alluded, he commits also the fallacy of "reference." He quotes just as much from the different writers as will make it appear that they conceived the disease to be typhoid, or that they described a disease which must have been of that form of fever. Whereas, if he had quoted from these writers at length and correctly, it would have been demonstrated that they neither denominated the fever as typhoid, nor described a disease similar to typhoid, but, on the other hand, designated the disease typhus; and that their description was quite characteristic of this form of the disease. Dr. Murchison seems to have considered that the larger number, if not the whole of his readers, would take for granted that his quotations were correct, and would never take the trouble of examining the original passages.

I regret to add that the foregoing fallacies pervade the whole of the numerous quotations and references, with the exception of one from Dr. Jenner. Strange enough, the experience of this able defender of the doctrine of non-identity is quite opposed to the views of Dr. Murchison.

6. Dr. Jenner states, "that out of sixty-five cases of fever, it is recorded of two only where effluvia from drains existed; the one was taken ill after working in a cellar, in which he had observed a most offensive odour. In the other case, the house was found to be filthy, and the inhabitants complained bitterly of the 'offensive sewer.'" Again, Dr. Jenner informs us in his essay published in the *Medico-Chirurgical Transactions*, vol. 33, p. 40, that—"I may observe that I have visited in a few instances the houses from which more than one individual affected with typhoid, or typhus fever, were brought to the hospital, without being able to detect any hygienic deficiencies in the condition of the people or in the localities themselves to modify the exciting cause;" thus evidently implying that the exciting cause had no connection with localities.

Thus we find that the experience of Dr. Jenner is quite opposed to the theories enunciated by Dr. Murchison as to typhoid being

the result of putrid emanations, and typhus being the effect of overcrowding and destitution.

7. Dr. W. T. Gairdner, a recent convert to the doctrine of non-identity, mentions a case of two families residing in Milne's Court, Lawn Market, Edinburgh, in which typhoid fever broke out and attacked several members, one of whom died. The doctor visited both houses, and he tells us they were rather comfortable, well placed, well ventilated, though rather too crowded; high above the level of the street, and far removed from cesspools, common sewers, untrapped drains, or any of the ordinary concomitants of what has been called filth fever. In one of the houses, however, he found "a serious flaw." "Though free from all bad odour, and very clean to all appearance at the time I visited it, I found the roof of the room to be of the most flimsy construction, and in a state of great disrepair, and the painted canvas, which barely concealed the rafters, was at points saturated with liquid abominations which had soaked through above." Let it be recollected that this only applies to one of the houses, and that Dr. Gairdner afterwards alludes to the remarkable fact that no fever had occurred to the members of the filthy family who lived above—a fact which quite upsets the theory that the fever was caused by the filth. As emanations proceed upwards, the family above must have been much more exposed to the influence of "the liquid abominations" than those below. Dr. Gairdner, with his accustomed candour and straight-forwardness, admits that, with one or two exceptions, all the houses in which enteric fever originated were in tolerably well-aired and clean apartments, above the level of the street—facts quite fatal to the theories of Dr. Murchison; and proving that the typhoid form of fever often appears in localities where no trace of putrid emanations from sewers, cesspools, or graveyards can be detected.

We come next to the consideration of a new theory stated by Dr. Murchison. He alludes to an outbreak of fever in Dudley Street, Paddington, and tells us that Dr. Sanderson assured him "that there were some cases of typhus and typhoid, including the presence of the eruptions." (P. 276.) He also quotes the following important cases:—"A girl, aged seventeen, was admitted into the London fever hospital, from 17 Windmill Row, Lambeth—ill a week. Her body was covered with an unmistakable mulberry rush, and she presented all the usual symptoms of typhus. . . . Two days after, the symptoms underwent a complete change. The mulberry rush faded, and was succeeded by red spots, which came out in successive crops for more than a week, accompanied by diarrhœa and abdominal tenderness."—*Med. Chir. Trans.*, vol. xli., p. 276. Lastly, Dr. Murchison refers to an outbreak of fever which prevailed in the gaol at Rheims in 1840; and described by M. Landouzy in the 13th vol. 3rd series, of

*Archives Generales*, p. 7. Dr. Murchison admits that "many of the symptoms during life were those of typhus, but the intestines after death presented the lesions characteristic of typhoid."

Such are the facts—facts which, undoubtedly, tend to establish identity of origin of the two forms of fever, clearly indicating that both forms arise under the same circumstances, and that the symptoms are often convertible from those of the one to those of the other species.

To Dr. Murchison belongs the credit of propounding a new theory, by which the logical teachings of such facts are quite perverted, and the profession flattered into the belief of the existence of a "plurality of poisons" in such cases. In other words, he explains away these instances on the hypothesis that the patients were exposed to filthy emanations on the one hand, and overcrowding on the other; and hence they became infected with the poisons simultaneously, and after the typhus poison had exhausted its influence, the typhoid then took up the morbid warfare. In the Rheims gaol, he states, "That the fever was really typhoid is proved by the lesions found after death. On the other hand, a cause was not wanting to account for the symptoms of typhus during life." Thus, the patients had typhus during life, and after death it was proved that the affection was really typhoid. How self-contradictory—how truly unphilosophical are such suppositions. Dr. Murchison tells us that the disease was considered due to overcrowding; that the gaol was calculated to hold 130 to 150, but at the outbreak the number of inmates was about 190. This circumstance would give the product typhus; but as the intestinal condition after death was that of typhoid, Dr. Murchison has *discovered* a circumstance which he imagines will uphold the flimsy fabric of his doctrines, viz., "a disagreeable odour in the gaol proceeding from the grease of the woollen fabrics manufactured by the prisoners. Hence "the really typhoid character of the disease." What will Dr. Simpson say to this? The learned Professor has published a most elaborate paper to show that the grease used in woollen manufactures is not only innocuous, but most beneficial to the health of the workers; and he quotes largely in support of said views from the experience of medical men familiar with the works at Hawick, Galashiels, Glasgow, and other places at which woollen manufactures exist. Amongst other valuable hygienic influences which the Professor and his correspondents ascribe to the inhalation of the odours, is an immunity from epidemic disease. If the greasy exhalations could produce typhoid, why the workers in certain departments of the woollen manufacture would never be free from it. Now, the fact is, that very few of such people have either typhoid or any other form of fever. I beg to refer the reader for further information on this point to the *Edinburgh Medical Journal*, October, 1853.

But let us examine the account given of the Rheims fever by M. Landouzy himself, and contrast it with the version which Dr. Murchison gives. Dr. Murchison tells us that the outbreak occurred during autumn of 1840. M. Landouzy states the very opposite. He informs us that the first case occurred on the 2d October, 1839. He then gives us a monthly return of the number of cases and deaths, thus:—

| | Cases. | Deaths. | | Cases. | Deaths. |
|---|---|---|---|---|---|
| October, 1839, | 32 | 3 | January, 1840, | 12 | 1 |
| November, " | 45 | 1 | February, " | 14 | 3 |
| December, " | 20 | 3 | March, " | 11 | 4 |
| | | | April, " | 14 | 1 |
| | | | May, " | 0 | 0 |
| | | | June, " | 0 | 1 |

So that, instead of being an autumnal fever of 1840, it was in reality a winter and spring fever of 1839–40. It is obvious, however, that autumn would have suited Dr. Murchison's theory better than either winter or spring; but "facts are stubborn chiels that winna ding." The reader should note this additional fallacy of "reference." Let us next advert to the cause of the fever. M. Landouzy, after a most careful account of the hygienic conditions of the prisoners, the meteorological and atmospherical states, &c., came to the conclusion that the disease was owing to overcrowding. His words are—"L'encombrement des prisons doit donc etre regardé comme la cause determinante de l'epidemie de Rheims."

With regard to the greasy odours, M. Landouzy informs us that the smell proceeded from the high temperature of the *oil* (not grease) used in the manufacture of woollen; but he ascribes no influence to it, as such disagreeable smell *always existed* about the workshops. His words are—"La prison est bien tenue et propre en general; cependant il y régne en tous temps, et surtout dans les parties qui avoisinent les ateliers, une odeur desagreable due au suint de la laine travaillée par les detenus, et a la haute temperature de l'huile necessaire a cette fabrication." Thus clearly proving that the fever could not arise from the oily smell which was *always* in existence.

Lastly, as to the character of the fever, M. Landouzy makes a most elaborate analysis of the symptoms during life, and the post-mortem appearances; and he comes to the conclusion that the outbreak was one of *genuine typhus*. I may remark that he is a supporter of the non-identity theory; therefore his evidence should have the more weight with Dr. Murchison. His concluding words are—"Quant a moi je le repete, sans vouloir prejuger la question d'identitie pour le typhus epidemique en general, je conclurai en disant qu'il existait entre *le typhus carcera de Rheims et la fievre typhoide* trop de ressemblance pour que ces deux affec-

tions ne soient pas regardées comme analogues, et trop de differences pour quelles soient regardées comme identiques." P. 330.

We therefore perceive that instead of the views of Dr. Murchison receiving any confirmation from the history of this outbreak of fever, they are completely overturned, and that his theory of the existence of two poisons is positively contradicted; and, besides this, we find that the version which he gives of the disease is quite inconsistent with the account furnished by its historian, M. Landouzy.

2d. With regard to the Dudley Street cases, Dr. Murchison tries to induce us to believe that two sources of poison existed here—one arising from overcrowding, and the other from "selections from the street sweepings, such as old *grease pots*, &c., materials sufficiently prone to decomposition in hot weather."—*Med. Chir. Trans.*, vol. xli., p. 277. With respect to the "&c.," I cannot offer an opinion as to the effects of heat in effecting their decomposition, but I contend that no such result could be produced on the old *grease pots!!!* The old *pots* afford truly a very *fragile* foundation on which to rest the theory of a pestiferous exhalation capable of originating typhoid fever.

3rd. Lastly, with regard to the girl from "Windmill Row," it is worthy of remark that the *mulberry rush* disappeared on the ninth day of the attack. This circumstance is a most unusual one in the typhus rush; indeed, I may say unprecedented. Was there no mistake here? May not the mulberry rush have been produced by the well-known companions of a hawker (which this girl was), viz., the "fleas?" Or is it not likely that the comforts of the hospital, by increasing the vital powers of the girl, had converted the typhus into typhoid spots? Either of these suppositions is much more tenable than the hypothesis of a double poison. Thus we find that Dr. Murchison has not adduced a single fact capable of affording the slightest foundation for his theory of a duality of poisons.

Let it be observed that Dr. Murchison does not deny the occurrence of typhus and typhoid fever in the same families, and in the same localities. Instead of following the example of Dr. Jenner and others in this respect, he admits the fact, and quotes the instances just referred to. But he denies the very obvious conclusion to which such facts point, and explains away their evidence by starting the theory of the coexistence of two poisons —one emanating either from cesspools or some other decomposing matters, and the other arising from overcrowding or contagion.

It must be obvious to the reflective reader that this theory is established on the same fallacy on which his other conclusions are based, viz., "begging the question." No doubt the premises in which the assumptions are implied, are still cautiously kept out of view. It is evident that, in the first place, he takes it for

granted that the two forms of fever are non-identical, and reasons thus:—As certain non-identical diseases, such as scarlet fever and variola, have different foci of infection, therefore typhoid and typhus fever must have different foci of infection, because they are non-identical diseases. Secondly, those instances of the two forms of fever occurring in the same family, and in the same localities (and which facts contradict the theory of two foci of infection), he explains by assuming as proved the very point in dispute, namely, the existence of two foci of infection; and asserts that in such mixed instances the two foci or poisons must have existed. Having taken this for granted, he sets about to discover, on the one hand, exhalations, so as to account for typhoid, and, on the other, overcrowding, in order to explain the typhus; and when gully-holes, stinking sewers, uncleaned privies, putrid drains, fail him, he finds consolation in "stable lofts," "water tanks," and the decomposition of "old grease pots."

I have examined at some length the statements and theories advanced by Drs. Jenner and Murchison, because much importance has been attached to them, both by the writers themselves and by their disciples, on account of the alleged support which, it is considered, such theories and statements afford in favour of non-identity, especially in setting aside the evidence in favour of a community of origin which is furnished by the undoubted facts to which I have referred.

I trust that I have proved to the satisfaction of every candid inquirer that both these writers have most signally failed in their object; that their arguments consist of a series of fallacies; that their references are incorrectly quoted; and that their conclusions are untenable. Therefore, the facts which I have brought forward from the experience of Stokes, Huss, and from my own observation, will not admit of any of the explanations suggested by the theories of Drs. Jenner and Murchison, but remain as cogent teachers of the community of origin in both forms of fever.

This is still further confirmed by the following considerations, which have been kept out of view by the advocates of non-identity:—

III. *What is the essential difference between the conditions of those who are exposed to exhalations from cesspools, &c., and those who are subjected to overcrowding and bad ventilation?*

It appears to me that it would be much more philosophic, if the advocates of the theory of different foci of infection would try to determine by chemical analysis the differences which exist between the effluvia proceeding from sewers, privies, cesspools, and those arising from the bodies of human beings, when overcrowded and living in badly-ventilated dwellings, than to use their energies in vain endeavours to uphold their theories by such a series of fallacies as those which I have pointed out. Until they bring before us some such analysis, I beg to direct attention

to the important fact which they seem to have forgotten; namely, that we never have destitute people, crowded together in badly-ventilated apartments, without filth and decomposing animal and vegetable remains being also present. We may have exhalations from decomposing organic substances without the coexistence of poverty, bad ventilation, or overcrowding; but we will fail to discover poverty, overcrowding, and bad ventilation without filthy accumulations. It is at once conceded that an important difference does exist in the condition of a community exposed simply to fetid exhalations, and another subjected to the evils of destitution and overcrowding. The difference is, however, simply one of degree. The effect of either agency is to lower the tone of the energies of the system; and when several or all are combined, we must have the effect much aggravated. When disease of an epidemic character visits a rural district in which filthy abominations only exist, the type will be much less malignant, than when the same disease breaks out in communities labouring under the conjoint influences of destitution, overcrowding, bad ventilation, and the effluvia resulting from decomposing matters. In the former localities fever will assume the typhoid, but in the latter it will present the typhus character. The one class of people have only experienced the depressing effect of one debilitating agent, viz., effluvia from decompositions; the other, besides being exposed to such exhalations, are simultaneously subjected to several other exhausting influences. The first predisposing cause may exist alone in the former places; but it always forms an integral part of the latter. Hence, why it is that fever generally presents the typhoid type in rural districts and among the young and opulent, and assumes the typhus form when it attacks the destitute denizens of overcrowded and badly-ventilated buildings, or the old and infirm.

The facts which the history of fever furnishes, fairly justify this opinion—an opinion which I am inclined to hold, until some of the advocates of the non-identity theory succeed in demonstrating an essential difference in the constitution of the atmosphere which is breathed by those who live at the mouth of gully-holes, sewers, &c., and that inhaled by the people who are huddled together in badly-ventilated apartments. These dissimilar conditions, therefore, act only as predisposing causes; they merely modify the influence of the poison of fever, just in the same way as they powerfully modify the manifestations of the exciting causes of every other epidemic disease. We see the very same circumstances occurring in the outbreaks of cholera, scarlatina, rubeola, variola, &c. Such dissimilarities in the virulence of these diseases surely cannot authorize the notion of the coexistence of a plurality of exciting causes. The morbid poison is essentially the same, both in the mild and the malignant cases of these respective diseases; but its effects are modified by

the amount absorbed, and by the condition of the constitution of the person into whose system it has gained access.

In conclusion, I have to examine very briefly another argument which has been brought forward by almost every advocate of the non-identity theory, to prove that typhoid and typhus fever must arise from totally distinct causes:—

1. It is alleged that patients labouring under typhus, when admitted into a fever hospital, have been attacked with the typhoid during their convalescence.

2. It is asserted that patients who have passed through the typhoid form have been attacked, after partial or complete recovery, with typhus. From these circumstances, the conclusion is deduced that the two forms of fever must arise from different poisons, and consequently are non-identical in their origin.

A careful examination into the circumstances connected with such cases will at once show that the above conclusion is quite illogical, that the real facts connected with such instances clearly indicate a community of origin. In support of this statement, I beg to refer to a case reported by Dr. Corrigan in his able lectures on fever. I may remark he is an advocate of non-identity, and therefore his opinions should have great influence with those who take the same side of the question :—

"Mary Cope, aged twenty-two, previously in the enjoyment of the best health, was admitted into the Hardwicke fever hospital on the 23rd January, ill of maculated fever. There was nothing unusual in her case. She was soon convalescent, when, on the 5th February, fourteen days before her death, she complained of debility and of diarrhœa; her tongue became brown and dry in centre, but not furred; there was no tenderness of abdomen nor tympanitis, but there was gargouillement over the cœcum; and the stools were like gruel, but neither mucous nor bloody, nor was there any tenesmus. The pulse became quicker and weaker. On the 18th February she required wine in considerable quantity; the diarrhœa became uncontrollable. On the 19th February, fourteenth day of her attack, she died."

*Post-mortem Examination.*—The peritoneal covering of the abdominal viscera was sound; but the ileum and a portion of the colon presented follicular enteritis in all its stages. The greatest intensity was at the ileo-cœcal valve, the entire circle of which was occupied by a depressed, jagged, greyish, irregular ulcer. In the ileum both the isolated follicles and the "glandulæ agminatæ," or glands of Peyer, were attached. The sites of the affected glandulæ agminatæ were marked by oval ulcers, while around and above them isolated follicles were seen in similar ulcerations, but not to such a degree. In addition to these ulcerations, the isolated follicles in both the lower portion of the ileum and the upper portion of the colon presented every stage of the disease. Some follicles were just protruding under the

mucous membrane filled with a cheesy-looking purulent matter, the mucous membrane around being swollen, red, and prominent; and from the orifices of the follicles looking like depressions; these follicles bore a strong resemblance to variolous pustules. In others the matter was in such quantity, that the affected follicles presented the appearance of spherical projections attached by pedicles, and covered by mucous membrane, hard and firm to the touch; while, in others still more advanced, the follicles had gone on to ulceration, destroying the mucous membrane, and leaving only a grey slough of cellular tissue in place of the follicle itself," &c.

Such then is an example of the instances which are brought forward to prove that typhus and typhoid must be dissimilar diseases. It is taken for granted that this girl recovered from typhus, and then, during convalescence, was attacked with typhoid, under which she succumbed.

A careful examination of the circumstances of her case will prove the very reverse :—

1*st*—She was admitted on the 23rd January, ill with typhus—how long is not mentioned; but the disease must have been advanced, as we are informed she was soon marked convalescent; but, on the 5th February, she was attacked with typhoid; that is, on the 12th day after admission. She could, therefore, have only been three or four days convalescent. She dies on the 19th February. That is about the 26th day after admission, or on the 14th day of the typhoid disease. We next come to the post-mortem examination, the results of which are perfectly inconsistent with the appearances which usually are detected on the 14th day of typhoid fever. The best pathologists, as we have pointed out in a former number of this Journal, all unite in asserting, that at the end of the second week, Peyer's glands are only slightly ulcerated, and that the ulcer at that period only exists in the primary form; but, in Dr. Corrigan's case, we find the ileo-cæcal valve was surrounded by a deep, jagged ulcer, and that many other deep ulcerations existed; in fact, the state of the intestines exactly corresponds with the condition which is usually met with in typhoid cases at the end of the fourth week, the time which in reality the girl's illness had lasted. The post-mortem appearances, I repeat, clearly prove that the case could not have been of fourteen days' duration. The condition of the intestines unquestionably proves that the disease must have been progressing throughout her whole illness, and that the convalescence marked the subsidence of the action of the fever-poison on the general system; but that with this subsidence the local disease did not cease, but slowly progressed until the primary ulcers had extended and excited secondary inflammation; and, hence, on the 5th of February secondary constitutional and local disturbance made their appearance. I have met with many similar cases; the patients

had all the symptoms of maculated typhus, became convalescent, but after a few days, usually three or four, febrile symptoms reappeared, either attended with, or soon succeeded by abdominal symptoms. The true explanation of such cases is simply this, that the fever-poison having exhausted itself about the usual period, the constitutional disturbance ceases, the patient seems to be quite convalescent; but unfortunately the state of the "glandulæ agminatæ" has not improved; instead of the primary ulceration cicatrizing, the ulceration extends, and soon involves the adjacent mucous membrane and other tissues. Very soon febrile symptoms reappear, along with diarrhœa and other abdominal symptoms. But I contend that, in such instances, we have not two fevers, but one fever. Instead, therefore, of affording any support in favour of the theory of non-identity, such cases furnish a most convincing proof of the identity of the two forms of fever. They show that, in the cases of well-marked typhus, Peyer's glands are involved, but often give no indication of being diseased, until the constitutional effects of the poison are thrown off, and then afterwards rapidly proceed to extend and degenerate into the chronic ulcer. Such cases are common, and point out very cogently that it is the duty of the physicians carefully to attend to the condition of the intestinal mucous membrane in all cases of fever, whether typhoid or typhus.

With regard to those cases, in which it is made to appear that typhoid preceded the typhus attack, such instances will be found abundantly recorded in the writings of the greater portion of the non-identity advocates. At first sight it seems a very formidable argument which is deduced from these occurrences.

Patients are admitted labouring under typhoid symptoms; they recover more or less (some are even dismissed), and are then seized with typhus. It is affirmed that they must have got the typhus-poison in the hospital; and that, ergo, typhus and typhoid fever must be non-identical.

But I feel assured that a careful study of such cases will soon dispel the force of this argument, and show that the first attacks were not cases of typhoid fever, but examples of simple inflammation of the mucous membrane of the small intestine. I have seen many such cases admitted into the Glasgow Royal Infirmary; some few took typhus, but the great majority escaped; and I have read a great many reported cases, but I can point to facts in their history which will show satisfactorily that no typhoid existed, but simple muco-enteritis, arising from non-specific causes. In every instance which I have witnessed of such cases being followed by typhus, and in every properly recorded case which I have read, the abdominal disturbance either preceded the febrile symptoms or occurred simultaneously with them. This is a most important circumstance in reference to the true nature of such cases. In genuine typhoid fever we find the very reverse taking

place. The rigor and the febrile symptoms precede by several days the abdominal manifestations.

Therefore, those instances in which diarrhœa, abdominal pain, &c., exist prior to, or commence with the pyrexial symptoms, cannot be accepted as genuine cases of fever. Indeed, there are examples of simple muco-enteritis, either arising from exposure to cold or from the use of dietetic articles, which have irritated and inflamed the intestinal mucous membrane. It would be an easy matter to quote scores of such cases, described under the name of "gastric," "enteric," "follicular," and "typhoid fever;" but it must be obvious, on the slightest reflection, that a grave mistake in diagnosis must have been perpetrated, as it is quite opposed to the usual order in the essential pathological conditions characteristic of the progress of fever. We have seen at an early portion of these papers, that the intestinal lesion is acknowledged by all modern writers to be the effect of the fever poison, and not the cause of the fever; and that, consequently, this local manifestation does not take place either prior to, or simultaneously with, but at a period considerably subsequent to the development of the pyrexial symptoms. It is nothing, therefore, extraordinary, if patients labouring under this pseudo-febrile disease—this local affection—be exposed to the contagion of typhus, that they should take the disease. This, I repeat, I have seen occur more than once, but I have never yet seen an instance of a patient who had passed through an attack of genuine typhoid, becoming attacked with the typhus.

I contend that cases in which the abdominal manifestations precede the febrile disturbance should not be considered as examples of typhoid fever, but as instances of non-specific muco-enteritis, or inflammation of the mucous membrane of the intestines, and distinguished from typhoid by the circumstance that in this latter affection the constitutional disturbance precedes the abdominal symptoms. In the one, the local manifestations have a prior existence to the general disorder; in the other, the general disturbance is followed by the local symptoms. When a patient labouring under this simple non-specific affection of the mucous membrane is admitted into a fever ward, if he has never had fever, the chances are that he will take the disease, and will present the symptoms of typhus, from the circumstance of his exhausted condition when exposed to the contagion. I repeat, that I have seen this occur occasionally, but no argument can be deduced in favour of non-identity from such occurrences, because these cases were not originally instances of genuine typhoid fever. The alleged cases which have been published are liable to the grave objection, that the diarrhœa commenced either prior to, or along with the febrile symptoms, and consequently are of no value in reference to the question in dispute. At all events such equivocal cases afford no foundation for the argument

deduced by the advocates of non-identity in order to set aside the conclusion pointed out by such positive facts as I have brought forward in this paper. But to conclude—

1. In the last number of this Journal it was satisfactorily established that both forms of fever were essentially identical in their symptoms, at least so far as the term identical is applicable to the symptoms of disease. (In a strictly philosophical sense this term cannot be applied to the phenomena of any disease, because these phenomena exhibit always more or less variation in different persons, and at different times in the same individual.)

2. It was shown in the same paper that the intestinal lesions in both forms of fever were essentially the same, differing merely in the degree of intensity and extent.

3. In the present paper, I flatter myself that the facts adduced prove unquestionably that the two forms of fever have a common origin, and that the objections which have been brought forward, not against the facts let it be remembered, but against this conclusion, are perfectly untenable, founded on obvious fallacies, bold assumptions, and misrepresentations.

The similarity of the symptoms during life, the essential resemblance of the intestinal lesions after death, and the community of origin, form the threefold basis on which I am content to support the doctrine of the identity of typhus and typhoid fever; a foundation capable of resisting both the plausible assaults of the sophist, and the fanciful attacks of the speculative theorist. Their clamour may tend to confuse and obscure for a time, but ere long light will break through and exhibit the banner unsullied—"Magna est veritas, et prævalebit."

---

## CLINICAL RECORD & PATHOLOGICAL REGISTER.

COLLECTED AND ARRANGED BY JAMES M'GHIE, M.D.

### III. AMPUTATIONS.

#### 1. Amputation of Thigh.

*Case* XVII. (Under the care of Dr. Andrew Buchanan. Reported by Robert B. Gilland, M.D.)—M.M'M., aged 6, was admitted into ward 8 of the Glasgow Royal Infirmary, on the 21st of August, 1860. About four years ago patient's left knee-joint without apparent cause became gradually swollen, stiff, and painful. Two years previous to admission, a small abscess formed upon the posterior and outer aspect of the joint, which burst and discharged a quantity of thin serous fluid. Several small abscesses

of a similar character formed afterwards on the anterior and superior aspect of right knee-joint.

On admission, patient's right knee-joint is equally enlarged anteriorly, the swelling being firm, and spherical, and most distinctly marked on the inner aspect. The integument is smooth, glistening, and of a deep red colour. The muscles of right leg are much emaciated, and the leg is immovably flexed upon the thigh. Any motion of bones composing articulation is attended with very severe pain. A chain of small, circular, strumous ulcers extend across the anterior and upper margin of the swelling from the inner to the outer aspect of the joint. They are the sequences of the abscesses already described. In the centre of two of the ulcers openings exist, into which a probe may be passed for an inch and a half; but no bare bone is detected. Patient's general health tolerably good; bowels regular.

*Sep.* 1*st*—A consultation was held to-day upon the case, when amputation of the thigh was unanimously recommended.

7*th*—Amputation of the thigh at the lower third was performed to-day, patient being under chloroform. The method of operation was by anterior and posterior flaps of equal length.

18*th*—Stitches removed to-day; edges of wound in opposition and adhering.

21*st*—Doing well.

*Case* XVIII. (Under the care of Dr. Eben Watson. Reported by Dr. W. Sloan).—J. T., tobacco-spinner, aged 19, was admitted into ward 5 of the Glasgow Royal Infirmary, on the 17th July, 1860. Three months previous to admission, from the constant walking to which he was subjected, his left thigh commenced to swell and feel painful. The swelling was situated on the lower two-thirds of the femur, which gradually enlarged and became daily more painful, until he was obliged to keep to bed. At this period an opening being made by a surgeon on the outer aspect of the thigh, a large quantity of bloody purulent matter was discharged to his great relief; and his health improving he was able again to walk about after two months' confinement. A week previous to admission, he accidentally twisted his leg, while sitting on a chair, and he felt something give way with a snap. He found, then, that he was unable to walk, and the thigh admitted of being bent upon itself at a point situated about four inches above the knee.

On admission the whole of the lower three-fourths of the femur is found to be the seat of a tumour, this portion of the thigh measuring four inches more in circumference than at the corresponding portion of its fellow. The thigh can be bent upon itself slightly at the thickest portion of the tumour. A probe introduced into the opening which still exists, impinges upon bare bone. The knee fixed in the straight position. Suffers no pain when the limb is undisturbed. General health pretty good.