**Occasional papers on medical subjects, 1855-1896 / by W. Howship Dickinson.**

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BY

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# PREFACE.

The papers which have been brought together in this volume were written as opportunity occurred during the course of the last forty years. They touch upon many subjects, and offer a variety which should prevent their being uniformly uninteresting. To avoid needless repetition I have excluded for the most part those of which the chief matter has been incorporated in subsequent works. I have selected for republication some which purported to contain new observations, however slight, and others which had the object of presenting old conclusions for new considerations. A few of the later papers were put together with the design of their ultimately finding place in a collection such as this, while the last two have not before appeared in their present shape. The general system of arrangement has been chronological.

Scattered as these *opuscula* have been among Transactions and periodicals, it is probable that some may have almost entirely escaped notice, and may present themselves even now with an air of novelty. For the use of those who have not read them and may care to do so, if such there be, I venture to present them in the form of a book.

9 Chesterfield Street, Mayfair.

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# LIST OF ILLUSTRATIONS.

## *ON THE ACTION OF DIGITALIS UPON THE UTERUS.*[1]

Last October, in the Burton[2] Ward of St George's, a case of most severe menorrhagia, which had nearly proved fatal, was cured by the infusion of digitalis, administered almost by accident. The circumstances are sufficiently striking to deserve a full account of the case.

Catharine Mackenzie, aged 48, a widow, was admitted on the 18th of September, 1854. She stated that her health had been good, and that the uterine functions had been performed naturally, until twelve months previously, when she fell from a ladder, about twelve feet high, and got considerably bruised about the lower part of the back. A week after this blood began to flow from the vagina; at first in small quantities, but it soon became profuse and accompanied with pain; and so it continued, without intermitting for a single day, until her admission.

She was first treated in another department of the hospital without benefit; and when she was transferred to Dr. Lee, her case was regarded in the most unfavourable light.

She was perfectly blanched, and was scarcely able to stand, on account of weakness and vertigo. There was a discharge of blood from the vagina, so copious that none of the ordinary appliances were sufficient to keep it within bounds; it poured into the bed; and during micturition it passed from the vagina in gushes. There was slight pain about the sacrum. The ankles were œdematous; the tongue flabby and rather coated; the pulse 80, not very unnatural. On examination, the uterus was found in a perfectly healthy condition, though slightly anteverted; the os was quite soft and smooth, and there was no organic disease of any kind within reach.

Nothing was made out which could give any particular direc-

[1] 1855. *Medico-Chirurgical Transacti ns*, vol. xxxix.

[2] The Obstetric Ward.

tion to the treatment. Sulphuric acid and sulphate of magnesia were given, but with no effect, except upon the bowels.

Three days after this the patient complained of pain in the region of the heart; and said that during the night she had had several attacks of shortness of breath, in which she thought she should have died. On examining the chest, the signs of pericarditis were thought to be found, and the patient was referred to Dr. Nairne.

On the 28th of September all acute symptoms had subsided, and the only unnatural sound in the chest was a regurgitant aortic murmur. The hemorrhage was more profuse than ever, and the patient was extremely low. Four grains of acetate of lead, with half a grain of opium, were given three times a day, and wine was ordered.

On the 2nd of October a violent attack of purging and vomiting, with much coldness of surface, made a change of treatment necessary. The discharge was not in the least diminished. On the following day medicine was given containing sulphuric acid and chloric ether. This seemed to do rather harm than good; and, on the 5th, the acetate of lead was recommenced, with the same result as before. Sulphuric acid was again given, conjoined with ethereal and alcoholic stimulants, which had now become indispensable, on account of the excessive faintness and weakness of pulse which had resulted from the loss of blood. On the 9th, tannic acid was prescribed, but it proved as ineffectual as the previous medicines.

The condition of the patient was now hazardous in the extreme. She was evidently sinking. The blood which escaped during micturition was apparently nearly as copious as the urine, and enough passed into the bed to make it necessary to change the sheets several times a day. There was a loud diastolic murmur heard all over the central portion of the heart; and the pulse was 94, jerking and extremely weak. Another vaginal examination was instituted, but no additional light was thrown upon the source of the hemorrhage. With a vague idea that the cardiac derangement might, in some way, lie at the root of the evil, a trial of digitalis occurred to me. In such a case any chance was worth seizing; and Dr. Lee consented to the experiment. On the 10th the medicine was commenced; half an ounce of the infusion being given three times a day. On the morrow the improvement was remarkable. The discharge was comparatively

scanty; the appetite had returned; and the patient expressed herself immensely relieved. During the night the discharge totally and finally ceased.

From this time the improvement was most rapid. On the 13th the digitalis was exchanged for iron; and on the 31st the patient left the hospital, tolerably strong, and declaring herself perfectly well. And there is reason to believe that she so remains; for she has never come back to the hospital, though she promised to do so at once in case of a relapse.

It now became a question whether this sudden discontinuance of the hemorrhage was to be regarded as a mere accidental coincidence, or was to be attributed to the action of the digitalis. And supposing that the change was in consequence of the medicine, it remained to be discovered in what way the medicine operated; whether directly or indirectly; whether upon the general circulation first and secondarily upon the uterine; or whether upon the uterus especially, in some manner hitherto unexplained. It is the object of this paper to answer these inquiries.

Since the occurrence of the aforesaid case patients have been admitted into the Burton Ward labouring under menorrhagia, arising from different causes; in age they have varied from 19 to 64; and in other particulars they have differed as widely. Many of the cases have been of a kind to get well without much medical interference; these have always been allowed to do so. In every case requiring active remedies, admitted since October, 1854, the administration of digitalis has constituted the sole treatment. The discharge has invariably been extinguished; the time of its cessation varying with the strength of the medicine. In cases where the medicine was given in the largest doses, the discharge has not appeared after the second day; where the remedy has been given in a more dilute form, the hemorrhage has never outlasted the fourth.

The effects which follow the administration of a large dose —an ounce and a half of the infusion, for instance—are such as to establish beyond question the powerful influence of digitalis upon the uterus. A few minutes after the draught is swallowed, the patient complains of acute pains in the

back and hypogastrium, which she compares to the pains of the first stage of labour; a quantity of blood, solid and fluid, is then ejected; and the discharge is absent until, after several hours, the pain subsides, and the bleeding returns with diminished force. After each repetition of the remedy, the cessation becomes longer, and the recommencement more feeble, until after three or four doses the discharge ceases.

It is not my purpose at present to dwell upon the *modus operandi* of the drug; I merely wish to show that its effects are immediate and well marked. When a morbid action gradually subsides, after a course of treatment, the amelioration may be quite independent of the medicine swallowed; but when each draught is followed by a temporary removal of the disease, we have no choice but to admit that the recovery is in consequence of the treatment. A case may be here introduced, which will serve to illustrate some of the preceding statements.

A married woman, named Mary Lee, 37 years of age, the mother of a large family, was admitted on the 18th of January. She gave the following account of the progress of her disorder:

"Ever since the last labour, which occurred nine years ago, the menses have been profuse, and mostly mixed with coagula. Eleven months ago, after much hard work, with insufficient food, the hemorrhagic discharge became nearly continual: and she came to St. George's, at first as out-, latterly as in-patient, undergoing treatment for four months without much temporary or any permanent advantage. Short irregular intervals have occurred until within the last eight weeks, during which period the discharge has been excessively copious, and has not intermitted for a single day."

When she was admitted she was in a state of the most extreme anæmia; the heart's beats were very feeble, but without murmur; the pulse was 70, and extremely weak and small. The discharge was constantly flowing into the bed; besides which, whenever urine was evacuated, great quantities of blood gushed from the vagina. The quantity of sanguineous fluid thus voided was estimated at four pints in the day and night; of this a great

portion was urine; but it seemed that about a quarter of the amount was blood.

Half an ounce of the infusion of digitalis was now ordered three times a day, in an ounce of water, commencing on the 18th. On the morning of the 22nd, the discharge ceased somewhat suddenly. No other effect was observed from the medicine; the pulse was reduced only two beats: it remained quite regular, and of about the same character as before. On the 24th, the medicine caused vomiting, and the pulse became reduced from 68 to 60, still remaining regular. Whereupon the digitalis was exchanged for a solution of citrate of iron.

The patient progressed favourably until the 12th of February, when a little sanguineous discharge reappeared, which was merely supposed to indicate the return of the monthly period. But on the following day the patient was fainting with loss of blood, which was pouring out with nearly its former profusion; the pulse then numbering 72. The infusion was now given at the rate of an ounce and a half three times a day. The effect of the first dose was to cause pain, which supervened in about five minutes, chiefly referred to the back, but also affecting the lower part of the belly, and said accurately to resemble pains in the first stage of labour. This lasted acutely for about half an hour, and then gradually diminished, not entirely disappearing for some hours. The accession of the pain was immediately followed by the forcible expulsion of a quantity of blood, mostly coagulated; after which the bleeding ceased, and remained absent so long as the pain endured.

Except that each recurrence of the discharge was scantier than the preceding, the same phenomena occurred after each repetition of the medicine, until the fifth dose, after which no blood was passed. The medicine was continued until the 18th, when the patient was attacked with vomiting, looseness of the bowels, and a sensation of heat about the mouth and throat; and the pulse, which had been gradually increasing in rate, was found to have reached 92. The unpleasant symptoms presently subsided; iron was prescribed; and the patient speedily acquired a condition of health to which she had been long a stranger.

A vaginal examination was now made, and the uterus was found in a perfectly natural condition. There was a little thickening of the os, such as is generally observed in women who

have borne many children. It is to be regretted that circumstances prevented an examination immediately before the administration of the digitalis; but perhaps it would not be too much to assume that the uterus was then in the same state as it was some months previously, when the symptoms were much the same. The body and neck were then so distended, and the os so open, that a fibrous tumour was suspected to exist.

After a due interval the natural catamenial discharge appeared, no coagula were passed, and the evacuation was scanty, and lasted but two days. The patient then left the hospital in a state of perfect health, and resumed her former laborious occupation. She was seen very recently (September 17th); she was perfectly well, and the menses had never shown the slightest tendency to transgress their natural limits.

It is not necessary to dwell longer upon individual cases; it is enough to repeat that, so far as regards a speedy cessation of the discharge, the result has been the same in *every* case subjected to the treatment. I should here mention that the digitalis has always been given in company with wine and good diet. Owing to the kindness of Dr. Barclay, I am enabled to confirm my assertions by the results of his practice. Dr. Barclay writes, 'The cases in which I have employed it' (*i.e.* the digitalis) 'were such as seemed to me to be instances simply of functional derangement, which did not require the interference of the accoucheur; and I can say with confidence, that this mode of treatment has been more satisfactory in its results than any other I have hitherto adopted. Cases, indeed, have not been wanting which have resisted the influence of tonic and astringent remedies, which have yielded readily to this.'

Before quitting the first part of my subject, I may allude to the results of a few experiments upon the sanguineous discharge resulting from organic disease of the uterus. It seems that in these cases the remedy acts with somewhat less certainty than in the preceding, a larger dose is required, and the effect is sometimes transient.

In a case of polypus, where violent hemorrhages were apt to occur, one of the attacks was suddenly cut short, while at

its height, by the administration of an ounce of the infusion. The discharge ceased within an hour of the taking of the first dose, and never occurred again, although the tumour was allowed to remain untouched for three weeks. Where hemorrhage resulted from a fibrous tumour distending the uterus, the same dose produced the same effect, except that it was less permanent. In a case of advanced cancer of the os uteri, with continual and profuse sanguineous discharge, an ounce and a half of the infusion was found to cause total cessation of the hemorrhage within a quarter of an hour, the suspension generally lasting for about twelve hours. This sequence was observed as often as the experiment was made. In the same case it was found that half an ounce of the same preparation was perfectly ineffectual. Where severe attacks of bleeding depended upon rigidity of the uterus, with a roughened and tubercular state of the os, the hemorrhages were found to cease after five or six half-ounce doses. The advantage here was merely temporary.

Taking into consideration the preceding facts, I think we are justified in believing that digitalis cures, with more certainty than any remedy hitherto employed, cases of menorrhagia not connected with organic disease ; and that, where organic disease gives rise to the symptom, the action of the medicine is scarcely less manifest, although the advantage may be temporary.

I shall now consider the *manner* in which digitalis influences the uterus so as to produce these effects. The alteration must depend on some change, either in the local organism or in the general circulation. That the change is not in the latter is evident from the fact that hemorrhages from other parts of the body are not controlled like those from the uterus. Moreover, it was found, that on the cessation of the hemorrhage, the pulse was not uniformly slower or weaker than when the treatment was commenced. It was found to have become slower in only seven of the sixteen observations, and even then the

retardation was generally slight. Then as to the force of the circulation, the pulse was not found to have become perceptibly weaker, except in one solitary instance. Thus it seems that the peculiar uterine influence is quite independent of any change in the heart's action or general circulation. It must, therefore, depend on some change wrought by the medicine in the local organism.

As far as I know there are but two kinds of action which could be thus established in the uterus which would serve to account for the phenomena. Contraction of the capillary vessels, or the firm closing up of the uterus itself, might either of them prevent the effusion of blood from the lining membrane.

That constriction of the vessels is the change induced is a theory which cannot be upheld. Such an effect would probably not be confined to the uterus, but would be equally observed in other parts of the body. Hemorrhage from the lungs, bowels, and kidneys, would be amenable to the remedy. Besides this, it will be seen that there are certain particulars in the effect of the medicine which are not compatible with the supposition.

All the facts point to the conclusion that the digitalis acts by causing muscular action in the uterus itself. A short time, generally about ten minutes, after a considerable dose of the medicine is swallowed, the patient complains of severe pain in the region of the sacrum, which passes into the hypogastrium, and in every respect resembles the pain of the first stage of labour; very shortly after this a considerable quantity of blood, generally partly coagulated, is forced out of the uterus. No more discharge is then observed for several hours, so long as the contractile pain continues; and the same effect is noticed after each subsequent dose until the hemorrhage is abolished. In the case of Mary Lee, given at page 4, the succession of events was observed four times. Other cases have occurred in the hospital, in which the same effects were noticed.

A patient, named Ellen Morling, who was affected with very severe uterine hemorrhage, was ordered an ounce and

a half of the infusion three times a day. The first dose was taken in the evening; very shortly afterwards pain came on in the back, and along the inside of the left thigh, precisely resembling, in character and position, the pain of her last labour. Within fifteen minutes of the taking of the medicine, a clot, said to have been as large as an egg, was expelled, and the discharge totally ceased until the following morning, when it recommenced. Another dose was then administered, and the same results were observed to follow with more quickness; a mixture of solid and fluid blood had been expelled, and the discharge had totally ceased, within five minutes. The discharge reappeared a short time before the third dose was given in the afternoon. The effects were the same, but more immediate; scarcely any interval was said to have existed between the taking of the draught and the expulsion of the contents of the uterus and suspension of the discharge; the pain was more severe than it had previously been. On the following morning the discharge reappeared in very small quantity; the remedy was given in half-ounce doses, and the cure presently completed.

The chain of symptoms here observed—the labour-pains, the expulsion of the contents of the uterus, the suspension of the discharge, seem to be the precise effects which might be expected to result from forcible contraction of the uterus; and it is not easy to suggest any other explanation.

A striking confirmation of this theory was supplied by a case, which, at first sight, seemed contradictory. A patient, named Ann Cage, was brought into the Burton Ward, suffering from the symptoms of malignant disease of the uterus. She had a profuse sanguineous discharge. Hemorrhage, under similar circumstances, had previously been suspended by digitalis, and now the medicine was given in the same doses as before. An ounce and a half of the infusion was ordered twice a day. The first dose was ineffectual; it was repeated after two hours with no better result. After two more repetitions of the medicine, the discharge was still flowing with unabated profusion, and it

became manifest that the symptom was not amenable to the remedy. This was the first and only exceptional case. On making a vaginal examination, an irregular fungoid tumour was found attached to the anterior lip of the os uteri. This was extremely vascular, and its surface was the source of the discharge. The entire disease was external to the cavity of the uterus; the organ itself was healthy. If the medicine possessed a merely astringent action, its effect would be the same whether the cancer were within or without the cavity of the uterus.

Before concluding this portion of my subject, I may give the results of some experiments upon the recently gravid uterus, which, if not sufficient in themselves to establish the stimulant effect of digitalis upon the organ, are valuable as tending strongly to confirm the conclusions previously formed.

E. Waters, being, as she supposed, very near the termination of her fifth pregnancy, had an attack of vague abdominal pains, which presently passed off. On the following day no pain whatever was experienced, and there was no sign of impending labour; whereupon an experimental dose of 20 drops of tincture of digitalis was administered. In fifteen minutes slight uterine pain was experienced; and in fifteen minutes more the patient was unmistakably in labour. Within nine hours the child was born. Ten minutes after that event 50 drops more of the tincture were given. This was followed, in ten minutes, by a most marked increase in the uterine pains; and for the next twelve hours the after-pains were excessively frequent and severe, more so than she had ever before known them.

Mary Macaulay was delivered of her first child on the 1st of August. After-pains were complained of on the following day. On the 3rd and 4th they were completely absent. On the evening of the 4th, half an ounce of the infusion of digitalis was given, and severe pains were felt through the night. The medicine was continued three times a day until the 7th. During the whole of that period the pains were

frequent, and latterly they were very acute. When the medicine was discontinued they at once subsided.

Harriet Killick was delivered of her sixth child on the 18th of August. In previous labours she never had after-pains severely, and they never lasted beyond the second day. An hour after the completion of the labour, the patient being free from pain at the time, one ounce of the infusion was administered, and ordered to be repeated twice a day. A quarter of an hour after the first dose acute labour-pains came on, as severe as while the child was in the uterus. These subsided after two hours, and the patient was free from pain until the second dose was taken. This was followed by another attack of pain, which, however, was not so severe as the previous. When the medicine was taken for the third time, the patient had been for hours without uneasiness of any sort. In a quarter of an hour she was seized with after-pains of the same character as previously, but of much greater severity. 'She thought she should have been torn to pieces.' The patient now declined to take any more of the medicine, and had no further attacks of pain. It was observed in this case that there was almost a total absence of sanguineous discharge, although in all previous labours it had been profuse.

In four other cases the pain of uterine contraction was observed to depend on the administration of digitalis. The particulars are not given, as the cases very closely resemble those already narrated.

In these experiments the fact sought to be established is simply the connection between the pain of uterine contraction and the absorption of digitalis into the system. It is not expedient to multiply cases, as the experiment seems not devoid of cruelty, and, if it were recklessly enforced, might well be productive of danger. These cases seem quite sufficient to prove that the drug has the action upon the uterus which has been previously attributed to it. It stimulates the nervous system of the organ and excites the muscle to contract. It stimulates the ganglia in which the

motor power of the uterus resides. This fact is not without significance. It may help us eventually to a more general and philosophical view of the action of digitalis than we have yet attained. It will be seen that this effect upon the uterus is precisely analogous to the influence exerted by the same medicine upon the heart. In a lecture given by Dr. Bence Jones before the College of Physicians, and printed in the 'Medical Times,' the action of digitalis upon that organ is very fully explained. It is therefore not necessary, in this place, to do more than repeat the conclusions there arrived at. It is there shown that the medicine acts as a stimulant upon the musculo-motor system of the heart, although that effect is at first masked by an action of the same kind upon the pneumogastric nerves. 'It may safely be assumed that in man digitalis acts on the nerves that regulate the heart's action, first as a stimulant, and in large doses as a sedative.'

The operation of digitalis upon the other involuntary muscular organs yet requires to be correctly observed. It is by no means impossible that its effects upon the heart and uterus may turn out to be merely portions of an action which occupies a wider field than has hitherto been supposed.

In concluding, I wish to express my regret that I am unable to give any comparative statement of the manner in which the uterus is affected by the various preparations of digitalis; but I cannot now supply deficiencies, as my opportunities for observation have drawn to an end.

I cannot sufficiently express my obligation to Dr. Lee, for allowing me to make free use of the cases in his ward. It is to his kindness that this paper owes its existence. I am, moreover, indebted to Dr. Barclay for valuable suggestions, and to Dr. Bence Jones for invaluable counsel and encouragement.

## *THREE CASES OF ANGINA PECTORIS, DEPENDING UPON OCCLUSION OF THE MOUTHS OF THE CORONARY ARTERIES.*[1]

Case I.—The first case was that of a gentleman, 45 years of age, a patient of Dr. Dudfield of Kensington. Dr. Dickinson was asked to assist at his *post-mortem* examination. The patient, a tall muscular man, had had symptoms of disease of the heart for eight years, for the last three of which he had had well-marked fits of angina. In an attack within a day or two of his death, Dr. Dudfield described him as presenting a ghastly appearance, with a pale face, glazed eyes, and upraised arms. He complained of a horrible sense of constriction in the chest, with pain, numbness and coldness in the left arm. He died at last rather suddenly. At the examination of the body, the stomach was found to be dilated, but there was nothing else worth mentioning, excepting the state of the heart. This organ was increased in size and weight. The ventricles were both contracted. The muscular substance was generally slightly fatty, but was not more altered in this respect than most hearts are found to be. The root of the aorta and the aortic valves were much altered by atheromatous deposit. This was soft, and lay under the lining membrane of the vessel, especially about the origin of the coronary arteries. By the encroachment of this material the left coronary artery had become completely closed, so that its position could only be found by tracing up the vessel from the outside. The right artery was much narrowed, but still remained pervious. The coats of these vessels were free from atheroma. The left artery had become much shrunk, while the right retained its full proportions.

Case II.—A spare, muscular man, 42 years of age, by occupation a gamekeeper, became a patient at St. George's Hospital, under Dr. Fuller, October 4, 1865. He had for more than a year had pain after food, about the epigastrium. Latterly he had had attacks, described as angina, during which he stood still, with the elbows raised from the sides and the face expressive of anxiety. These attacks were, like the epigastric pain, prone to attack him after food. While in the hospital he did not keep his bed, the general health being pretty good.

[1] 1865. *Pathological Transactions*, vol. xvii.

Treatment was chiefly directed to the dyspepsia. On the afternoon of the fifth day of his stay in the hospital, while in conversation with his physician, he had a sudden attack of the sort described, with epigastric pain. In a few seconds his face became blue, and he had a short but distinct epileptiform convulsion. After this, though galvanism and other means were promptly employed, he gave no further signs of life.

At the *post-mortem* examination, a few points of extravasated blood were found upon the surface of the heart, possibly the result of the artificial respiration which had been employed. The left ventricle was quite uncontracted, and contained fluid blood. The valves were natural. There was a good deal of cushion-like soft atheroma on the aorta, under the lining membrane. Some of this material had encroached upon the mouths of the coronary arteries, and narrowed them so that neither could admit the head of a common probe, without some pressure. The heart was rather increased in size—thirteen ounces and a half. The muscular substance was of natural colour and texture. Under the microscope a trifling amount of diffuse fatty dotting was seen, but not such as to indicate any morbid change. The brain was natural; the lining membrane of the stomach was congested.

Case III.—The third case is that of a soldier, 35 years of age, who had been discharged from the army in consequence of some cardiac affection. He was brought into St. George's Hospital dead, in the evening of the 28th of April, 1864. An agitated young woman came with the body, and from her account it appeared that the two had been passing some time together in the park, after having had supper. While in her company, as she said sitting down, he fell dead. From the fact that the glans penis was found to be covered with spermatozoa, it was presumed that either at the time of death, or very shortly before, he had been indulging in sexual intercourse. Possibly he may have died in the act, the excitement having produced a paroxysm of the disease, from which he had before suffered.

*Post-mortem examination.*—The heart, as in the preceding cases, was slightly enlarged—fourteen ounces. The left ventricle was quite uncontracted; the right, partly so. Both contained fluid blood. The arch of the aorta was covered with soft atheroma, which was under the lining membrane. By the encroachment of this deposit, the opening of the right coronary artery was completely closed, so that not a trace of it remained.

Its position could not be found, except by following up the artery from the outside to its origin. The lining membrane of the aorta appeared to be continued over the mouth of the vessel. The opening of the left coronary artery was narrowed so as to be nearly gauged by the head of a probe. The coronary arteries in their course were natural. The muscular fibres of the heart were slightly dotted with oil. The valves were all effective; the aortic were thickened to a very trifling extent.

The stomach was distended with gas, and with undigested, almost unmasticated, food, comprising a considerable quantity of potatoes and pickles. All the organs were examined, but nothing further could be found, excepting that the lungs were slightly emphysematous, and that there was a little muco-purulent secretion in the bronchial tubes.

Ever since Heberden drew attention to a 'disorder of the breast,' in the year 1772, morbid anatomists have been busy in endeavouring to connect the symptoms with some constant alteration of structure. It does not appear that much progress has been made in this direction. Reference to any modern treatise on diseases of the heart will show that there are few, if any, morbid conditions of the heart which have not been assigned as causes of angina. Atheroma of the aorta and of the coronary arteries has been frequently observed. In the first *post-mortem* examination recorded, of a victim to this disease —it was made by John Hunter, and is recorded in Vol. III. of the *Transactions of the College of Physicians*—there is a careful description of an extreme state of atheroma of the beginning of the aorta, and of the semilunar valves. Indeed, of all the changes which have been associated with the symptoms, atheroma of the aorta appears to have been that most often observed. Atheroma, or 'ossification,' as it used to be called, of the coronary arteries, has been regarded as a cause of the complaint, but Laennec showed that this state is of less frequent occurrence in connection with the symptoms than is atheroma of the aorta. Often as this change of the arch has been noticed, it does not appear that the state of the coronary openings has attracted observation; an omission which seems strange, consider-

ing that the coronary arteries, in their course, were frequently made the subject of dissection.

The closure of the mouths of these vessels by soft atheroma, spreading under the lining membrane of the aorta may, as these cases show, be a not infrequent cause of the disease, and a cause which is exceedingly liable to be overlooked. One of the coronary openings may appear to be wanting; the lining membrane of the aorta looks as if smoothly continued over it; and if the mouth of the vessel is not especially looked for, its absence is not detected. The atheroma is soft and cushion-like, and is less conspicuous than the rough bony variety.[1]

In the Museum of St. George's Hospital is a drawing by Mr. Perry, which was made many years ago. It represents the aorta of a patient of Dr. Seymour's, who had a paroxysm of acute pain in the chest, with orthopnœa every evening, for some time preceding his death. The aorta is covered with the smooth atheroma, described. The orifices of all the vessels leading from it are narrowed: those of the coronary arteries are invisible, and appear to have been occluded. No notice appeared to have been taken of this peculiarity. The accuracy of Mr. Perry, as an artist, is sufficient warrant for the truth of the representation.

Whether considered as spasm of the heart—a view inconsistent with the frequent absence of contraction in the ventricles after death—or as a neuralgic affection—there can be no doubt that the nervous structures of the organ are radically concerned in the production of the symptoms. In connection with this we cannot but observe the relation of the coronary arteries to the nerves which cover the ventricles. The vessels lie just beneath the

[1] Dr. Arnold, of Rugby, who, as is well known, died of angina pectoris, probably furnished an instance of the pathological change which has been described. It is stated, as the result of *post-mortem* examination, that '*there was only one coronary artery*' and that this was of small size, with difficulty admitting a small director. We may presume that the only part of the missing vessel which was looked for was its aortic orifice, and that this was closed and smoothed over and obliterated in the manner described. Had the outside of the aorta been examined as well as the inside, the trunk of the lost artery would have come into view.

pericardium, and are closely entwined by the nerves, and, judging by their obvious arrangement and situation, would seem to have more to do with the nerves than with the muscular fibre. They are, of course, too large to be needed by the nerves alone; but it may be presumed that they supply the nerves, and the more superficial part of the organ. In the cases recorded, though they were in great part closed, and from the chronic nature of the cases must have been so for some time, there was no atrophy of the muscular substance. Indeed, the closure of the coronary vessels does not prevent considerable hypertrophy of the heart, when there is any valvular disease to set it up. Probably the muscle of the heart is nourished in great part by imbibition from its cavities, in the same manner as are the walls of the great vessels.

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## *PERFORATION OF THE SIGMOID FLEXURE OF THE COLON, PROBABLY DUE TO THE IRRITATION OF FÆCES.*[1]

Case I.—A woman, named M. H., aged 42, died in St. George's Hospital, under the care of Dr. Barclay, August 28th, 1863. She had for some months before admission suffered from constipation, and she described herself as of a "bilious habit." She was attacked about a month before her death with pain and tenderness in the left iliac region, and she eventually sank with symptoms of circumscribed peritonitis.

At the post-mortem examination a partially empty cavity, as large as a cocoa-nut, was found occupying the left iliac region of the peritoneal cavity. The front of this cavity was formed by the abdominal wall, the back by the bowels, which were matted together by firm lymph. The cavity contained pus and fæcal matter. It communicated with the sigmoid flexure of the colon by means of some ulcerated perforations.

On laying open the intestine about half-a-dozen ulcers became visible. These were oval, like button-holes, and of much the same size. They were all near together, parallel to each other, and transverse to the axis of the bowel. They were without

[1] 1867. *Pathological Transactions*, vol. xviii.

thickening, looking as if a piece of the bowel had been cleanly cut out. The mucous coat was more widely destroyed than the serous. There was no trace of tubercular disease in their neighbourhood. The only signs of morbid action in their vicinity were some transverse brown lines, chiefly occupying the folds of the bowel, and looking as if they had been produced by contact of the fæces. These, however, were small, did not correspond accurately with the ulcers, and from this specimen it was doubtful how far the ulcers and the brown stains were connected.

The right kidney was atrophied and contained tuberculous matter. There was no tubercle elsewhere.

Case II.—C. R., a tailor, aged 42, died in St. George's Hospital, February 10th, 1865. He had for some years occasional pain in the left hypochondrium, latterly severe. This apparently was associated with constipation, for it was generally relieved by purging. He became œdematous; the urine was found to be albuminous, and finally he had diarrhœa, which proved incontrollable and appeared to be the chief cause of death.

At the post-mortem examination a large circumscribed cavity, full of blackish foul matter, was found in the left side of the belly, bounded by the diaphragm, spleen, and descending colon. In the upper part of the descending colon were two small narrow ulcers, which opened into the cavity. There was no thickening of the coats of the bowel, nor was there any evidence of morbid action in this situation, excepting the ulcers themselves. These were like button-holes, the two close together, parallel, and with their long diameters across the axis of the bowel.

The kidneys were infiltrated with waxy deposit. The small bowel was similarly affected. There was no tubercle in the body, nor any other morbid condition, excepting that the cystic duct was blocked up by a calculus.

Case III.—A. B., aged 21, died in St. George's Hospital, October 10th, 1865. She had suffered habitually from constipation. Of late she had had much pain in the belly, with vomiting, tympanitic distension, diarrhœa, and extreme prostration. After these symptoms had lasted with more or less variation for two months, she sank. It appeared that a year previously she had had violent abdominal pain, which was attributed to gall-stones.

At the post-mortem examination, a circumscribed fæcal abscess was found in the neighbourhood of the left psoas muscle, lying

in the peritoneal cavity circumscribed by adhesions. This was in connection with three small ulcers which had perforated the descending colon. These openings were about the size of a pea, the edges were sharp, and the adjoining bowel was perfectly natural.

The peritoneum had many minute tubercular deposits upon its free surface, particularly where it covered the abdominal wall. In some cases the tubercle had perforated the membrane, so as to be in contact with the tissues beneath. A single cheesy tubercle was found in the right lung.

The femoral veins each contained old, soft, buff-coloured coagulum.

The perforation of the colon in these cases appears to be of a kind which, though perhaps not very uncommon, has not obtained especial notice. No cases clearly of the same sort are reported in the *Transactions* of the Society.

The ulcers are quite peculiar in appearance. They are not dependent upon typhoid fever, or upon malignant or tubercular deposit. They are formed upon the prominent transverse folds of the bowel, by reason of which they are parallel to each other, and across the axis of the intestine. They begin from within, affecting the mucous membrane more widely than the serous. They are frequently associated with peculiar brown stripes which lie upon the prominences of mucous membrane in such situations as the ulcers occupy. These marks look like fæcal stains. They are accompanied by change in the mucous membrane, as appears from their being sometimes spotted with minute extravasations of blood. That these stripes are apt to terminate in ulceration is clearly seen in early cases where a minute shallow elongated ulceration may be seen to occupy the centre of each brown stripe and the summit of the intestinal fold. In cases of longer standing the connection between the brown mark, and the ulcer which arose in it, of course becomes less evident.[1]

These appearances cannot fail to suggest that ulcers of the kind described have arisen from the irritation of the

[1] These statements are based upon cases which were not represented by specimens, and therefore are not related in detail. I think there is little doubt that the ulceration in all was of the same nature.

fæces. That hardened excrement is capable of causing ulceration by its contact is shown by the frequent perforation of the vermiform appendix by fæcal concretions. In all the cases recorded constipation had apparently preceded the ulceration. Whether any unnatural condition of the fæces has rendered them more than naturally irritating is a question which cannot as yet be answered.

It will be observed that there is a great tendency to circumscription of the peritonitis resulting from the affection described. The fixed and protected condition of the sigmoid flexure gives facilities for the processes of adhesion and limitation which do not pertain to the more movable parts of the bowel.

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## *ON THE CHANGES IN THE NERVOUS SYSTEM WHICH FOLLOW THE AMPUTATION OF LIMBS.*[1]

Some years ago, in the hope of throwing light upon a conclusion which I had formed to the effect that the cerebellum[2] directed its influence in an especial manner to the lower limbs, I sought opportunities of examining the brain after amputation of the extremities in the vain expectation of finding that the portion of the encephalon which regulated the movements of each limb would be declared by a localized atrophy consequent upon its removal.

After weighing and examining with the microscope the various parts of the brain, in several cases in which one or more limbs have long been absent, I was driven to the conclusion that loss of the extremities was not followed by loss of weight, or by appreciable change of structure, either in the cerebellum or in the hemispheres or great ganglia of the cerebrum.[3]

[1] *Journal of Anatomy and Physiology*, November 1868. In the original paper several illustrations may be seen which are not here reproduced.

[2] See paper on the Functions of the Cerebellum, *British and Foreign Medico-Chirurgical Review*, October 1865.

[3] This statement rests upon the examination of four cases of amputation of one leg, and one of amputation of both. In the four cases, one leg had been removed at periods varying from two years to fifty-three years before death. In each instance I divided the cerebrum and cerebellum as carefully

Failing to find atrophy within the cranium as the result of these mutilations, I next sought to trace evidences of change by commencing at the stump and working upwards, hoping by this means to secure evidence of a more positive kind.

I will give a brief statement of the facts I have obtained. The subjects of the following observations were seamen who died in Greenwich Hospital:—

Case I.—A seaman who was fighting on board the *Dictator* Frigate, in an action with the Danes, on the 6th of July, 1812, had his left leg carried away by a cannon shot. Amputation was performed at the junction of the upper and middle thirds of the thigh, and a wooden leg subsequently provided. He died in the year 1865 at the age of 74, having survived the loss of his limb for 53 years. Through the kindness of Dr. Maclaurin I was enabled to attend the post-mortem examination.

The stump was conical and much wasted, the muscles being very soft and fatty. At the end of the sciatic nerve was a large globular neuroma. The nerve was dissected out up to the pelvis, as also was the corresponding portion of the sciatic nerve on the right side. The two nerves laid side by side looked almost exactly alike, no difference being observed in bulk, texture, or colour. They were hardened in chromic acid, and transverse sections were examined with the following results.

as possible along the median plane and then weighed the separated halves of each structure. The inequality of weight was very small, such only as must necessarily have resulted from accidental unfairness of section, and the loss was sometimes on the side of the amputation, sometimes on the opposite side. With regard to the cerebellum in particular I found that the lobe on the side of the operation appeared in three of the cases to have lost weight to the amount of about 30 grains, while in the fourth case that lobe was heavier than its fellow by 8 grains. I judged, therefore, that neither cerebrum nor cerebellum acquired any lateral inequality of bulk as the result of the removal of one leg.

In the case in which both legs had been removed, the operation had been performed on both limbs just below the knee, twenty-five years before death. As there was no reason to expect any difference between the two sides, the halves of the brain were not divided. The cerebrum weighed 46¼ ounces; the cerebellum 4 ounces 300 grains; the cerebellum having a proportion to the encephalon of 1 to 10·2, a proportion which could not be looked upon as abnormal. I may add that the microscope showed nothing unnatural in the structure of any part of the encephalon, and that in the four cases previously mentioned, the same instrument gave similar negative results.

The bundles of nerve-fibres were not perceptibly altered in size or arrangement, and to the naked eye no difference could be discerned save that in the left sciatic nerve the contents of the fasciculi became more deeply coloured with carmine than those on the right side. Under the microscope, however, a most striking difference was found.

In the nerve of the right, or complete limb, the nerve-bundles each surrounded by the natural thin envelope of fibrous tissue, consisted of a close regular arrangement of nerve-tubes, each of which showed distinctly the outer white matter and the central, grey cylinder. The nerve-tubes were comparatively uniform in size, and were in contact with each other, excepting where they were separated by minute blood-vessels or by the fine fibrous septa by which those structures are naturally supported.

In the nerve of the stump the characteristic structure had undergone so complete a disorganisation that the nervous structure could scarcely be recognised. While the nerve-bundles retained their natural bulk and external conformation, the uniform tubular arrangement proper to their interior was replaced by a material which, under a low power of the microscope, presented merely a coarse granulation which absorbed carmine more freely than the natural nerve structure. A higher power (300 diameters) showed that the coarse granules were for the most part minute circles, which differed from the sections of nerve-tubes not only in their exceeding minuteness, but in the fact that they presented no distinction between white and grey matter. These dwindled representatives of nerve-fibres were not, like the healthy tubes, in contact with each other, but were separated by, and imbedded in, a structureless material, which from its power of imbibing carmine was the means of imparting the peculiar pink tint to the section which has been referred to.

Some of the bundles composing the nerve consisted wholly of the disorganised structure described; in others a few normal nerve-tubes could be detected. All were surrounded by an envelope of connective tissue somewhat thicker than existed in the normal nerve.[1]

The perfect preservation of the outer aspect and larger

[1] I may here state by way of parenthesis that in the case of another Greenwich Pensioner who had suffered amputation of the thigh many years before death, precisely the same destruction of the fibres of the sciatic nerve was found, with the same retention of external appearance. I have not given this case in detail, as I had no opportunity of examining the cord.

anatomy of the nerve is interesting, and would, had not the microscope been used, have led to the belief that the essential structure was natural.

The spinal cord was examined in section after hardening in chromic acid, and the changes traced from below upwards.

In the portion between the lower extremity and the lumbar enlargement, the grey matter was somewhat smaller on the left side than the right. The difference was very slight and might easily have escaped observation. No difference was observed in the shape of the grey crescent, or in its intimate structure. The nerve-cells were apparently the same on both sides. In this situation the white matter was symmetrical.

In the lumbar enlargement there was a decided loss of thickness in the left posterior column, which was sufficient to cause distortion of the cord, giving in particular a slanting direction to the transverse commissure, in the neighbourhood of which the wasting of the posterior column was greater than nearer the circumference of the cord. The wasted column showed a relative increase in connective tissue, as if this material had fallen together on the removal of nerve-tubes. The nerve-tubes which remained exactly resembled those of the opposite side in the corresponding situation. The atrophy of the left posterior column was traced up through the dorsal region. Here as in the lumbar enlargement the grey matter was symmetrical and apparently unaltered. No change was found in the white matter excepting what has been described as affecting the left posterior column. It was roughly estimated that this portion of the cord had lost, in the lumbar region rather more, in the dorsal region rather less, than a quarter of its bulk. (Fig. 1, L.)

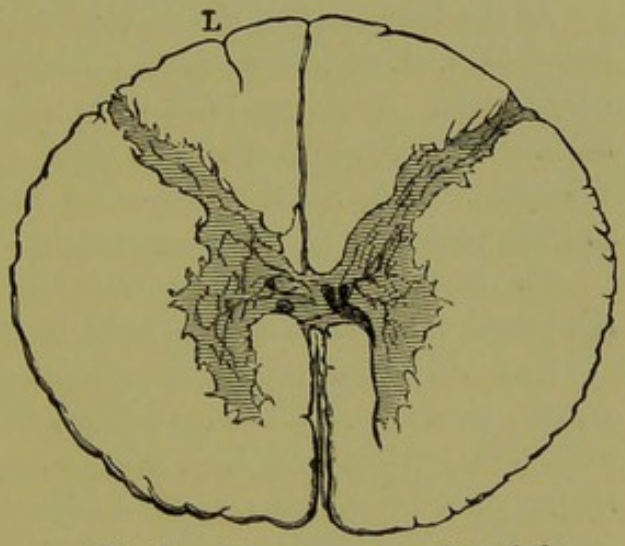


Fig. 1.—A transverse section of the dorsal part of spinal cord. L, the left posterior column, is distinctly smaller than the right.

The cervical part of the cord was not obtained for examina-

tion. Various sections, however, were made through the medulla oblongata, the two sides of which proved to be perfectly symmetrical and natural in all respects. The brain was examined with the negative result which has been already stated.

Case II.—In the year 1865, I examined the body of a Greenwich Pensioner who had lost the left arm 23 years previously. In the year 1842 he had fallen from the rigging of his ship, and so injured the limb that amputation had to be performed two inches from the shoulder-joint.

It is only necessary to describe the changes in the nervous system.

At the lower end of the cervical region, for the space of about two inches, there was a striking diminution of the left posterior nerve-roots. The strands of nerve-fibres were less than half the thickness of those on the right side; and were also considerably thinner than the anterior roots on the same side. No change in their colour or texture was evident to the naked eye.

On making transparent sections of the roots in different situations the following facts were apparent.

The dwindled posterior roots were materially altered in minute structure. The nerve-tubes instead of being uniformly packed in contact with each other were here and there separated by irregular masses of a carmine-tinted material in which a high magnifying power showed the shrivelled remains of nerve-tubes—minute circles with a central speck. The tubes which retained their bulk were not unnatural.

The left anterior roots corresponding to the atrophied posterior roots, though they were not appreciably altered in thickness, showed under the microscope traces of the same partial destruction of nerve-fibres. In the anterior roots, however, the amount of the change was much smaller than in the posterior.

The spinal cord, examined after hardening in chromic acid, proved to be natural excepting in the cervical region.

In the cervical enlargement the left posterior column (Fig. 2, L) was reduced in thickness, presenting in section about two-thirds the surface shown by its fellow. The loss of bulk chiefly affected the anterior part of the column, the part, that is, towards the commissure, where it had a very pointed outline compared with the column on the opposite side.

Beside the mere loss of bulk the wasted column was tra-

versed by a line of altered structure, which passed from the circumference towards the centre in a radiating direction so

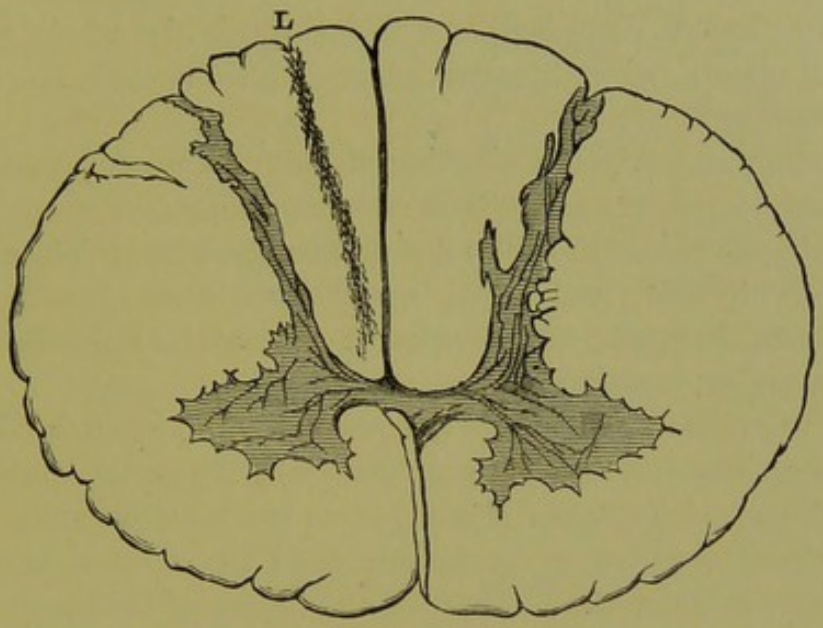


Fig. 2.—A transverse section of the cervical part of the spinal cord from a man whose left arm had been amputated twenty-three years before death. The left posterior column (L) is smaller than the right.

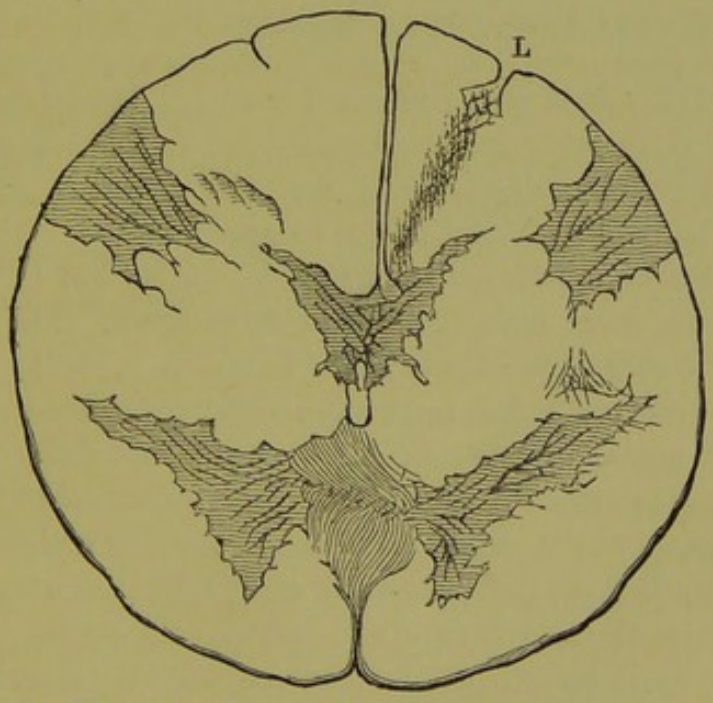


Fig. 3.—Transverse section of the medulla oblongata from the same patient, showing the left posterior column (L) smaller than the right.

as to divide the column into two nearly equal parts. Nerve-tubes were nowhere unnatural or totally absent, but the line

of change was indicated by a condensation of areolar tissue, as if the nerve-tubes had, at some period, been withdrawn from the structure.

The change described extended through the whole of the cervical region, being, however, more declared in the enlargement than elsewhere.

There was a slight diminution of bulk in the grey matter of the cervical enlargement. The crescent retained its shape, every part being apparently slightly and uniformly shrunk. The change was very trifling, and might easily have escaped notice. It showed no change in its microscopic characters, the cells being unchanged in appearance.

In the medulla oblongata it was found that on a level with the decussation of the pyramids there was the same wasting of the left posterior column (Fig. 3, L) as has been described in the cord, the change being continuous from the cervical region to the upper limit of the decussation. The loss of bulk and alteration of structure which have been descried in the left posterior column of the cord were equally evident in the corresponding portion of the medulla in the position stated. Above the decussation all traces of the atrophy were lost, the medulla being symmetrical and natural in all respects.

Case III.—Last year I received from Dr. Middleton the cord of an old Greenwich Pensioner who had in consequence of an accident suffered amputation of the right forearm, in the year 1845. The cord had been placed in spirit before I obtained it, and did not yield such satisfactory sections as did the preparations which have been heretofore dwelt upon. The following points, however, were clearly made out.

In the cervical enlargement the right posterior column was narrowed, especially towards the commissure. This column at the point of greatest loss presented about two-thirds of the bulk of the column of the left side. The grey matter had also suffered a slight diminution in bulk on the same side, the anterior horn being decidedly narrowed from before backwards. As in the preceding cases no change of structure could be detected in the grey matter.

The medulla was not obtained for examination.

Placing together the several observations, it appears that when a limb has been absent, as the result of opera-

tion, for twenty or more years, the following changes have been found in the nervous system.

First, atrophy of the nerves of the stump, of which a large proportion of the fibres have perished, notwithstanding that, supported by the fibrous tissue which enters into their structure, they retain their bulk and external appearance almost without alteration.

Secondly, wasting of the nerve-roots, especially the posterior. The wasting of the tubes, in the absence of such fibrous investure as belongs to the mixed nerves, produces an attenuation, which in the case of the posterior root is very conspicuous.

Thirdly, a slight loss of bulk in the grey matter of the cord, on the side of the lost member, near the origin of its nerves, without any intimate change discernible by the microscope.

Lastly, a remarkable shrinking of the posterior column of the cord on the side of the mutilation, attended by a condensation of areolar tissue. The atrophy extended upwards, and in the case of the loss of an arm can be traced into the medulla oblongata as far as the upper limit of the decussation of the pyramids.

The cerebrum and cerebellum remain unchanged.

I am aware that many details relating to this subject remain to be worked out, and I should have waited for further opportunities had not my purpose been forestalled by M. Vulpian, who has, since these observations were made, published two similar cases.

His results differ very materially from mine. Both the cases he reports were of amputation of the leg, a little distance above the ankle. In one case the leg had been removed for 47 years, in the other for 20 years. In both cases M. Vulpian describes the spinal cord as slightly lessened in bulk on the side of the amputation. This diminution affected the grey matter generally, the white matter *with the exception of the posterior column.*

The cells of grey matter were not altered in character, or appreciably in number. In one of the cases some spots of

disintegration were supposed to exist in the grey horn. No changes were detected in the nerves or nerve-roots.

My results differ from those of M. Vulpian in the atrophy of the nerves, posterior nerve-roots, and posterior columns of the cord, which were found in my cases but not in his. His cases and mine coincide in attributing a slight loss of bulk to the grey matter on the side of the mutilation.

Dr. Waller long ago pointed out that nerves separated from their centres rapidly became atrophied. With regard to the spinal roots in particular he found that when an anterior root was cut the part retaining its connection with the cord remained unaltered, while the outer extremity wasted. After dividing a posterior root the reverse took place ; the central end wasted, the peripheral end retained its structure. From these and other experiments, Dr. Waller was led to conclude that the outer portion of the severed posterior root owed its retention of structure to the ganglion to which it was attached, while the nutrition of the motor root depended on the cord.

The facts brought forward in this paper appear to show that these conclusions need modification. It would seem that the posterior root may waste though still in connection with the ganglion, the anterior though still in connection with the cord. The ganglion, therefore, is not the sole controller of the nutrition of one root, or the cord of the other. It appears that long disuse of a nerve is sufficient to lead to its atrophy, notwithstanding that those nervous structures which more immediately regulate its nutrition are complete.

There are some points which as yet must be left without explanation, namely, the greater atrophy of the sensory than of the motor roots, and the peculiar wasting of the posterior columns, passing vertically up the cord, and in the case of loss of the arm affecting the medulla, a course not corresponding with that of the sensory fibres, which soon lose themselves in the grey matter.

I must in conclusion convey my thanks to the gentlemen who have assisted me on the present occasion, more

especially to Sir Alexander Bryson, the Director-General of the Navy Medical Department, and to Sir Edward Hilditch, Dr. Maclaurin and Dr. Middleton, formerly Officers at Greenwich Hospital.

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## *ON COUNTER-IRRITATION*,[1]

### CONSIDERED IN REFERENCE TO THE REMOTE AND INDIRECT EFFECTS OF LOCAL MORBID CHANGES.

The rapid changes which are taking place in our knowledge of function and of disease make it necessary for us to review from time to time the rules which direct the application of remedies. The remedial branch of medicine, all-important though it be, can hardly yet be said to exist as a science. We hold to measures of treatment which our forefathers introduced, notwithstanding that our forefathers were led to them by suppositions now known to be erroneous. We allow the therapeutics of bygone generations to hold its place beside the pathology of to-day, and place in ill-matched apposition the art of one century and the science of another. Therapeutical traditions long survive the theories which gave them birth. In our use of drugs we are often unwittingly guided by considerations as mythical as the cabalistic reference to Jupiter with which we never fail to commence our prescriptions. The ancient doctrine of signatures taught that rose-leaves would stop bleeding because they were of the colour of blood; and to the present day infusion of the red rose commonly forms the basis of a styptic draught. The use of external applications is often based upon the effete extravagances of humoral pathology, and guided by doctrines as mysterious and as fanciful as those which were put forth by Sir Kenelm Digby, who professed to cure wounds by his 'powder of sympathy,' the efficacy of which was undiminished by distance, and which could be applied in one country for the advantage of patients living in another.

Rules of treatment once authoritatively placed in the

[1] 1868. *St. George's Hospital Reports*, vol. iii.

code of medical practice are exceedingly difficult to displace. They become the property of 'practical' men, who are content to resort to the usage of their day without inquiring for the evidence on which it rests, and who hold to therapeutical traditions with a steadfast faith worthy of a more rational creed. The practice of such men is necessarily confirmed by their experience, since, while they are apt to take to themselves the credit of every improvement, they attribute all changes for the worse to the inevitable progress of the complaint. The murderous extravagances in the use of blood-letting and mercury which characterised the earlier part of this century could never have held their ground, had not the results of treatment been assigned to disease; and there probably remain many expedients in common use which would long since have been forgotten, were they not frequently credited with favourable events they have had no share in producing.

The *post hoc, propter hoc* argument, never more fallacious than in therapeutical matters, often connects together as cause and effect circumstances which have no more to do with each other than, to use a proverbial illustration, Tenterden steeple and Goodwin sands.[1] To suppose that excoriations of the surface of the body, in themselves

[1] The story which associates Tenterden steeple and Goodwin sands as cause and effect has antecedents of such an eminently respectable character, that I may be excused for repeating it. It is told by Bishop Latimer of Sir Thomas More. It occurs in one of Latimer's sermons, whence I have copied it with slight abridgment:

'Master More was once sent in commission into Kent to help to try out, if it might be, what was the cause of Goodwin sands, and the shelf that stopped up Sandwich haven. Thither cometh Master More, and calleth the country afore him, such as were thought to be men of experience, and men that could of likelihood best certify him of that matter. Among others came in before him an old man with a white head, and one that was thought to be little less than an hundred years old. Quoth Master More: "How say you in this matter? What think ye to be the cause of these shelves and flats that stop up Sandwich haven?" "Forsooth, sir," quoth he, "I am an old man, and I remember the building of Tenterton steeple, and I remember when there was no steeple at all there. And before that Tenterton steeple was in building, there was no manner of speaking of any flats or sands that stopped up the haven; and therefore I think that Tenterton steeple is the cause of Goodwin sands."'

trifling, can produce deep-seated alterations in unconnected though neighbouring organs; to imagine that bedaubing the chest with tincture of iodine can modify the course of tubercular disease in the apex beneath; or that a superficial vesication can promote the restoration of a hepatised lung—are views founded probably on no better reasoning than that which ascribed a formation the result of a profound geological or oceanic change to an artificial superaddition to the surface of the neighbouring soil.

My object at present is to consider the custom which prevails of attacking the diseases of internal organs through the few square inches of skin which have the misfortune to lie within the shortest distance, 'as the crow flies,' of the seat of the morbid change, notwithstanding that no continuity of structure exist, and that there be neither vessels nor nerves which maintain a direct communication between the peccant organ and the suffering cuticle. The custom may be likened to a practice which once prevailed at the Scottish Court, where the children of the royal household were not punished in their own persons for any faults they might have committed, but the stripes were inflicted vicariously upon a boy unconnected with the royal lineage, who was kept for such uses.

John Hunter insisted upon the existence of what he termed 'contiguous sympathy' between parts which lay in contact with each other though not continuous in structure, as between the bowels and the integuments of the abdomen, the lungs and the chest, the brain and the scalp, the testicles and the scrotum; but he explains the sympathy so defined as depending on no other connection than that which arises from the contact of separate parts, and which therefore amounts to much the same thing as his 'continuous sympathy,' which was the term by which he expressed the tendency which morbid processes, particularly of an inflammatory kind, have to spread to structures which are in continuity with that first attacked.[1]

Other writers, however, have not been content with the

[1] Hunter's *Works*, Palmer's edition, vol. iii. p. 6.

simple doctrine, that internal organs may become involved in morbid changes which commenced in the walls of the cavities in which they are held, but have superadded an apocryphal creed, in which antipathy has taken the place of sympathy, which asserts that a superficial inflammation tends to counteract a change of the same nature in deeper structures.

Dr. Paris,[1] a philosopher as well as a physician, states that 'in all inflammatory affections of the internal organs, a blister placed on the contiguous surface affords great relief, not only by the discharge it occasions, but by *a transference of the inflammatory action to the surface*.'

Dr. Headland, in his recent work on the *Action of Medicines*, explains counter-irritation by the statement that 'a powerful impression on any surface of the body seems to be capable of arresting and diverting, as it were, the attention of the system, and thus, for a time, of checking a morbid process.'[2]

And looking at the details of practice at the present day, or within a recent period, we find that counter-irritation is used in the treatment of disease on the principles put forth by these authors; the upshot of which is, that the surface of the body and the organs beneath have a relationship which causes them to alternate in disease, one being relieved when the other is attacked.

Affections of the brain, whether supposed to depend upon excessive vascular action, as inflammation or congestion, or upon the contrary condition of anæmia; whether dependent on changes originating in the brain itself, or upon disorders, like renal disease and fever, which arise elsewhere; whether associated with recognisable causes, or with disturbances which we have not learned to distinguish,—all alike expose the patient to the chance of having painful excoriations added to his other sufferings.[3]

[1] *Pharmacologia*, 1843, p. 243.

[2] *Action of Medicines*, 1867, p. 87.

[3] The popular and even the professional trust in counter-irritation finds expression in the address of Mrs. Gamp to the senile Chuffey: 'Spanish flies is the only thing to draw this nonsense out of you; and if anybody

If he be the victim of diabetes, his neck is liable to be blistered for no better reason than because that disorder is supposed to be sometimes connected with profound changes in the structure of the medulla oblongata.

Disorders of the spinal cord are sometimes treated by blisters and other irritants upon the skin which overlies the spinal column.

Affections of the eyes, particularly of an inflammatory sort, suggest to some practitioners the insertion of setons in the back of the neck.

Tubercular disease of the lung is held to warrant the application of iodine or blisters to the wall of the chest which overlies the part affected. Pneumonia and bronchitis are often met by similar applications.

Ulcer of the stomach brings the skin of the epigastrium within danger of the actual cautery, and obstinate vomiting upon whatever cause it may depend is regarded as a reason for placing blisters on the same part of the abdominal wall.

Cholera has been treated by a great variety of applications to the outside of the belly, among which may be mentioned irritating oils, nitric acid, boiling water, and heated irons.

It is not, however, necessary to multiply examples to show the general acceptance and application of the tradition of counter-irritation which insists that deep-seated disturbances of function or circulation are influenced advantageously by irritants addressed to the skin, providing only that they are applied within the shortest distance, in a straight line, of the organ which is the seat of the disorder. It does not appear to be necessary, according to this view, that there should be any direct connection between the deep organ and the overlying skin, or that the affection excited on the surface should be of the same kind as that of which the more important organ needs to be relieved.

wanted to do you a kindness, they'd clap a blister of 'em on your head, and put a mustard poultige on your back.'

It appears that this system has its foundation in the doctrines of the old humoral pathology, which, although containing a large admixture of truth, have been fruitful of mischievous practice. Each disease was supposed to exist as a separate essence, which was capable of movement from place to place. Our forefathers talked of 'a pleurisy' as of a distinct entity, which, though now affecting the chest, might under certain circumstances transfer itself to another part of the body. The idea of the translation of disease still holds its ground as regards the exanthemata, in which the appearance of the rash is often coincident with a relief of the constitutional symptoms; and it is probable that there are other disorders of which the local manifestations leave one place to reappear in another. Tuberculous disease of the lungs has often been thought to be relieved on the appearance of an anal fistula.

These instances, however, only show that a specific change in one place may alternate with the same specific change in another. A deposition of tubercle near the rectum may lessen the tendency to the deposition of tubercle in the lung. A scarlatinal eruption may relieve scarlatinal delirium, or a measly rash may relieve measly bronchitis. But the rule of translation is not applicable to any diseases excepting such as are caused by the presence, and relieved by the exit, of a morbid material; and the transference occurs in each disease as part of its natural course, and in a manner peculiar to itself. We have no power of producing the salutary movement, though we may sometimes hinder or prevent it.

In the majority of the diseases of internal organs which we are called upon to treat, we cannot expect any such transference of morbid action as occurs in the specific fevers. Inflammatory attacks usually depend upon some irritation or disturbance of circulation belonging especially to the organ *affected*, not upon any changes in the common fluids of the body. Meningitis is usually tubercular. Pneumonia is often associated with tubercle, or with valvular disease of the heart. Peritonitis generally depends

on changes of long standing in one or other of the organs covered by the peritoneum; and in further detail it would be easy to show that acute inflammation is continually the sequel of chronic disease. A local inflammatory attack, the result of local causes, cannot be supposed to have any such reciprocity with the skin as holds in the case of eruptive fevers between the several parts of the body which are its chosen points of attack.

We may now proceed to examine into the evidence on which this system of vicarious therapeutics rests. What association in their morbid processes is there between the skin and the organs which lie in the serous cavities underneath? Do curative influences traverse the body in straight lines, as if thrust at point of bayonet, regardless of the ramifications of nerve and vessel?

If, on the theory advanced by Dr. Paris, we seek to 'transfer the inflammatory action to the surface,' should we consider proximity rather than anatomical connection? Does the essential principle of the inflammation, which it is our hope thus to remove from one place to another, travel by channels other than those which convey the natural fluids of the body? Have we to deal with a ghostly essence which moves unhindered by corporeal bar?

If, with Dr. Headland, we endeavour by external measures to divert the attention of the system from a mischievous occupation; if we address measures to the less vital parts of the body of such an exasperating sort as shall induce the disease, like a ferocious animal, to forget its first object of attack and turn its spite upon the new assailant,—are we more likely to engage the attention of the enemy if we apply to its immediate neighbourhood, notwithstanding that it may have to come a long way round?

In order to deal with these and similar questions we must ascertain what is the extent of our knowledge upon the subject, and refuse to advance beyond its limits. We must act upon our information as far as it goes, and humbly confess our ignorance and our helplessness when we find

that we have neither demonstrable laws nor conclusive experience to direct our conduct. It is not easy, nor is it necessary, to disprove the fanciful theories which have at different times been allowed to guide medical practice. Therapeutical maxims, if not clearly warranted by experience, must be discarded without ceremony, however supported by usage or tradition.

With regard to the therapeutics of counter-irritation, we may take a circumscribed and superficial lesion, and ask what we know regarding the nature and direction of its effects. What influence will it exert, and where? Does it, according to the theory which guides the practice of the present day, influence in an especial manner parts which, though structurally unconnected, lie in its vicinity?

Supposing a sore or superficial locus of irritation to have been established by nature or art upon a given portion of the surface of the body, we must ascertain what influences issue therefrom, by what channels they travel, and to what ends they tend.

It will be convenient to consider first the lines of communication which exist in the body; to ascertain the paths by which a local affection can influence remote organs or the system at large.

The several parts of the body communicate with each other by means of

1. The blood-vessels.
2. The absorbents.
3. The nerves.
4. Continuity or apposition of structure.

A local change may affect other parts by either of these channels. It will be necessary to review each of them separately, with an especial regard to the conveyance of such influences as may be salutary or remedial. Having in this manner analysed the modes by which each part of the body is brought to bear upon the rest, we shall learn how far the practice of counter-irritation is consistent with our present knowledge.

1. *Influences which travel by the blood-vessels.*

The veins may convey the products of disease from their primary seat and carry them in the direction of the circulation, to become sources of mischief in distant parts of the body. Thus pyæmic abscesses, and more rarely malignant growths, are produced in situations which are regulated by the course of the blood-vessels. But it is not necessary to dwell upon the function of the veins in conveying morbid matter from one part to another, since we can imagine no circumstances in which such a process could be employed therapeutically.

If a local sore be attended with a discharge, instead of adding to the contents of the blood-vessels, it may be the means of subtracting from them. The greater part of every discharge is produced at the expense of the blood. The portion which consists of the débris of solid tissue is necessarily small; all the rest must be supplied by the circulating fluid. The blood, therefore, which leaves the source of a discharge differs from that which went to it by the material which has been thus extracted.

When the flux is purulent, if it be in large amount and long continued, such changes are produced by its withdrawal as to cause lardaceous disease all over the body. If the discharge be such as to remove a large quantity of serum or of aqueous fluid, we can trace the result in the general demand for fluid of the kind which has been removed. The blood has a power of self-rectification in virtue of which it attracts to itself materials in which it is deficient. If in a case of dropsy a large quantity of serum is poured out into a serous cavity as the result of pleurisy or peritonitis, we may often observe its simultaneous disappearance from the areolar tissue. Similarly, thirst follows excessive discharges of fluid from the bowels or kidneys.

When the discharge is small, the effect, though it may escape observation, must be equally real. The discharge is supplied by the capillaries of the affected part; in these, therefore, the deficiency in the blood is most marked. The

same want must be likewise evident in the veins into which those capillaries empty themselves; the peculiarity of the blood being diminished as it mixes with converging streams, until it is lost in the general circulation. It may happen that a discharge too small to influence perceptibly the general mass of blood may yet produce a decided effect upon the plexus of vessels which have suffered the drain. If the discharge be of serum, serum will be wanting in the vessels of the affected part—a deficiency which will be apt to be supplied at the expense of any collections of serum within the influence of those vessels. Thus a superficial vesication, although too trifling to act through the general circulation, may drain the fluid from a serous cavity which has a vascular association with the affected surface. A blister over a distended knee-joint will fill at the expense of that cavity; the skin and the synovial-membrane being supplied by the same, or intimately anastomosing vessels. The same rule applies to many other joints, and in a less degree to some of the larger serous cavities, the pericardium in particular. As illustrating the local action of a drain of serum from the skin, I may mention the following case:

A child six years old had an effusion of fluid into both knee-joints as the result of rheumatism. The synovial membranes were visibly distended, and fluctuated to the touch. When this condition had lasted a fortnight a blister was put upon the right knee, which was the more distended. This produced considerable vesication, after which the joint, instead of being larger than the other, was found to be smaller. The left joint being now the larger was painted with a concentrated solution of iodine, which produced much irritation but no vesication, and no decided change in the size of the joint. A blister being now applied to this knee, the fluid diminished as it had done in the other limb—the left knee now becoming, as at first, smaller than the right. Subsequently a blister was put at the same time upon both joints; after which no trace of fluid could be detected in either.

In this case the removal of fluid was accomplished by

vesication, not by simple irritation, and within the territory of the vessels which supplied the serum, not at a distance.

A local discharge, then, acts by changing the composition of the blood. This effect may be injurious or beneficial according to circumstances. When the discharge is of pus, lardaceous infiltration may result. When it is of serum, it may cause the removal of serum from elsewhere. Whatever the nature of the discharge may be, the first effect is within the territory of the blood-vessels which yielded it, the action diminishing as the altered blood is mixed with other streams, and only existing in a very reduced degree in the general circulation.

2. *Influences which travel by the absorbents.*

The function of the absorbents being, as far as we are aware, limited to the conveyance of matter from the circumference to the centre, it is unnecessary to consider their operation in any detail in connection with the salutary effect of local lesions. Insoluble pigments artificially placed under the skin, cancer, tubercle, and the syphilitic virus, all travel by these canals rather than by the blood-vessels; while the frequency of enlargement and congestion of the lymphatic glands in connection with discharging sores of almost every kind shows that the ordinary products of a broken surface are also liable to be taken up by these vessels.

This process, however beneficial to the locality from which the material is taken, is not likely to do good to any other part of the body, and can never be applied therapeutically. A local sore, therefore, cannot work medicinally through the absorbents.

3. *Influences which travel by the nerves.*

Many of the affections which are recognised as consequences of a local injury or disease appear to be conveyed by the nerves, and to reside essentially in the nervous system. Whether any influences so propagated can be

directed to the cure of disease, is a question which may find its answer in the consideration of their character.

A local irritation, though it be unattended by any discharge which can exhaust, or by any secretion which can poison, and though it be confined to structures which exercise no function immediately essential to life, may be productive of dangerous or fatal consequences.

According to the nature and extent of the primary lesion, the disturbances which result may be general throughout the system, or may be limited to particular nerves.

The following may serve as examples of a general change in the nervous system as the consequence of a local irritation.

If a thin layer of fæces or pus be spread over the peritoneum, a condition of collapse is produced which seldom fails to destroy life in a short time, although there may be no mechanical interference with the function of the abdominal organs. In such cases, indeed, death generally takes place more rapidly than would be the case from mere suspension of the functions of the alimentary organs. A small ulcer in the vermiform appendix, in itself unimportant, causes rapid death if it lead to effusion of irritating material over a considerable surface of the peritoneum. We can only attribute the result to the influence which the irritated surface produces upon the nervous system, the effect being chiefly manifested upon the nerves which regulate the circulation. The pulse becomes small, the surface cold, the features pinched, and, as appears after death, the left ventricle of the heart closely contracted.

Similar consequences may follow the burning or scalding of a considerable surface of cuticle, though the amount of skin destroyed may have been unimportant as far as regards cutaneous function. Death is produced, as in the former instance, by a failure of circulation.

Similar results have been known to follow accidents and operations which have been attended with no considerable loss of blood, and have involved no organ immediately necessary to life. And it would be easy to multiply

examples of the fact that local disturbances affecting portions of skin, or of serous membrane, or other structures which could for a time be spared without serious injury to health, may be followed by the failure of circulation which constitutes collapse, although there have been no such loss of blood or its constituents as could suffice to produce the result.

If the patient survive the period of collapse, a febrile condition may supervene, which is probably due, like the collapse, to an influence which the nerves convey.

Regarding collapse and reactionary fever as consequences affecting the entire circulation through the nervous system, we may next proceed to inquire what localised results are produced through the same channels by lesions too small to give rise to such general effects.

The more limited results of a local irritation, regarding such only as travel by the nerves, are of two kinds.

First, structural changes in the nerve-centres, more especially in that upon which the irritated nerve impinges.

Secondly, affections which have been described as reflex; alterations in the function of nerves distributed to some part of the body distant from the source of irritation, but connected with it by central attachments.

As examples of structural changes in the nerve-centres, as the consequence of a superficial irritation, the following cases may be mentioned.

It is well known that permanent softening of the lower part of the cord, with consequent paraplegia, not infrequently happens as the result of masturbation or excessive sexual indulgence.

A case is recorded in which acute inflammation of the brain followed the application of a ligature, which was intended for the subclavian artery, to the brachial plexus.

Tetanus, the pathology of which we are now beginning to understand, may be described if not as inflammation of the cord, as only one step short of it. In this case we are able to discern the important share which the arteries and their nerves bear in the production of change in the nerve-

centres. A peripheral irritation is conveyed to the arteries of the cord, which become paralysed, dilated, and unnaturally gorged with blood. Portions of their accumulated contents traverse their overstretched walls and mingle with and disturb the surrounding nerve-tissue.[1]

Epilepsy also appears to be due to a change in the innervation of the vessels of a certain part of the nervous system, in consequence of an irritation which may be conveyed from the periphery. It is believed that the vessels which are altered in capacity are those of the base of the brain.

Passing now to the second class of cases, a local irritation may be reflected by a nervous centre (according to the phraseology of the present day), and cause a 'reflex' affection somewhere else. This reflex affection may be of three kinds; loss of motor power, loss of common or special sensation, inflammation.

As instances of the loss of motor power in consequence of a reflex irritation, the following examples may be mentioned. Paraplegia has been known to occur as the sequence of intestinal disturbance.[2] A child under my care had paralysis of one leg, after having had a severe chilblain upon the corresponding foot; and I may mention a similar case recorded by Dr. Graves, in which loss of power in one leg accompanied an attack of erysipelatous inflammation of the calf, and passed off upon the subsidence of the cutaneous affection.[3] In the preceding cases the motor nerves have been chiefly affected; but it not infrequently happens that the sensitive faculty is similarly suspended or destroyed by an irritation acting primarily upon a distant part. This is especially the case with the retina; amaurosis has often been known to result from irritation conveyed by the fifth nerve. A splinter of wood in a carious tooth caused amaurosis, which was cured by the removal of the tooth. An injury connected with the supra-orbital nerve has been

[1] It is scarcely necessary to point out that when this was written the bacillus of tetanus had not been discovered.

[2] Graves' *Clinical Lectures*, vol. i. p. 547.

[3] *Clinical Lectures*, vol. i. p. 557.

known to cause amaurosis, which ceased on the section of the nerve. The same symptom has resulted from the impression of cold upon the surface of the face.

A circumscribed irritation may, by a reflex action, cause inflammation, or in other words, paralysis of the vaso-motor nerves, and consequent dilatation and repletion of the blood-vessels.

If the conjunctiva of one eye become inflamed, as often happens from the use of the microscope, the conjunctiva of the other eye will often undergo the same change though it has not been exposed to the irritation. A serious disease in one eye is not infrequently followed by destructive inflammation in the other.

It appears, then, that as regards 'reflex' nervous affections they may consist of loss of motor power, loss of common or special sensation, or loss of vascular contractility, giving rise to congestion or inflammation. The alteration, of whichever kind it may be, occurs in a situation which is determined by nervous connections; the source of the irritation and the seat of the disturbance deriving their nerves from a common source. It appears in the highest degree probable, considering the nature and situation of these 'reflex' affections, that they are really, one and all, occasioned by a morbid change, though perhaps only of temporary nature, in the nerve-centre which is the recipient of the irritation. It is not my object, however, to discuss at the present time the nature of reflex nervous affections, but simply to define their general character as loss of function in motor, sensory, or vaso-motor nerves.

It appears, then, that a local irritation may, by means of the nervous system, produce the following results:

1. Affecting the nervous system generally; collapse and febrile disturbance.

2. Affecting certain nerve-centres; structural changes in the nerve-centre upon which the irritation falls, mostly of an inflammatory or congestive nature; such as acute inflammation, chronic softening, or the congestion which is associated with tetanus.

3. 'Reflex' disturbances, or changes in the innervation of parts of the body remote from the source of irritation, though associated with it by central connection. These affections generally consist of a loss of nervous function, such as paralysis, loss of common or special sensation, or inflammation.

Whether a local irritation produce constitutional disturbance, central change, or reflex loss of function, it does not appear that, with our present knowledge, either of these consequences can be made subservient to the cure of disease. With regard to the reflex effects, which are the least mischievous, even they are of a kind which it can seldom be advisable to produce. And were such results as desirable as they are the contrary, they are not under our command; we can neither produce them at will, nor regulate their direction.

4. *Influences which travel by continuity or apposition of structure.*

A local sore or source of irritation may extend by contiguity of tissue; in other words, the original disease may spread.

A cancer or maligant tumour, by virtue of the energy of its nutrition, grows at the expense of surrounding structures; the continuous pressure to which they are subjected causes them to become atrophied, and eventually broken down and destroyed, to be replaced by the younger and more vigorous growth. Beside proceeding by this high-handed method, killing and taking possession, a malignant growth makes its way also by disseminating its germs into the vacant spaces around it, advancing not only by conquest but by colonisation. Each cell has an independent vitality, and a power of reproducing itself. Every outlying cell, every cell which by the movements of the body, or other chances, has become detached from the primary growth, becomes the centre of a new formation. Thus malignant growths cross the cavities of the pleura or peritoneum, cells formed on one side being rubbed off, and fixing themselves on the opposite

membrane; in the same manner the same formation may cross the vagina or any mucous cavity of which the sides come into contact, passing from one wall to the contiguous part of the opposite wall. By these means, then, independently of vascular connections, a malignant growth may make its way from the surface of the body, through solid textures and across cavities, extending by continuity or apposition of structure.

Taking a disease other than malignant, such, for instance, as a circumscribed area of inflammation, such as might result from a superficial injury or ulceration, the process may travel inwards through contiguous structures, much after the manner of a malignant growth. The products of inflammation may infiltrate adjoining tissues until they reach deeply-seated organs, which may then become the seat of a change resembling that which commenced around the original sore. An injury of the scalp may thus give rise to meningitis. The bone subjacent to the wound becomes infiltrated with pus, the dura mater underlying the bone takes on a suppurative process, which thence spreads to the arachnoid cavity; so that at last a state of suppurative meningitis has resulted from an injury at first confined to the outer coverings of the skull. In the same manner pleurisy may result from superficial injuries or morbid affections of the chest-wall, or peritonitis from apparently trifling alterations external to the peritoneum. This affection has been known in several cases to result from very trifling operations in the neighbourhood of the peritoneum, such, for example, as the destruction of vascular excrescences near the orifice of the female urethra. In such cases there can be little doubt that the fatal inflammation has been set up by a simple extension of the suppurative process through continuous or apposed structures. I might instance a case in which an operation for fistula was followed by the infiltration of pus into the cellular tissue around the rectum, which in its turn set up fatal peritonitis. But it is not necessary to multiply cases to prove that the inflammatory process set up by a super-

ficial lesion may extend by simple contiguity to a neighbouring serous membrane. The spreading of inflammation from one structure to another appears to be due to the fact that inflammation may be excited by the irritation of inflammatory products, so that the process may be conveyed by contact, after the manner of a malignant growth. This appears to be the truth, which, as far as morbid actions are concerned, is contained in the doctrine of contiguous sympathy.

We may now place in small compass what has been advanced, and review the consequences which can be recognised as springing from a circumscribed morbid process.

*By vessels.*

As a means of withdrawing material from the blood, a discharging sore may produce various effects according to the nature of the discharge and the circumstances of the case. A loss of pus may cause mischievous deterioration of the blood, and consequent organic change. When the discharge is serous, it may lead to the absorption of serous accumulations in the vicinity, and in appropriate cases be remedial.

A sore may produce various morbid changes consequent upon the contamination of the fluids of the body by the absorption of morbid products.

*By nerves.*

Independently of discharge, a local irritation may, if extensive, cause collapse and febrile disturbance.

It may cause change in the nerve-centre upon which the irritation falls, such as to give rise to acute inflammation, chronic degeneration, or the vascular alterations which belong to epilepsy and tetanus.

It may cause 'reflex' loss of function in especial nerves which are associated by central connection with the nerves which are the channels of the irritation, and so occasion localised paralysis, loss of sensibility, or inflammation.

*By contiguity.*

A superficial morbid process may extend by continuity, and occasion a change of the same nature in deeper structures.

We have inspected all the lines of communication which traverse the body, and found none which are adapted to convey the traditional virtue of counter-irritation in the direction which it is reputed to follow.

When therapeutical results ensue from an artificial discharge, as when vesication removes a serous effusion, the course of the salutary action is guided by the distribution of the blood-vessels. When simple irritation has been established—a proceeding of which we can recognise none but injurious effects—the nerves supply the route by which it travels. When, irrespective of vessels or nerves, a deep organ is influenced by a superficial change, the process is limited to extension by contiguity, the deeper structure participating in the morbid change which began on the surface, or in the inflammation consequent upon it.

Although in certain specific diseases the morbid action sometimes transfers itself from one place to another, we cannot expect such a translation except in disorders which, like measles and scarlatina, depend upon a circulating and erratic poison. And even under these circumstances we can rarely, if ever, produce artificially the desired transfer.

A local application has a local action; it warms or cools, soothes or stimulates, or produces its appropriate effect, be it what it may, upon the tissues which lie within the short range of its immediate influence. If the skin be made to pour out a serous discharge, the serum may be withdrawn from a neighbouring accumulation. But we have no knowledge which will warrant us in ascribing any remote or indirect remedial action to the excoriations and other local inflictions which have been practised under the idea of counter-irritation. We have no reason to suppose that we can under any circumstances lessen an internal

inflammation by exciting inflammation of the superincumbent but disconnected skin. Unless there be some great undiscovered law, of which pathology shows no trace, and of the existence of which clinical experience has given no proof, we cannot hope for benefit from counter-irritation. We may therefore cease to apply irritations to the skin of the head in disturbance of the brain; to the back in affections of the spinal cord; to the chest in diseases of the lung; and in general forbear to apply remedies to parts which have no direct vascular connection with the structure diseased, unless the remedies are of such a kind and of such magnitude as to bring the whole system under their influence.

P.S. 1895.—Although this paper was written twenty-seven years ago, there is little in it which I should now wish to modify. The practice of *derivation*, drawing blood or serum out of a suffering part by vessels in communication with it, has uses which I have sufficiently admitted. The irrational application of *counter-irritation* has been considerably restricted and modified since the publication of this essay, and the discussions to which it gave rise. Whether the practice will ever take a place in rational medicine I know not; it certainly has not done so yet. The nervous system is the *terra incognita* of therapeutics.

If we can benefit the stomach or the lung through the nerves of the skin it has yet to be inquired what superficial nerves are in relation with these deep and remotely connected organs, and what salutary influence can be directed upon them by such channels. The 'happy-go-lucky' method of attacking the superincumbent skin regardless of the course of nerve and vessel is simple, but not satisfying. Two advantages indeed may be admitted. The blister, if this be the irritant employed, sometimes, but not always, indicates the place of the disease and provides an outward and visible sign of the mischief underneath. And what is better still, it shows the patient that something is being done which he hopes may prove to his advantage, and soothes his mind by excoriating his body.

## *ON THE ENLARGEMENT OF THE VISCERA WHICH OCCURS IN RICKETS.*[1]

It has long been known that with rickety children certain of the abdominal organs are apt to increase in bulk. Dr. Whistler, who gave in his dissertation 'De Morbo Puerili Anglorum,' published in 1645, the first account which we have of *the rickets*, as the complaint was even then popularly termed, described an enlargement of the liver and spleen as pertaining to the disease, and considered the 'obstruction' in these viscera as the first departure from health. He introduced the more than sesquipedalian word Pædosplanchnosteocace, as indicating that this disorder of childhood affected both the viscera and the bones.

Glisson, in his elaborate treatise upon rachitis, published five years later, refers to enlargement of the liver and mesenteric glands as belonging to the complaint, but considers that the spleen is not affected except as an accidental complication.

Dr. Bright,[2] in the year 1838, described an enlargement of the spleen as occurring especially in early childhood, though he did not associate it with the rickety condition. He described this organ as becoming hard like a half-ripe apple, as having on section the lustre of damson cheese, as presenting numerous opaque whitish granules (apparently the enlarged Malpighian corpuscles), and as being swollen to such an extent as often to occupy the greater part of the abdominal cavity.

It appears, however, that little attention had of late years been directed to the state of the abdominal organs in rickets until Sir William Jenner, in his well-known lectures,[3] dwelt upon their changes in bulk and texture, and attributed them to an albuminoid infiltration. He

[1] 1869. *Medico-Chirurgical Transactions*, vol. lii. In the original paper are some illustrations which it was not practicable to reproduce.

[2] *Guy's Hospital Reports*, vol. iii. p. 401.

[3] *Medical Times*, 1860.

pointed out that in the rickety enlargement the organs differ from the lardaceous or amyloid state in the absence of reaction with iodine, and as regards the spleen in particular, in the absence of the sago-like transformation of the Malpighian corpuscles. He, however, made only a partial separation between the two conditions.

It seems that the alteration which the viscera undergo in rickets is still imperfectly understood, and is generally confused with the more common lardaceous or amyloid change, to which it bears a superficial resemblance while severed from it by essential differences.

I will now proceed to describe the changes which occur in the liver, spleen, and lymphatic glands in rickety children.

During the progress of rickets, if the disease be severe, and particularly if it be attended with emaciation, the liver is often found to extend below its normal situation, occasionally as low as the umbilicus, the smooth surface of the organ being very evident to the touch beneath the thin abdominal walls. The spleen under the same circumstances becomes enlarged, more frequently and to a greater extent, often acquiring seven or eight times its normal weight and occupying the greater part of the left side of the belly. This organ, dragged down by its increased weight, often lies with more of its bulk below than above the navel, the upper edge of the spleen being below and nearly parallel to the lower edge of the liver. A swelling often takes place at the same time in the absorbent glands, particularly in those of the mesentery, The glands which lie near the surface of the body are often evident to the touch like shots under the skin, but the swelling is seldom such as to cause visible protuberance.

Before dealing with these changes in their clinical results their morbid anatomy may be taken into consideration.

The liver, to commence with this organ, undergoes an increase of size evenly throughout its whole bulk. It becomes less friable than in health, hard, dense, and elastic. It is

usually pale in colour, containing little blood. The acini, themselves of yellowish colour, are often each surrounded by

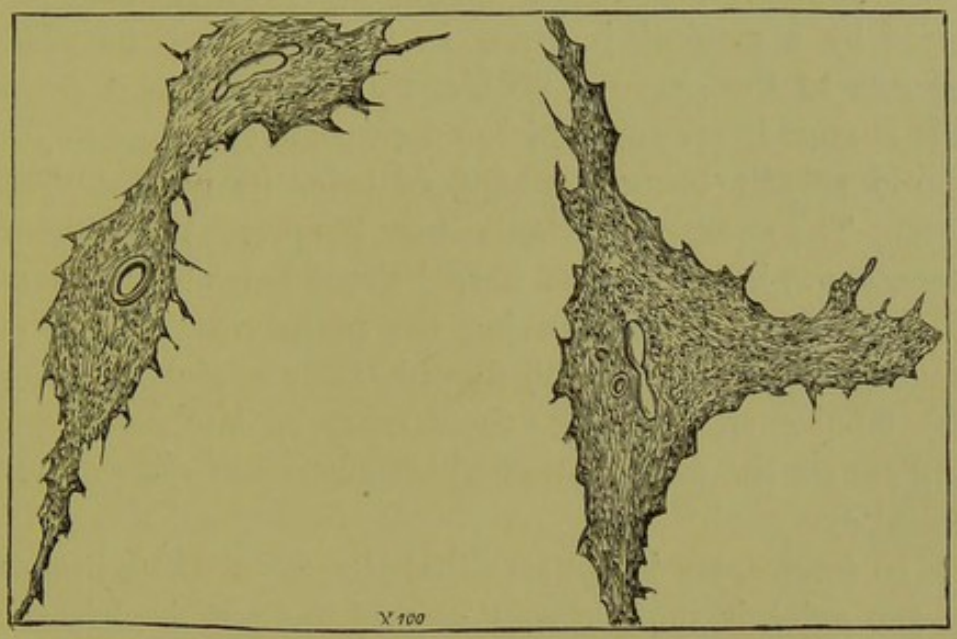


Fig. 4.—Portal canals in rickety liver, showing increase of fibroid tissue.

a thin pinkish or grey line, so that the cut surface is covered with a fine uniform polygonal pattern, such as may

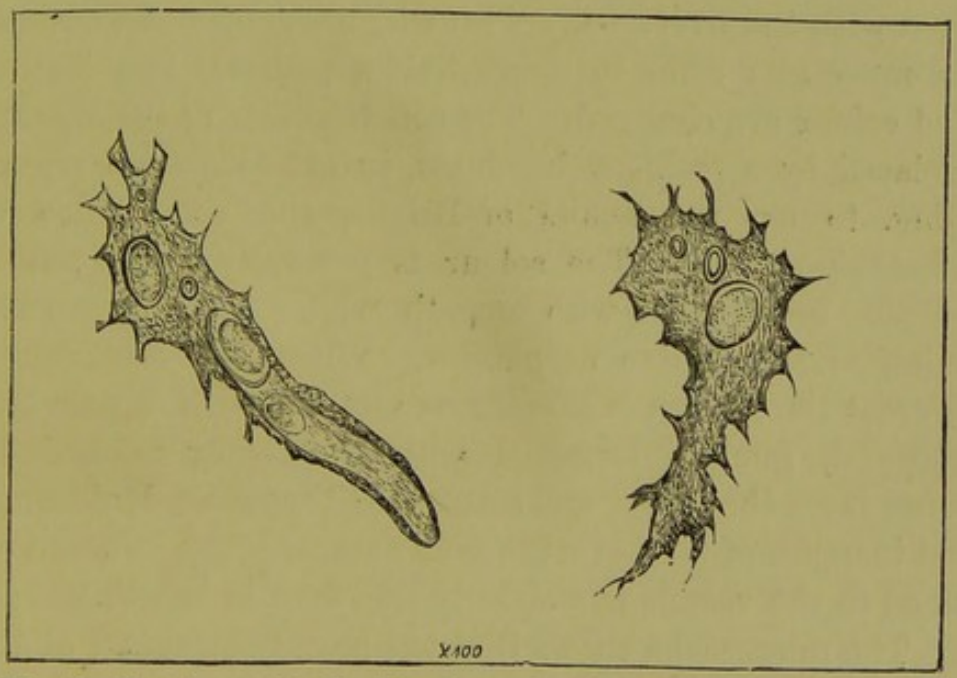


Fig. 5.—Portal canals in healthy liver, showing relation of fibroid tissue to vessels.

be produced when from any cause there is an increase in the fibrous tissue. Sometimes in these cases it is evident to

the naked eye that there is an increased quantity of loose material surrounding the smaller branches of the portal vein. The alterations which have been described are, in fact, produced by a general increase in the fibroid tissue which belongs to the organ. Within the smaller portal canals this change is very evident to microscopic examination, the fibroid sheath being, perhaps, of twice its normal thickness. The annexed woodcuts show the proportion which in disease and in health the fibroid tissue bears to the vessels it surrounds. Accompanying the portal vessels, and extending beyond them, the fibroid tissue stretches between the lobules, circumscribes them more or less completely, and causes the demarcation which is so often evident to the naked eye.

In some cases it appears that the cell-holding network of the acini is more closely packed than in health, as if from an excess of epithelial growth. The epithelium is sometimes fatty, sometimes almost free from oil. In this respect there is no constant change.

With the spleen the increase of bulk is usually greater than with the liver. A spleen which ought not to weigh an ounce may come to weigh half a pound. The texture and colour are changed. The soft friability of the organ is replaced by a resilient hardness, in extreme cases resembling, to use the simile of Bright, the consistence of a half-ripe apple. The colour is generally a deep red or purple, besprinkled with smooth white spots, which are enlarged Malpighian corpuscles. When the enlargement is great the section is sometimes mottled with a pale buff material, finely intermixed with the deeper colour. In some cases the colour and texture are thus altered, although the increase of bulk is trifling or absent. The amount of blood in the vessels is generally less than in health.

The microscope shows that the minute structure of the spleen is modified in the following respects.

There is an increase in the delicate reticulum in which the splenic pulp is immediately contained. This network in the healthy spleen is of extreme tenuity, the threads

slender, the interspaces comparatively large. In the rickety spleen the trabeculæ are often irregularly swollen, sometimes to such an extent that where the threads are thickest they may be as wide as the spaces they enclose.

The larger fibrous divisions of the spleen are often widely separated by the swelling of the tissue between them, but in other particulars they are usually unaltered. In the case of a rickety dwarf, who died at the age of twenty-six, they were found to have become thickened.

In a case where the spleen was characteristically altered in consistence, but not increased in bulk, it was found that while the reticular basis of the organ was abnormally developed, the cellular and corpuscular contents of the network had become atrophied, being replaced by shapeless masses of blood-coloured or rusty matter.

When (as in the case of Wood, below) great enlargement has occurred, there is, in addition to the change in the trabecular tissue, an abnormal development of the contents of the meshes, the corpuscles being crowded together in a manner distinctly different from what occurs in health.

The absorbent glands, particularly those of the mesentery, are often enlarged and hard. On section they are white and opaque, in consequence of an accumulation of their cellular and corpuscular contents.

The kidneys are often increased in size, smooth and pale, the convoluted tubes being more than naturally distended by epithelium. I have not been able to discover any other change in their structure.

None of the organs affected as described give any reaction with iodine.

The change which occurs in the rickety viscera are due, not to any foreign growth or deposit in their tissue, but to an irregular hypertrophy of their fibroid or epithelial elements conjoined, as there is reason to believe, with a deficiency of their earthy salts. It thus appears that the alteration in the viscera is closely analogous to that which occurs in the bones.

The following cases illustrate the preceding statements.

Case I.—William Wood, 7 months old, was brought to me at the Hospital for Sick Children, suffering from rickets and abdominal enlargement. He was a twin. He had been fed entirely upon artificial food—milk and biscuit. The parents had had three other children, all of whom had died, as I was told, with abdominal enlargement and symptoms similar to those observed in the present case. The child to whom the present statement refers failed in health from the age of four months, when he began to lose flesh, to vomit frequently, and to increase in bulk about the belly, the latter symptom having been very conspicuous for the last six weeks. He had had otorrhœa from birth. There had been no symptoms to lead to any inference of syphilis.

The child had nodulated ribs, and the external signs of rickets to an extreme degree. The chief complaint was of diarrhœa and vomiting. He was emaciated and anæmic, and had the waxy complexion of splenic disease.

The belly was greatly enlarged, owing apparently to great increase in the size of the liver and spleen. The liver extended from its normal position downwards to the level of the umbilicus, its lower edge passing in a slanting direction from the ensiform cartilage to the right iliac fossa. The spleen had fallen below its usual situation, and occupied the greater part of the left side of the abdomen between the thorax and the pelvis, while a small portion of the organ could be felt on the right of the median line. These organs were traced in outline upon the skin, presenting the configuration shown in the annexed diagram (fig. 6).

In spite of nourishing food, cod-liver oil, and the iodides of iron and potassium, the child gradually sank, without any material change in the symptoms, and died five days after it was brought to the hospital.

At the post-mortem examination the swelling of the belly proved to be due, as anticipated, to enlargement of the liver and spleen, which occupied the position assigned to them during life.

The liver, which, under the circumstances in which the examination was made, could not be weighed, was much increased in size. It was smooth externally, firm in texture, and of a pale fawn colour. The acini were preternaturally distinct, each being surrounded by a pale pinkish-grey line, so that the section was covered with a honeycomb pattern of adjacent hexagons. Sub-

sequent examination showed that this appearance was produced by an increase of the fibroid tissue which normally surrounds the portal vessels and makes its appearance at intervals between the lobules. Though the colour of the liver was suggestive of fatty change, the epithelium, which was finely granular and opaque, was unusually free from oil-globules.

The spleen weighed seven and three-quarter ounces, probably at least seven times its normal weight. It was firm and consistent, having much the feel of the lardaceous spleen, which, how-

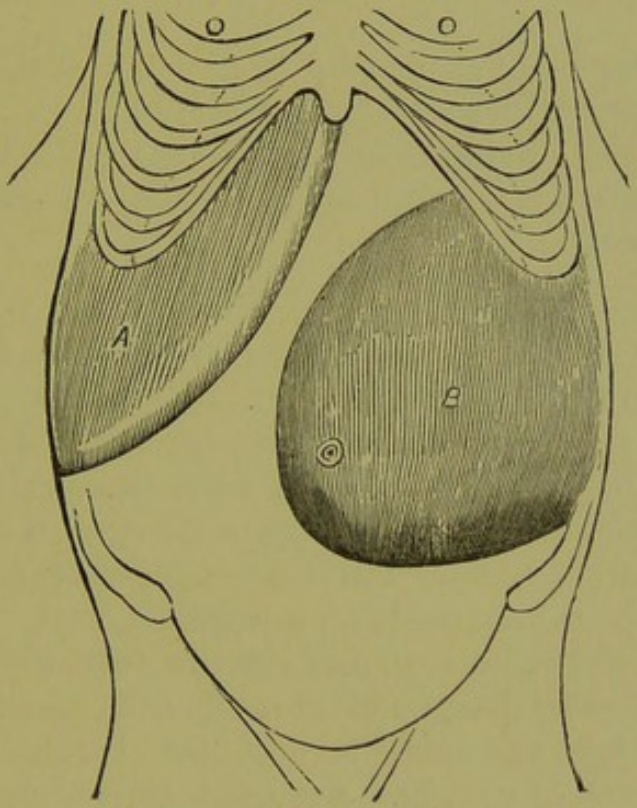


Fig. 6.—*A*, Liver. *B*, Spleen.

ever, it did not in other respects resemble. On section it appeared to consist of a fine intermixture of a red and a pale buff material, the lighter disposed in small specks imbedded in the red. Under the microscope it was found that the larger fibrous divisions of the spleen were much further apart than in health, owing to a great increase of the splenic structure between them.

The increase of this tissue was in great part owing to a swelling of the delicate translucent network in which the cells and corpuscles are immediately contained. The swollen trabeculæ lay in large and irregular breadth, covering a considerable proportion of the section, instead of existing, as in health, in the form of slender

threads, which, from their tenuity, lie almost concealed by the cellular and corpuscular elements they enclose.

Besides the increase of the trabecular tissue there was an absolute increase in the cells and corpuscles of the pulp. These were closely packed in the cavities of the swollen matrix, and were often in such close apposition that by mutual pressure many of the larger cells had been rendered polygonal.

The kidneys were perfectly natural.

The lumbar glands and all the absorbent glands within the abdominal cavity were swollen, and congested to a purple colour.

The pericardium was occupied by firm thin adhesions. The lungs were congested, particularly at their bases.

The organs which have been mentioned, as well as the stomach and bowels, were tested with iodine without the display of any 'amyloid' reaction.

There was no tubercle in the body.

Case II.—Ellen Smith died under my care at the Hospital for Sick Children at the age of four years. Since she was a year old she had had swelling of several of the joints and of the hands. When in the hospital she was evidently rickety, the cachexia being strongly developed, while the change in the shape of the skeleton was comparatively slight. The child was emaciate and anæmic, the complexion earthy, the teeth decayed.

She was pigeon-breasted, and the ribs were swollen at their cartilaginous ends, though not so much as is sometimes seen. The wrists, knees, and ankles were swollen, though the shafts of the bones remained straight. A remarkable point in the case was the manner in which the symptoms simulated those of osteo-arthritis.

There was a peculiar swelling of the fingers, apparently due to enlargement in the substance, or in the immediate neighbourhood of the first phalangeal joints.

Almost all the limbs and joints were at times the seat of severe aching pain, which varied with every change of weather, and were aggravated by the least movement.

The abdomen was prominent: the spleen was enlarged, being evident to the touch for a distance of two and a quarter inches below the ribs. The liver was also enlarged, reaching below the ribs for an inch and a half (fig. 7).

Her life was brought to a close by a febrile attack, which began

with slight inflammation of the tonsils, was attended with severe diarrhœa, and showed, at last, the symptoms of pneumonia.

At the post-mortem examination the bones proved to be extremely rickety; their articular ends were large and soft, and their cancellous tissue contained a deposit of red glutinous matter. The insides of the joints were natural.

The swelling of the fingers was due to an increase of the cancellous tissue of the first and second phalanges, like that which was found in the ends of the larger bones.

The pleuræ and pericardium were occupied by old adhesions.

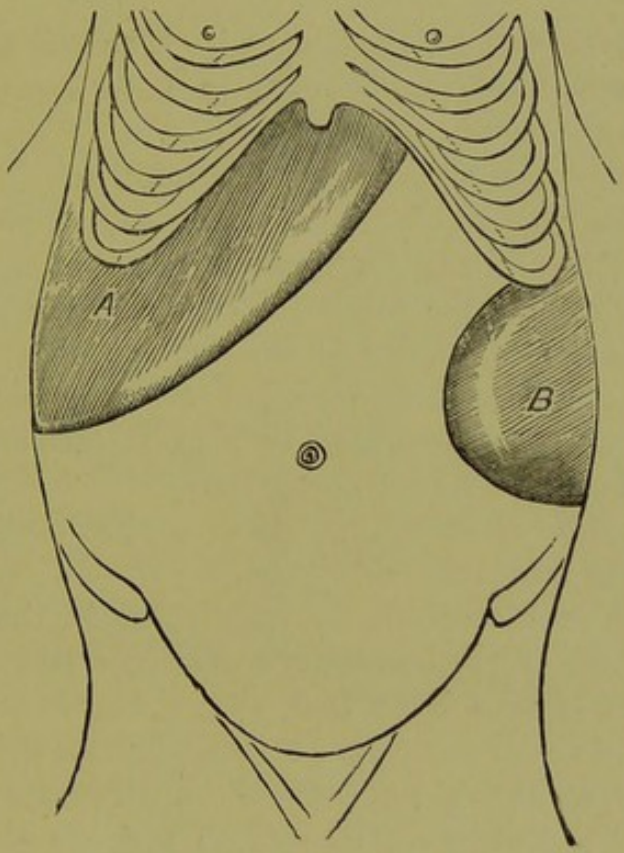


Fig. 7.—*A*, Liver. *B*, Spleen.

These were small recent vegetations upon both mitral and tricuspid valves. The lungs were congested, and in patches gave evidence of lobular pneumonia.

The liver was attached to the diaphragm, the spleen to the diaphragm and abdominal wall, by old adhesions.

The liver weighed eighteen and a half ounces. It was resistent and tough; generally of a pale fawn colour. The acini were indistinct in outline. The section was traversed by vessels which to the naked eye had an exaggerated appearance, which the microscope showed to be portal veins surrounded by an excess

of fibroid tissue. The epithelium, which was very closely packed, was slightly fatty.

The mesenteric glands were much enlarged. In section they were hard, close, and firm, the larger being opaque like the cortex of a kidney affected by scarlatinal inflammation ; their appearance being suggestive of epithelial accumulation. On section it appeared that the increase of bulk was due to a great increase in the cells and nuclei belonging to the gland, more particularly of the former, which pervaded the whole tissue with an abnormal uniformity. No change was found in the fibrous stroma.

The kidneys were pale and smooth. The pair weighed four ounces. Small ulcers were found in Peyer's patches at the lower end of the ileum.

None of the organs gave any iodine reaction.

The spleen weighed eight and a half ounces, and measured five and a half inches by three and a half. It was rigid and hard, crushing under the fingers with a grinding sound. The colour was deep red, which was closely besprinkled with white Malpighian corpuscles. The section under the microscope was unusually dense, chiefly owing to a great increase and close arrangement of the cells and corpuscles.

The reticulum of the spleen was somewhat exaggerated, but had undergone no marked alteration.

A portion of the spleen was incinerated for the purpose of ascertaining the proportionate amount of its earthy and alkaline components. It was found that, as in rickety bones, there was a deficiency of the earthy part. The results obtained are placed in apposition, for the sake of comparison, with a similar analysis of the ash of a healthy child's spleen ; and one also of a lardaceous spleen obtained from a child who had died of phthisis with large vomicæ. The diminution of the earthy salts in the rickety spleen is sufficiently evident.

Passing to the clinical part of the question, while it is not necessary that I should repeat much which is well known as regards the history and results of rickets, there are several points to which I must draw attention.

The condition of the viscera which has been described is, as far as my observation has gone, peculiar to childhood, belonging especially to the first four years. It usually occurs in connection with the well-known signs of rickets as they affect the bones. I

have never seen it well marked except in such conjunction, though sometimes the visceral appear to precede the osseous changes; and it not seldom happens that the visceral change may be extreme when the modification of the skeleton is but slight.

*Mineral constituents of* 100 *parts of fresh spleen.*[1]

| | Earthy salts | Alkaline salts | Composition of alkaline salts | | | | |
|---|---|---|---|---|---|---|---|
| | | | Potash | Soda | Phosphoric acid | Sulphuric acid | Chlorine |
| Rickety spleen (Ellen Smith) . . . . . | ·094 | ·989 | ·283 | ·116 | ·055 | ·025 | — |
| Healthy spleen . . | ·274 | 1·054 | ·322 | ·115 | ·118 | ·215 | ·096 |
| Lardaceous spleen . | ·258 | ·658 | ·218 | ·115 | ·070 | ·042 | ·105 |

The rickety state of the viscera, like the alteration in the texture of the bones, is transient in its nature. Under favourable circumstances the affected organs have a strong tendency to recovery, and even when swollen to the utmost will occasionally return to their natural dimensions.

The change in the abdominal viscera appears to interfere comparatively little with their functions. The swelling of the spleen, indeed, when considerable, often is accompanied by a tint of skin suggestive of leucocythæmia; but the excess of white corpuscles in the blood, as shown by microscopic examination, is comparatively slight.

The change in the liver is unaccompanied either by ascites or jaundice, and though the kidneys may be considerably enlarged, the urine remains free from albumen.

[1] I may state that in three healthy spleens the alkaline salts in 100 parts were found to vary from 1·054 to 1·092, the earthy salts from ·121 to ·274. From this it would seem that while in the healthy and the rickety organ the amount of alkaline salts is nearly the same, the earthy salts exist in health in a decidedly larger proportion than was found in the rickety spleen which was examined. I regret that I am not able to give further observations as to the mineral constituents of the viscera in rickets; future opportunities may supply the deficiency. Cases of considerable splenic enlargement from rickets occur so seldom in post-mortem examination that a considerable time may pass before another analysis can be made. The difference between the rickety and the lardaceous spleen is very striking, one being deficient in the earthy, the other in the alkaline, constituents.

When the visceral change has taken place to a considerable extent the child is generally emaciated and anæmic, having either the earthy complexion of rickets or somewhat of the waxy look of splenic disease.

Such a child is especially liable to be attacked by the diarrhœa, bronchitis, or pneumonia, to which rickety children are prone, and these affections constitute the chief danger to which it is exposed.

The following case illustrates the symptoms and the favourable issue of the disorder which has been described.

Case III.—Frederick Grant, fifteen months old, came under my care at the Hospital for Sick Children, in June, 1868. He was the second child of apparently healthy parents, the first being in good health. He had formerly been able to walk, but had failed in health after an attack of whooping-cough. He now could neither walk, nor sit upright, nor even hold up his head, but lay helpless, as his mother expressed it, 'all of a heap' in her arms. His joints were enlarged, the anterior ends of the ribs presenting globular swellings. The chest was laterally compressed and the anterior fontanelle was widely open. He had the peculiar waxy pallor suggestive of splenic disease. The belly was prominent, and both the spleen and the liver were much enlarged, their outlines being easily traced through the thin integuments. The spleen had fallen below its normal situation, and lay in great part below the umbilicus, barely touching the lateral edge of the thorax above, while it stretched downwards to the outer end of the groin, and crossed the median line, so that the extremity of the organ lay a little to the right of the navel. The liver was felt bulging downwards nearly as low as the umbilicus, in such a position that its lower edge was nearly parallel to, and a little above, the upper edge of the spleen. The position of the viscera is shown in the accompanying diagram (fig. 8). The urine was free from albumen and from excess of phosphates.

The child was still at the breast. It was ordered to be weaned at once, and had cod-liver oil, which was not well borne, with quinine and perchloride of iron.

A considerable quantity of blood was now passed from the bowels with diarrhœa, an occurrence which was immediately followed by a marked diminution in the bulk of the liver. The child did not at first improve, but became more anæmic and waxy in aspect. It had frequent diarrhœa, and further suffered

from an attack of bronchial catarrh. It was found, however, that cod-liver oil, in an emulsion with lime water, was retained, and by the help of wine and raw meat he began to amend.

When seven months had elapsed there was a marked improvement in every respect. The child was plump, much heavier, and had a more healthy tint of skin. The bowels were regular; the appetite good; and the child could now use its limbs freely, though still unable to stand. The spleen and liver had much diminished in size, the spleen measuring only two and three-quarter inches from above downwards, the liver projecting only two inches below the thorax.

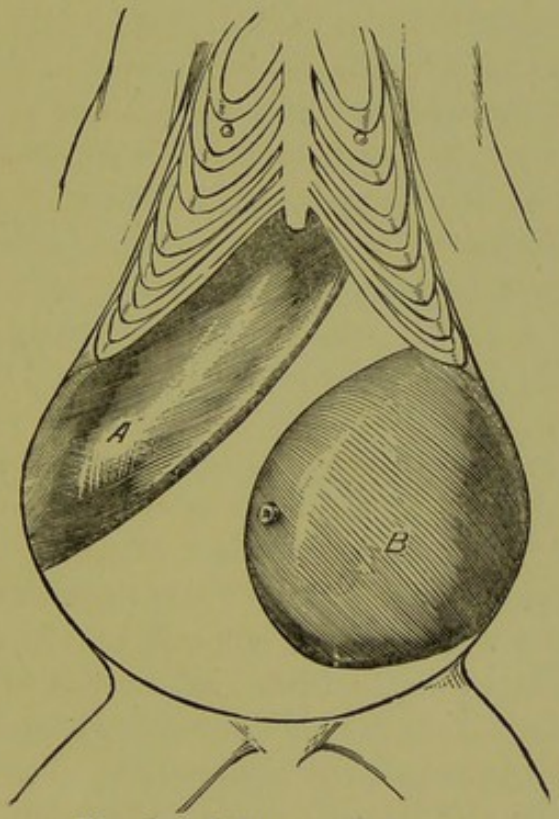


Fig. 8.—*A*, Liver. *B*, Spleen.

The cod-liver oil was now taken, unmixed, without trouble, and the improvement continued steadily. After about ten months the child had lost nearly all trace of the visceral enlargement, and was proportionately improved in other respects.

The spleen was entirely out of reach. The most careful examination failed to detect any sign of enlargement in this organ. The liver had diminished in bulk, insomuch that it could be felt only for an inch below the edge of the thorax, and this only with some difficulty. The general health of the child had improved in like measure. The appetite was good. He was now

rosy in complexion, and had the tint and plumpness of perfect health. He could stand alone and walk for a few steps.

There still remained some evidence of rickets, as evinced by the backwardness in walking, and by want of closure of the anterior fontanelle. In other respects the child seemed in perfect health.

That recovery may occur when the visceral enlargement is extreme, and the circumstances of the case are apparently desperate, the following statement will show.

Case IV.—In the early part of 1861, a child, eighteen months old, named Frances Lewis, the sixth of an otherwise healthy family, was brought to me at the Hospital for Sick Children. She had been falling off in health and flesh ever since she was a month old, and had never walked. She was weaned at nine months. When seen she was in a state of helpless prostration and looked as if she could not live many days. She was extremely emaciated, weighing only fourteen pounds. She was perfectly pallid, and had in a marked degree a waxy complexion like that of leucocythæmia. The joints, which were of rickety conformation, were remarkably flexible, so that the limbs could be moved in any direction almost regardless of their ligamentous attachments.

The belly was prominent; the abdominal muscles were loose and thin, allowing the outline of an enormous spleen to be traced with facility. The tumour which represented this organ was dull on percussion and lay close under the abdominal muscles. It occupied the whole of the left side of the abdomen, from beneath the ribs to within from half an inch to an inch of the fold of the groin. Near the hip the tumour approached the groin more closely than towards the median line. The position of the lower edge of the spleen was marked on the skin with nitrate of silver, so that any variations which might occur in the bulk of the organ might be ascertained.

Cod-liver oil, iron, quinine, and nourishing food were ordered, while the ointment of iodide of potassium was rubbed daily over the enlargement.[1]

At the end of a month she had improved in appearance, gained a pound and a half in weight, and the edge of the spleen had moved up about an inch. The same process of amelioration continued, the gradual diminution of the spleen being shown by the elevation of its lower edge, until at the end of six months

[1] The rubbing with the iodide was probably useless.

this organ was almost out of reach of the fingers, being felt with some difficulty close under the ribs. The child now looked plump and well; it could stand alone, and bore but little resemblance to its former self. She was seen from time to time until nearly three years old, the same treatment being continued. She became fat and rosy. The spleen shrank to nearly its normal dimensions, so that it could hardly be felt in the hypochondrium. She remained backward in walking, but in other respects appeared to be in perfect health.

It will be observed that the treatment adopted in the preceding cases was that ordinarily required in rickets. I formerly thought that outward applications assisted to reduce the bulk of the spleen, but later experience has led me to rely upon internal not external remedies. The diet should be nutritious, and carefully adjusted, consisting of milk, beef-tea, meat, and wine, according to the age and condition of the patient, while medicinally cod-liver oil is a prime necessity, and iron and quinine seldom fail to be beneficial. The importance of fresh air is well known in cases of rickets; but this remedy, unfortunately, is not always at the command of the physician.

The preceding details lead to the following conclusions:—

The state of the viscera which has been described as rickety belongs especially to childhood, and is generally associated with the external signs of rickets. It takes place simultaneously in several organs, and is analogous to the alteration which, under similar circumstances, occurs in the bones.

The change in the viscera is due, not to the presence of any formation foreign to their structure, but to an irregularity of growth, which alters the natural proportions of their tissues. The epithelial and corpuscular element is generally increased, while in the liver the capsule of Glisson, and in the spleen the trabecular tissue, are abnormally developed.

The more observable functions of the several organs are but little affected, neither jaundice, ascites, nor albuminuria resulting.

Under favourable circumstances the viscera recover their natural condition, the disease yielding, sometimes readily, to treatment.

The disease which has been described is distinct from the lardaceous or amyloid change on the one hand, and on the other, from that enlargement of the spleen and absorbent glands which has been associated with the name of Dr. Hodgkin.

Rickety differ from lardaceous organs in the absence of the peculiar bacony translucency, in the absence of iodine reaction, and in the exemption of their blood-vessels from evident alteration.

In the lardaceous there is a deficiency of the alkaline constituents, in the rickety, probably, of the earthy.

The lardaceous change depends usually upon chronic suppuration or syphilis, the rickety has no such antecedents. The lardaceous change is rare in early childhood, to which period the rickety disorder especially belongs.

The lardaceous disease, unlike the rickety, generally gives rise to albuminuria, dropsy, and other derangements of function, and in its relation to treatment is eminently intractable.

The enlargement of the absorbent glands and spleen, to which Dr. Hodgkin drew attention,[1] is separated from the rickety change by important differences.

In the disease described by Hodgkin the glands, swollen by a new growth of fibro-nucleated tissue, as well as by an increase in their secreting or corpuscular element, sometimes cause conspicuous lobulated tumours on the surface of the body, a degree of enlargement which, as far as I am aware, is never attained as the result simply of rickets.

A more important difference is the production, in Hodgkin's disease, of a fibro-nucleated growth distinct from the tissue in which it lies, often resembling, to the naked eye, lumps of crude tubercle occurring chiefly in

[1] *Medico-Chirurgical Transactions*, 1832. See also a paper by Dr. Wilks in the *Guy's Hospital Reports* for 1865.

the spleen, but also in the liver, kidneys, lungs, and absorbent glands.

Hodgkin's disease is attended with grave symptoms of anæmia, prostration, and anasarca, and appears to have little tendency to recovery.

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## *ON THE MORBID EFFECTS OF ALCOHOL, AS SHOWN IN PERSONS WHO TRADE IN LIQUOR.*

In my work on 'Albuminuria' I brought forward facts which appeared to indicate that the injurious effect of alcohol upon the kidney had been exaggerated. My conclusions in their turn have been disputed. However, it is not now my purpose to pursue the subject controversially, but to lay before the Society some details which have been extracted from the records of St. George's Hospital, the searching of which I began with especial relation to the glands in question, but have pursued with a widened scope, having regard to the effects of alcohol upon the whole body rather than upon any organ in particular.

The character of the books I have referred to is here well known. They are the work of a line of competent pathologists; they are systematic and complete, and insomuch as the observations were made without special object, we may believe them to be generally unbiassed.

I have started with the assumption that a person in whose vocation alcoholic drink, to be had for nothing, is ever suggestively present, will on an average take more of it than one in whose calling such liquor plays neither a necessary nor a gratuitous part. It needs but a slight experience of human nature to justify this surmise. The mouth is not muzzled of the ox who treadeth the corn or of the potman who carries the beer. With a reliance upon this general truth, justified by the notorious inebriety and the liability to delirium

[1] 1872. *Medico-Chirurgical Transactions*, vol. lvi.

tremens of those who trade in drink, I have contrasted the morbid appearances in the bodies of people of this class with those in the persons of others who have neither been professionally conversant with intoxicating liquids nor known to have been addicted to their intemperate or excessive use.

It is scarcely necessary to observe that any pathological observations as to the effect of alcohol to be valid must be comparative. Bare statements that among so many dead drunkards many or few were tuberculous, while in such a number the liver was cirrhosed or the kidneys granular, convey little information. It is necessary to know how frequent such lesions are in persons similarly circumstanced in other respects save drink. I have, therefore, used a strictly comparative method, and have applied it to as large a number of dissections, and to as many particulars in each, as the materials permitted.

The post-mortem books of St. George's Hospital, which were begun with the year 1842, contain in chronological order a detailed account of every examination made from that date to the present time. Subsequently to the beginning of 1844 the history and clinical particulars corresponding to each are adjoined. From the commencement of 1842 to the end of 1871, a term of thirty years, I have abstracted the morbid appearances, and with the exception of the first two years, the leading clinical facts relating to every person examined after death whose trade it had been to make, sell, store, or convey alcoholic liquor. As a counterpoise or standard of comparison for each such case (not that it was designed to compare single cases one with another, but in order to obtain two series as similar as possible, save in respect of drink) I took after each alcoholic post-mortem one upon a person of the same sex, not employed about liquor nor known to have had delirium tremens or to have been of intemperate habits. In order to avoid bias in selection I went by rule. After each alcoholic examination I abstracted the non-alcoholic next afterwards, except where the end of the annual volume compelled me to turn

backwards instead of forwards. Since the alcoholic series began at the age of sixteen, all post-mortems upon younger subjects were excluded. Also were excluded those which referred to workers in lead, the effect of this metal upon the kidneys possibly tending so far to obscure the result.

Thus, were contrasted two series of post-mortem examinations the subjects of which had been differently placed with regard to drink, but in no other respect were obviously dissimilar. They were of the same sex, of similar age, they belonged to the same stratum of society, they had been patients at the same time in the same hospital, and were at last examined, and the results recorded, by the same observers.

Each series comprised 149 post-mortem examinations [1] —146 men, 3 women.

The traders in liquor were thus divided:

| | |
|---|---|
| Potmen | 54 |
| Waiters | 43 |
| Cellarmen | 11 |
| Draymen | 11 |
| Brewers or brewers' men | 9 |
| Barmen | 8 |
| Publicans | 8 |
| Barmaids | 3 |
| Distillerymen | 1 |
| Maltsters | 1 |
| | 149 |

The opposed class, that not concerned with alcohol, comprised, as will be seen below, a great variety of occupations. The lead trades were excluded for reasons which have been stated; while billiard-markers, butlers, wine-coopers, and excisemen were not admitted on either side, their vocations holding with regard to drink an uncertain attitude.

In the course of the inquiry, the questions will arise how far certain differences in the morbid proclivities of the

[1] Full details of every case, in a tabular form, were laid before the Society. These tables, which were very voluminous, formed the bases of the abstracts which are printed.

two classes are to be attributed to causes other than alcohol. Chiefly how far do the classes differ in exposure to weather, one undoubted agent in the production of rheumatic and inflammatory disease.

The non-alcoholic traders are therefore arranged so as to show as nearly as may be how many work in the open air, how many in houses, and with how many their place of labour is uncertain or variable.

*Occupations of the non-alcoholic traders, arranged according to their probable exposure to weather.*

| Indoor | | Mixed or uncertain | | Outdoor | |
|---|---|---|---|---|---|
| Footmen or pages | 6 | Grooms | 8 | Labourers | 21 |
| Clerks | 4 | Railway or other porters | 6 | Coachmen, carmen, or postilions | 16 |
| Smiths or metal turners | 4 | Carpenters | 5 | Gardeners | 5 |
| Tailors | 4 | Stablemen or horse-keepers | 4 | Travellers or collectors | 4 |
| Shoemakers | 4 | Old soldiers or pensioners | 4 | Commissionaires or errand boys | 3 |
| Schoolmistress or wife | 3 | Plasterers | 3 | Sailors | 3 |
| Bakers | 2 | Butchers | 3 | Watermen or bargemen | 2 |
| Upholsterers | 2 | Stonemasons | 3 | Milkmen | 2 |
| Greengrocers | 2 | Watchman, detective, sweep, engineer, shipwright, woodcutter, chaff-cutter, veterinary surgeon, of no occupation, of each 1 | 9 | Hawker, brickmaker, postman, of each 1 | 3 |
| Cooper, wheelwright, birdstuffer, ironmonger, mattress maker, dentist, merchant, hairdresser, draper, tallow-chandler, saddler, cabinet-maker, fishmonger, brush-maker, of each 1 | 14 | | | | |
| Total | 45 | Total | 45 | Total | 59 |

Hence it appears that, excluding the callings of uncertain or mixed character, the outdoor trades preponderate as 59 to 45, or about 4 to 3.

With the traders in liquor indoor pursuits are the more numerous. Waiters, cellarmen, brewers, publicans, barmen, barmaids, distillers, and maltsters, all generally under

cover, together amounted to 84 ; potmen and draymen, mostly in the open air, together came to 65. Thus, the ratio of 4 to 3 will nearly represent both the preponderance of indoor occupations with the alcoholic series, and of out-door occupations with the non-alcoholic.

Comparing the two classes, the alcoholic and the non-alcoholic, the results are these :

*Age.*

The alcoholic traders who die in the hospital attain a mean age of 36·8 ; the non-alcoholic a mean age of 40·6.

Excluding all deaths from accident, dealing solely, therefore, with the effects of disease, the figures are practically unaltered ; alcoholic traders dying at the age of 36·8, non-alcoholic at the age of 39·9.

It, therefore, may be stated with general truth that to deal in liquor costs 3½ years of life.

The shortness of life in both classes is, no doubt, owing to the fact that a hospital is essentially the resort of disease not of age. A workhouse would have given different results, though, possibly, the proportion would not have been much altered.

The following statement of the number of deaths in each decade of life in the two series shows that the mortality from alcohol is most marked between the ages of 30 and 40.[1]

| Age at death. | Alcoholic trade. | Non-alcoholic trade. | Age at death. | Alcoholic trade. | Non alcoholic trade |
|---|---|---|---|---|---|
| From 16–20 | 10 | 11 | From 51–60 | 12 | 21 |
| „ 21–30 | 37 | 39 | „ 61–70 | 5 | 13 |
| „ 31–40 | 50 | 28 | „ 71–80 | 1 | 4 |
| „ 41–50 | 31 | 32 | „ 81–90 | 0 | 1 |

I will now consider the morbid changes pertaining relatively to the two classes, necessarily confining the inquiry to the more obvious and tangible, and taking the

[1] The increased mortality in the fourth decade among the alcoholic was chiefly due to delirium tremens, phthisis, diseases of the liver and kidneys, and sanguineous apoplexy. I have given some further details in the *British Medical Journal* for 1873, p. 8.

organs in the order in which they would be reached by a fluid absorbed by the veins of the stomach.

This course is sufficiently obvious. Anything which enters by the veins of the stomach and survives admixture with the blood must needs first pass through the liver. Leaving it by the hepatic vein it enters the right side of the heart and is there mixed with other venous streams. Thence it is sent undiminished, though diluted, to the lungs. In these organs it is evident that in the case of alcohol some escapes with the breath, and probable that some may be transformed by oxidation. As much as emerges by the pulmonary veins is poured into the left side of the heart, in contact with which, and with the walls of the systemic arteries, it is brought before its final distribution. It is now uniformly mixed with the systemic arterial blood, and is with it impartially distributed to every part of the body. If subsequently to this part of its course it affect one organ more than another, it is from the susceptibility of the structure, not from the direction of the poison.

*Stomach.*

The changes in the mucous membrane of the stomach are not patent enough to common observation to take the place in this enumeration to which their frequency would undoubtedly entitle them. It was noticed that in three of the alcoholic series simple ulcer of the stomach existed; in two of the non-alcoholic malignant disease.

*Liver.*

With regard to this organ the annexed abstract will show that there is in the alcoholic class a striking excess of disease, especially of the fatty and fibroid kinds.

Obvious fattiness of the liver, with or without enlargement or congestion, existed in a ratio of exactly three of the alcoholic to two of the non-alcoholic class.

*Abstract showing condition of liver in* 149 *alcoholic traders, as compared with the same number of non-alcoholic traders.*

| | | Alcoholic. | Non-alcoholic. |
|---|---|---|---|
| Natural | | 64 | 79 |
| Hyperæmia | Congested | 9 | 18 |
| | Congested and enlarged | 3 | 2 |
| Fatty change | Fatty | 12 | 11 |
| | Fatty and enlarged | 11 | 3 |
| | Fatty and congested | 4 | 4 |
| | Soft | 3 | 0 |
| Lardaceous | | 4 | 4 |
| Fibroid increase | Capsule thickened | 1 | 3 |
| | Capsule thickened, structure congested | 1 | 1 |
| | Surface puckered, organ enlarged | 0 | 1 |
| | Surface puckered, organ congested | 0 | 1 |
| | [1] Early or slight cirrhosis | 10 | 5 |
| | Advanced or well-marked cirrhosis | 10 | 3 |
| | Cirrhosed, fatty, and enlarged | 1 | 0 |
| | Cirrhosed, also containing hydatids | 1 | 0 |
| | Lobules conspicuous | 1 | 0 |
| | Diminished in size (simply) | 1 | 1 |
| | Simply enlarged | 7 | 5 |
| New formations, &c. | Abscesses, pyæmic or dysenteric | 1 | 5 |
| | Tubercle | 3 | 1 |
| | Cancer | 1 | 2 |
| | Hydatids | 1 | 0 |

*Analysis of hepatic symptoms referred to in preceding cases.*

| | Alcoholic. | Non-alcoholic. |
|---|---|---|
| Decided hepatic symptoms were observed in | 13 | 3 |
| „ jaundice (hepatic) „ | 9 | 3 |
| „ ascites (hepatic) „ | 5 | 1 |
| „ hæmatemesis (hepatic) „ | 1 | 0 |

Fibroid increase, which in an advanced stage produces the characteristic appearance of cirrhosis, preponderates even more heavily. In the alcoholic series were 24 instances in which thickening of the capsule, puckering of the surface, or early or advanced cirrhosis gave evidence of the change. In the contrasted series the total was 14.

Early cirrhosis among the alcoholic existed in 10 cases, advanced cirrhosis in 12. Among the non-alcoholic early cirrhosis existed in 5, advanced cirrhosis but in 3. Thus,

[1] This includes cases where the surface was roughened, and the section showed increase of fibrous tissue or globulation of structure.

on one side we have 22 instances of cirrhosis, against 8 on the other; in other words the liability to cirrhosis is nearly trebled by a liquorous pursuit.

The production of cirrhosis by alcohol, like most beliefs in medicine whether true or false, has been of late disputed. If there be any who still halt between two opinions in this matter, the facts stated can scarcely fail to bring them back to the old view.

With regard to the relative effects of beer and spirits in causing disease of the liver, it appears that of the 22 cases of cirrhosis referred to 15 concerned waiters or potmen; 3 cellarmen, 1 a brewer, who was notorious as a spirit drinker, while the remaining 3 occurred in brewers and draymen. Taking brewers and draymen as the representatives of uncomplicated beer drinking, it seems that considering the smallness of their number they present nearly their fair share of cirrhosis as compared with the other alcoholic traders. It may be observed, however, that cellarmen, who probably may be classed as drinkers of wine and spirit, display a much larger proportion of cirrhosis than the beer drinkers.

With regard to beer drinkers it was noted that in two of the cases the liver was fatty as well as cirrhosed; in one instance the organ weighed no less than 7 lb. 3 oz.

As to simple fatty change with enlargement, which also is a clear result of an alcoholic occupation, of the 11 persons presenting this alteration only one—a brewer—had been especially concerned with beer. The rest were potmen, waiters, cellarmen, barmen, and barmaids.

Thus the facts, though they do not enable us to distinguish one liquor from another in its morbid action, assert beyond question that alcoholic drink causes both cirrhosis and fatty deposition, or, in other words, increases both the fibroid and the fatty elements of the liver.

### *Lungs.*

Next to the liver in the course of imbibed alcohol come the lungs. Here the more important inferences relate to pneumonia, pleurisy, and tubercle.

Pneumonia or hepatization of the lungs, with or without pleurisy, was found in 8 persons of the alcoholic, in 12 of the non-alcoholic series. Pneumonia, as Dr. Stokes pointed out, is no uncommon associate of delirium tremens. The hepatization is usually grey or suppurative and the disease not infrequently masked. This undoubted concurrence would lead us to expect a preponderance of pneumonia under alcohol which does not exist. It will be found, however, that in other organs as well as in the lung there is with the alcoholic trade a lessened tendency to acute inflammation.

With regard to pleurisy the chief difference to be observed is the prevalence of empyema among the traders in liquor; this, as will be afterwards seen, accords with a general suppurative tendency which belongs to the class, made manifest by the products of serous inflammations and the results of injuries.

*Abstract showing condition of lungs in* 149 *alcoholic and* 149 *non-alcoholic traders.*

| | | Alcoholic. | Non-alcoholic. |
|---|---|---|---|
| | Natural | 20 | 34 |
| Congestion, &c. | Congested posteriorly or otherwise | 16 | 16 |
| | Congestion, &c., from typhoid | 2 | 2 |
| | Œdema (frothy or serous infiltration) | 7 | 5 |
| | Emphysema | 3 | 3 |
| | Congestion + œdema | 6 | 5 |
| | Congestion + emphysema | 4 | 2 |
| | Œdema + emphysema | 1 | 2 |
| | Congestion + œdema + emphysema | 2 | 0 |
| Bronchitis, &c. | Bronchitis (uncomplicated by other pulmonary change) | 1 | 0 |
| | Bronchitis + œdema | 0 | 2 |
| | Bronchitis + emphysema | 0 | 2 |
| Pneumonia and Pleurisy | Pneumonia | 1 | 3 |
| | Pneumonia + casts in bronchi | 0 | 1 |
| | Pneumonia + few tubercles, cheesy or cretaceous masses, no tubercles found in other organs | 3 | 3 |
| | Pleuro-pneumonia | 3 | 6 |
| | Pleuro-pneumonia from injury | 1 | 0 |
| | Pleurisy, recent, acute | 4 | 5 |
| | Pleurisy + few tubercles (no tubercles elsewhere) | 2 | 0 |
| | Pleurisy + few tubercles (tubercular disease in other organs) | 0 | 2 |
| | Empyema | 3 | 0 |
| | Hydrothorax | 4 | 5 |

| | | Alcoholic. | Non-alcoholic. |
|---|---|---|---|
| Tubercle, &c.[1] | Miliary tubercles, no vomicæ (no tubercle elsewhere) | 4 | 3 |
| | Miliary tubercles, no vomicæ (tubercle elsewhere) | 3 | 3 |
| | Crude or cheesy tubercle, no vomicæ (no tubercle elsewhere) | 4 | 2 |
| | Crude or cheesy tubercle, no vomicæ (tubercle elsewhere) | 2 | 1 |
| | Tubercles and vomicæ (no tubercles elsewhere) | 11[2] | 12 |
| | Tubercles and vomicæ (tubercular disease elsewhere) | 23 | 13 |
| | Cicatrix at apex (no tubercle elsewhere) | 1 | 0 |
| | Cretaceous mass (no tubercle elsewhere) | 6 | 4 |
| | Cretaceous mass (tubercular disease elsewhere) | 2 | 0 |
| | Pulmonary apoplexy | 2 | 5 |
| | Pulmonary apoplexy + clot in pulmonary artery | 1 | 0 |
| | Pulmonary apoplexy + cretaceous mass + pleurisy (no tubercle elsewhere) | 0 | 1 |
| | Pyæmic abscess or pneumonia | 7 | 6 |
| | Abscess from unexplained cause | 0 | 1 |

The most important question with regard to the lungs relates to the distribution of tubercle, a point to which further interest attaches in consequence of the differences of opinion which have prevailed as to the relation between alcohol and phthisis. Dr. Peter, of New York, whose conclusions though since attacked have been accepted by Dr. Walshe, inferred that drinking protects from tubercle.

Dr. Walshe,[3] indeed, whose opinion will be seen to be directly opposed to the observations here recorded, says 'that publicans, who unquestionably as a class largely consume their own vendibles, are *cæteris paribus* less destroyed by phthisis than persons in various other walks of life.' The antagonism of tubercle and alcohol appears to have

[1] With regard to cirrhosis of the lung this comparatively rare condition is not described in any instance. At the time the earlier notes were written the condition was scarcely recognised.

Two instances only occurred in the tables in which tubercle was found in other organs, none being in the lungs. Both were in the non-alcoholic series; one was of miliary subpleural tubercle, the other tubercle of brain.

[2] One of these was associated with diabetes. Pneumonia, bronchitis, and pleurisy are not mentioned when in connection with extensive tubercular disease of the lung. The same statement applies to emphysema, fibroid, and other change, clearly secondary to the tubercular disease.

[3] *Diseases of the Lungs*, 4th edition, p. 453.

been widely believed, though apparently on insufficient evidence. Trousseau,[1] however, states that his experience has led him to coincide with those who hold the contrary view, and the facts before us instead of supporting strongly contradict the view that alcohol is antagonistic to tubercle. In tuberculosis we see, if the facts adduced be trustworthy, one of the modes in which the body is most vulnerable to drink.

Mindful of differences of opinion as to the nature and varieties of tubercle, I have as far as practicable specified with regard to each case not only the nature of the pulmonary change but also the presence or absence of any conditions recognised as tubercular in any other part of the body. Doubts which may concern a change limited to the lung, as between tubercle, degenerating fibrosis or caseating pneumonia, can find no place where there are characteristic deposits or ulcers elsewhere. Community of tubercle-like changes to several organs may be held as sufficient indication of their truly tubercular character, at the same time that their being limited to one does not necessarily clear them from the imputation.

Taking first the more doubtful class, changes apparently tubercular but limited to the lung, such, comprising miliary, crude and cheesy tubercles, vomicæ, cicatrices and cretaceous masses, were found in an aggregate of 31 persons of the alcoholic, in 25 of the non-alcoholic.

The same changes in the lungs, certified as tubercular by the existence of tubercular disease in other organs, were present in 30 individuals of the alcoholic, in 19 of the non-alcoholic series.

Thus under alcohol reputed tubercle of the lung of every kind preponderates, and the preponderance is greatest where the tubercular nature of the change is the least doubtful. It will be observed that the typical form of tubercular phthisis with tubercle and vomicæ in the lungs and tubercular disease in other organs occurred in 23 of the alcoholic to 13 of the contrasted series.

[1] The results of this paper nearly coincide, in most respects, with Trousseau's views.

Including all kinds, reputed tubercle of the lung affected 61 persons of the alcoholic, 44 of the non-alcoholic class; nearly 3 to 2. This must be conclusive as to the fact that drink instead of preventing promotes phthisis.

It is believed, probably with truth, that alcohol causes pulmonary fibrosis. The cases before us, however, gave no instance to which the terms cirrhosis of the lung or fibroid phthisis could be confidently applied. Within the necessary limits of this inquiry the amount of fibroid associated with the tubercle-like changes in each instance could not be indicated. Care has been taken, however, to separate those cases in which other organs participated in the tubercular change from those in which the lung suffered alone. Any case of simple fibroid or cirrhotic disease which may have occurred has necessarily gone to swell the number of the latter class.

*Tubercle.*

After what has been said with regard to pulmonary tuberculosis, the wider question of tubercle without reference to its seat shrinks into a mere corollary. Only two instances were recorded, one of sub-pleural, one of cerebral tubercle, in which the lungs did not share in the tubercular disease. Both were in the non-alcoholic series, placing the sum of tubercular persons at 61 alcoholic; 46 non-alcoholic.

The question arises whether the prevalence of tubercular disease in the lung is to be interpreted as especially pulmonary, or especially tubercular? Is the tendency to tuberculosis regardless of its seat, or to disease local to the lung out of which the tubercle is engendered? It is clear that the tendency to tubercle is general since it preponderates in other organs even more than in the lung. In each part of the body amenable to tubercle—brain, liver, kidneys, bowels, and peritoneum—the frequency of its occurrence is at least doubled by the alcoholic pursuit. Attributing the excess, as we need must to the influence of the liquor, we arrive at an important and secure deduction that alcohol promotes tubercle.

*Tubercular disease in organs other than the lungs.*

| — | Alcoholic | Non-alcoholic | — | Alcoholic | Non-alcoholic |
|---|---|---|---|---|---|
| Brain . . . | 6 | 3 | Bowels (tubercular ulceration) . . | 12 | 5 |
| Liver . . . | 3 | 1 | Mesenteric glands . | 5 | 2 |
| Kidneys . . . | 8 | 4 | Peritoneum . . | 2 | 0 |
| Spleen . . . | 1 | 0 | | | |
| Total . . . . . | | | | 37 | 15 |

*Heart.*

In the alcoholic series atheroma and fatty degeneration prevail; in the non-alcoholic, endocarditis and pericarditis. Speaking roughly alcoholic pursuits increase the degenerative, and diminish the inflammatory tendency.

Taking atheroma first as often the initial change, it will be seen that labourers in liquor, notwithstanding that they die younger, are the more prone to this deterioration. Atheroma of the aorta in particular was noted in 39 persons of the alcoholic, in 29 of the non-alcoholic series, a proportion of about 4 to 3. Aneurism and sanguineous apoplexy, connected as they are with atheroma, preponderate on the same side.

*Abstract showing condition of heart in* 149 *alcoholic and* 149 *non-alcoholic traders.*

| | | Alcoholic. | Non-alcoholic. |
|---|---|---|---|
| | Healthy . . . . . | 57 | 67 |
| Pericardium, &c.[1] | White patch on surface . . | 8 | 7 |
| | White patch + valvular disease | 1 | 0 |
| | White patch + valvular disease + hypertrophy . . . | 2 | 1 |
| | Pericardial adhesions . . | 2 | 2 |
| | Pericardial adhesions + valvular disease . . . . . | 2 | 3 |
| | Pericardial adhesions + valvular disease + hypertrophy . . | 1 | 3 |
| | Recent pericarditis (lymph and serum) . . . . . | 2 | 2 |
| | Recent pericarditis (pus and serous purulent fluid) . . . | 3 | 0 |
| | Recent + old pericarditis . . | 0 | 2 |
| | Recent pericarditis + hypertrophy . . . . . | 3 | 1 |
| | Recent pericarditis + endocarditis . . . . . | 1 | 1 |
| | Recent pericarditis + old valvular disease . . . . | 0 | 2 |

[1] Fluid in pericardium is not recorded; when the result of inflammation it appears under the heading of pericarditis.

| | | Alcoholic. | Non alcoholic. |
|---|---|---|---|
| Valves, &c. | Valvular disease | 11 | 14 |
| | Valvular disease + hypertrophy | 5 | 9 |
| | Valvular disease + hypertrophy + dilatation | 0 | 3 |
| | Valvular disease + hypertrophy + fatty change | 1 | 2 |
| | Valvular disease + fatty change | 1 | 1 |
| | Valvular disease + dilatation | 1 | 1 |
| Muscular wall, &c. | Simple hypertrophy (*i.e.* unconnected with other cardiac or obvious arterial change) | 12 | 5 |
| | Hypertrophy + white patch on surface | 1 | 1 |
| | Hypertrophy + recent endocarditis | 1 | 0 |
| | Hypertrophy + aortic disease | 4 | 1 |
| | Dilatation | 7 | 5 |
| | Attenuation, flabbiness, or flaccidity | 10 | 9 |
| | Fatty | 6 | 4 |
| | Fatty + enlarged | 2 | 0 |
| | Fat on surface | 2 | 1 |
| Aneurism, &c. | Aneurism of aorta | 2 | 0 |
| | Aneurism of aorta + valvular disease | 1 | 0 |
| | Aneurism of coronary arteries + valvular disease (embolism) | 0 | 1 |
| | Aneurism of ventricle + pericarditis + valvular disease + hypertrophy | 0 | 1 |

*Atheroma in alcoholic and non-alcoholic traders* (149 *of each*).

| | Alcoholic. | Non-alcoholic. |
|---|---|---|
| Atheroma of valves | 3 | 7 |
| ,, coronary arteries | 1 | 2 |
| ,, aorta | 25 | 20 |
| ,, cerebral arteries | 3 | 1 |
| ,, valves + coronary arteries | 0 | 1 |
| ,, valves + aorta | 10 | 6 |
| ,, coronary arteries + aorta | 3 | 1 |
| ,, coronary arteries + cerebral arteries | 1 | 0 |
| ,, aorta + cerebral arteries | 1 | 1 |
| ,, aorta + valves + cerebral arteries | 0 | 1 |
| Total number of individuals in which atheroma was noted | 47 | 40 |

With regard to valvular disease the stress is in the contrary direction. Valvular disease, speaking inclusively without distinction of kind, affected 28 persons concerned with drink, 42 concerned otherwise, being less frequent with drink as 2 to 3. It was not practicable in the tabulations to distinguish the results of valvular atheroma from those of endocar-

ditis; if, however, atheroma on the valves, as elsewhere, is more frequent under liquor, the preponderance of endocarditis in the non-alcoholic series is proportionately increased. In any case it is clear that inflammatory affections of the valves are much less frequent in the drink-trades.

Pericarditis, evinced by recent lymph, purulent fluid, or adhesions, occurred in 14 persons of the alcoholic, 16 of the non-alcoholic class. Thus, in one shape or another the disorder falls pretty evenly, though we find differences when we take into account the diverse circumstances in which the serous inflammation arises. Pericarditis associated with valvular disease, often though not necessarily rheumatic, left its traces in lymph or adhesions in 4 of the alcoholic, 9 of the opposed series, the preponderance corresponding with that of valvular disease. Recent pericarditis, with simple hypertrophy, always renal, was more common with the alcoholic, the numbers being 3 to 1. The suppurative form of the disorder was only found in the alcoholic class; of this there were three instances. This has importance in connection with the prevalence of suppurative pleurisy under the same influence of an alcoholic pursuit.

White patches upon the heart which probably are sometimes though not always due to pericarditis, are slightly more frequent in the alcoholic. This is in accordance with an opinion which has been espoused by Lancereaux to the effect that these marks are increased in frequency by the use of alcohol.

With regard to changes in the muscular tissue of the heart, fatty degeneration or superficial excess of fat occurred in 12 of the alcoholic, in 8 of the non-alcoholic series, the fatty change prevailing in the heart as in the liver in the class where drink was at hand.

Simple hypertrophy or hypertrophy without tangible cause in the vascular system was more frequent with liquor, as 15 to 6; more than twice as common in the alcoholic as in the other class. Such hypertrophy is generally regarded as renal, and it is of interest to inquire whether its marked preponderance is to be traced to a corresponding increase of

renal disease under the influence of drink. I have, therefore, shown in detail the condition of the kidneys in every case of simple cardiac hypertrophy.

*Condition of kidneys accompanying hypertrophy of the heart, either simple or associated with recent pericarditis.*

Alcoholic series—12 cases simple hypertrophy + 3 cases hypertrophy with pericarditis = 15 cases.

Non-alcoholic series—5 cases simple hypertrophy + 1 case hypertrophy with pericarditis = 6 cases.

*Kidneys.*

| | | Alcoholic. | Non-alcoholic. |
|---|---|---|---|
| Intertubal | Advanced granular degeneration | 5 | 3 |
| | Early or slight granular degeneration | 2 | 0 |
| | Cysted, otherwise natural | 1 | 0 |
| Slight or uncertain | Coarse, large and coarse, or coarse and fatty | 3 | 0 |
| Tubal | Natural size, mottled, grey deposit in cones | 0 | 1 |
| | Large, solid, mottled | 0 | 1 |
| | Large and congested | 2 | 0 |
| | Healthy | 2 | 1 |
| | | 15 | 6 |

It would seem that simple hypertrophy of the heart may be associated not only with obviously granular kidneys, but with kidneys otherwise and variously altered, and also with kidneys which passed as natural. The preponderance of hypertrophy of the left ventricle under alcohol was not solely due to a preponderance of renal disease, notwithstanding that granular degeneration with consequent hypertrophy is more common with liquor than without. In more than half the cases of this form of cardiac hypertrophy the kidneys were either natural or were described as congested, fatty or coarse, conditions which could not be confidently regarded as enough to produce cardiac change. We may probably conclude that what is roughly termed simple hypertrophy, though generally dependent on renal disease, may sometimes occur without the intervention of the kidney as part of the cardio-vascular change recently brought before this Society[1] by Sir W. Gull and Dr. Sutton.

[1] Paper by Sir W. Gull and Dr. Sutton in vol. lv.

These minute arterial changes, if we may accept hypertrophy of the left ventricle as their index, prevail among the alcoholic to a larger extent than does obvious kidney disease, and must, therefore, be attributed either to the direct influence of the liquor upon the blood-vessels, or to the lesser degrees of renal change some of which have, found notice.

*Nervous system.*

It is unnecessary to repeat that by the rule of classification delirium tremens is confined to one series, this disease having been taken as sufficient evidence of excess in drink to exclude its subjects from the non-alcoholic category.

Of the alcoholic traders, 17 or about 1 in 9 were affected with this disease, proof enough if it were wanted of the habits of the traders in drink.

In other respects the alcoholic side displayed an excess of inflammatory and hæmorrhagic affections of the brain, and of cerebral uræmia. The preponderance was most striking with regard to inflammatory conditions, and was marked whether associated with or independent of tubercle.

Six cases of tubercular disease of the brain occurred in the alcoholic, 3 in the non-alcoholic series.

Acute inflammation of the brain, not connected with tubercle, described as cerebro-spinal meningitis, encephalitis, and inflammatory softening, occurred in four instances, all of which belonged to the alcoholic series. There is evidence to show that drunkenness is alone sufficient to set up meningitis in an acute form. One of the cases in the table was a striking example of this, and another came under my own observation. In neither was there any tubercular or other disease with which the attack could be associated.

There is also in the same category a strong disposition to the collection of aqueous fluid in the intracranial spaces; this, not usually manifest during life, is probably often simply a compensation for chronic shrinking of the cerebral matter.

An alcoholic trade increases the liability to cerebral

hæmorrhage. Putting aside a case of cerebral purpura the tables include 10 cases of cerebral hæmorrhage of the ordinary kind. In every case the kidneys were granular, and, no doubt, with the same regularity the vessels of the brain were diseased. Six of these cases occurred in the alcoholic, 4 in the non-alcoholic series. Thus, cerebral extravasation, like hypertrophy of the heart, both especially associated with renal disease and both taking their origin in vascular change, are fostered by alcohol.

Disturbances of the brain dependent on uræmia are also more frequent under alcohol; but this fact will be more appropriately considered in connection with renal disease.

*Abstract showing state of brain and nervous system in* 149 *alcoholic and* 149 *non-alcoholic traders.*

| | | Alcoholic. | Non-Alcoholic |
|---|---|---|---|
| Hæmorrhage. | Sanguineous apoplexy | 6 | 3 |
| | Purpuric hæmorrhage under arachnoid | 1 | 0 |
| | Slight hæmorrhage under arachnoid, with congestion of brain, with granular kidney | 0 | 1 |
| Delirium tremens. | Delirium tremens | 15 | 0 |
| | Delirium tremens + calcareous deposit (in falx) | 1 | 0 |
| | Delirium tremens + uræmia (epileptiform convulsions) | 1 | |
| Uræmia. | Uræmia (marked during life by convulsive attack, or partial or complete coma) | 5 | 3 |
| | Excess of fluid with anæmia, associated with renal disease, but without marked uræmic symptoms | 2 | 3 |
| Excess of watery fluid, &c. | Excess of fluid in and about brain, without cerebral symptoms, after death by cholera, fever, carbuncle, erysipelas, and suicide | 5 | 1 |
| | Excess of fluid after death from acute rheumatism + bony plate in arachnoid | 0 | 1 |
| | Congestion, or blood-tinged fluid under arachnoid, with erysipelas | 1 | 1 |
| | Excess of fluid in and about brain, productive of symptoms and due to disease primarily of brain | 2 | 3 |

| | | Alcoholic. | Non-Alcoholic. |
|---|---|---|---|
| Tubercle. | Tubercular meningitis | 4 | 2 |
| | Tubercular disease of brain | 2 | 1 |
| Inflammation. | Inflammation of brain or membranes from blow or injury | 3 | 3 |
| | Inflammation of brain or membranes from disease of skull | 1 | 1 |
| | Cerebro-spinal meningitis | 1 | 0 |
| | Acute encephalitis (from hard drinking); softening, with, in one case, pus in ventricles, in the other lymph under arachnoid | 2 | 0 |
| | Softening + excess of fluid + congestion (cerebral symptoms) | 1 | 0 |
| | Softening of brain, circumscribed (pyæmic ?) | 0 | 1 |
| | Softening of brain with symptoms of cerebral disease | 1 | 0 |
| | Old adhesions in arachnoid + insanity | 1 | 0 |
| | Coagula (ante mortem) in arteries | 0 | 1 |
| | Calcareous or osseous matter in membrane or choroid | 0 | 1 |
| | Tumour of brain | 1 | 1 |
| | Enlarged Pacchionian bodies | 1 | 0 |
| | Epilepsy | 1 | 0 |
| | Epilepsy + tetanus | 1 | 0 |
| | Paraplegia from disease of spine | 1 | 1 |
| | Paraplegia from disease primarily of cord | 0 | 2 |

*Condition of kidneys associated with extravasation of blood within the cranium.*

*Alcoholic series.*

Common sanguineous apoplexy, 6 cases:

| | |
|---|---|
| Kidneys, advanced granular in | 4 |
| „ slightly „ | 2 |
| | 6 |

Purpuric hæmorrhage, 1 case:

Kidneys natural.

*Non-alcoholic series.*

Common sanguineous apoplexy, 4 cases:

| | |
|---|---|
| Kidneys, advanced granular in | 3 |
| „ slightly „ | 1 |
| | 4 |

*Results of accidents and injuries.*

It is well known that with drunkards the power of healing is weakened, that processes of repair are slowly and unsafely conducted, and that small injuries and trivial operations are

attended with disproportionate danger. The facts before us bear upon this point.

Traders in liquor are less exposed in their way of life to external hurts than those to which they are compared. Draymen meet with horse accidents, and potmen are sometimes involved in disputations which end in a violent misuse of the pewter pot, but the class to which they belong is less often subjected to accidental injury though, possibly, suffering more fatally from its effects, than the other. This is shown by the distribution of necessarily and immediately fatal accidents. Such accidents as are inevitably fatal, and are therefore unaffected in their issue by the antecedents of the victims, will by the number on either side indicate the liability of the class to violent bodily damage. We find five necessarily fatal accidents in the alcoholic, ten in the non-alcoholic series. Thus, it would seem that traders in drink are only half as often the subjects of such accidents as those who follow other and in this respect more dangerous callings. If the same rule applies to small accidents as to great, traders in liquor have a comparatively small exposure to mechanical violence. But accidents among them, though less frequent, are more fatal.

*List of fatal accidents.*

| Alcoholic. | | Non-Alcoholic. |
|---|---|---|
| 5 | *Directly fatal.* | 10 |
| Fracture of ribs, laceration of lung. | | Fracture of skull. |
| Fracture of pelvis. | | Fracture of thigh, &c. |
| Cut throat (suicidal). | | Rupture of bowel. |
| Fracture of skull. | | Fracture of skull. |
| Laceration of liver. | | Laceration of liver. |
| | | Gunshot (suicidal). |
| | | Fracture of skull. |
| | | Laceration of liver. |
| | | Fracture of leg, &c. |
| | | Wound of brachial artery. |
| 10 | *Succeeded by delirium tremens.* | 0 |
| Bruise of chest. | | |
| Burn, ulcer of duodenum. | | |
| Compound fracture of leg. | | |
| Slight injury of nose. | | |
| Cut wrist. | | |
| Dislocation of ankle. | | |
| Compound fracture of leg. | | |
| Slight bruise of head. | | |

| Alcoholic. | | Non-Alcoholic. |
|---|---|---|
| 4 | *Succeeded by pyæmia.* | 4 |
| Compound fracture of leg; amputation. | | Compound fracture of leg. |
| Scalp wound, pus outside dura mater, &c. | | Abscess of thumb, followed by cellulitis. |
| Compound fracture of leg. | | Crushed hand, amputation. |
| Compound fracture of thigh. | | Compound fracture of skull. |
| 5 | *Succeeded by diffuse cellulitis.* | 3 |
| Scalp wound. | | Scalp wound, fracture of spine. |
| Slight injury of thigh (delirium tremens occurred also). | | Fracture of skull, laceration of brain. |
| Fracture of thigh, ununited. | | Severely cut throat. |
| Bite of donkey. | | |
| Pugilistic bruises. | | |
| 1 | *Succeeded by erysipelas.* | 1 |
| Injury of leg, suppuration of knee, &c. | | Small scalp wound. |
| 1 | *Succeeded by gangrene.* | 0 |
| Fracture of fingers. | | |
| 2 | *Succeeded by empyema and pleuro-pneumonia.* | 0 |
| Simple fracture of ribs. | | |
| Fracture of ribs, &c. | | |
| 1 | *Succeeded by tetanus.* | 0 |
| Burn. | | |
| 2 | *Succeeded by superficial softening of brain.* | 0 |
| Blow on head. | | |
| 1 | *Succeeded by cystitis.* | |
| Fracture of leg. | | |

Pyæmia seizes more than its share.

Diffuse inflammation of the cellular tissue is fatal and readily induced.

*Complications of accidents ultimately, but not immediately or necessarily fatal.*

| | Alcoholic | Non-Alcoholic. |
|---|---|---|
| Pyæmia | 4 | 4 |
| Cellulitis | 5 | 3 |
| Erysipelas | 1 | 1 |
| Gangrene | 1 | 0 |
| Pleuro-pneumonia from broken ribs | 1 | 0 |
| Empyema from broken ribs | 1 | 0 |
| Pus between skull and dura mater (from scalp wound) | 1 | 0 |
| Tetanus | 1 | 0 |
| Superficial softening of brain (from blow on head) | 1 | 0 |
| Accidents directly and necessarily fatal | 5 | 10 |

The non-alcoholic series yielded three fatal cases of cellulitis which were respectively produced by fractures of the skull and spine and a deeply cut throat. The alcoholic

series gave five, one of which only had its origin in a severe injury, namely, a fracture of the thigh which had refused to unite; the others were consequent upon a scalp wound, a slight injury of the thigh, pugilistic bruises, and the bite of a donkey.

An increased liability to spreading inflammation of the cellular tissue belongs, therefore, to the trader in drink, produced, no doubt, by the liquor. In other words, under the chronic influence of alcohol, the product of inflammation is apt to be purulent and non-adhesive, such as can spread and disseminate, rather than of the plastic and circumscribing sort, which limits the process by means of fibrillating lymph, and eventuates in false membrane and solid repair. The formation of pus instead of plastic lymph under the same influence is seen in the generation of empyema and of abscess within the skull from external injuries, nothing of the kind having occurred in the non-alcoholic category. These observations have additional weight when taken with reference to what has already been shown with regard to the distribution of the suppurative forms of pleurisy and pericarditis.

These conclusions are consistent with some observations lately published by Dr. Péronne,[1] who reckons among the effects of chronic alcoholism in relation to the healing of wounds, abundant suppuration, diffuse inflammation, partial gangrene, secondary hæmorrhage, and tardiness of healing.

### *Kidneys.*

The effect of alcohol upon the common channels of exit may naturally be considered last. The kidney, which has been credited with a morbid susceptibility to alcohol much in excess of what is warranted by facts, is the only gland of excretion which need occupy us in this relation.

By reference to the annexed abstract it will be seen that the kidneys were described as free from disease in almost exactly the same number of the two classes. The same statement holds good with regard to kidneys which presented

[1] *De l'Alcoolisme dans ses Rapports avec le Traumatisme*, p. 154.

no further departure from their natural state than simple congestion, probably in many instances a fleeting and almost accidental condition. Thus it remains that the number of kidneys presenting tangible morbid changes was almost exactly the same on the two sides; or to speak precisely, the kidneys of 82 persons in the alcoholic, of 83 in the non-alcoholic series, presented distinct morbid changes. This statement includes, of course, not only the alterations recognised by albuminuria, but comprehends also suppurative, tubercular, cancerous, embolic, and calculous affections.

*Condition of kidneys in* 149 *cases of each kind.*

| | | Alcoholic. | Non-Alcoholic. |
|---|---|---|---|
| | Natural | 49 | 48 |
| Hyperæmia. | Congested | 18 | 18 |
| | Congested and enlarged | 10 | 5 |
| Tubal changes. | Coarse | 3 | 3 |
| | Coarse and enlarged | 4 | 1 |
| | Slight or uncertain change in cortex | 2 | 4 |
| | Large, smooth, mottled[1] | 5 | 9 |
| | Mottled, normal size, grey deposit in cones | 0 | 1 |
| | Smooth, pale, yellow or grey, cortex shrunk | 1 | 1 |
| Fatty change. | Fatty or flabby | 2 | 1 |
| | Fatty or flabby and enlarged | 3 | 3 |
| | Lardaceous | 3 | 6 |
| Intertubal, fibroid increase. | Cysts without other change | 1 | 2 |
| | Cysts and depressions | 3 | 1 |
| | Cysts and tubercles | 0 | 1 |
| | Slightly granular | 10 | 11 |
| | Highly granular | 18 | 15 |
| | Granular + pyelitis | 1 | 0 |
| | Granular + tubercles | 1 | 0 |
| | Granular + conversion of other kidney into a cretaceous mass | 0 | 1 |
| | Granular + stone | 1 | 0 |
| | Pyelitis | 0 | 2 |
| | Abscesses in kidney associated with pyelitis | 1 | 6 |
| | Abscesses from pyæmia | 1 | 1 |
| | Abscess of uncertain nature | 1 | 0 |
| | Tubercular disease | 8 | 4 |
| | One converted into cretaceous mass, other natural | 0 | 1 |
| | Cancer | 1 | 1 |
| | Fibrinous blocks, without other change | 0 | 2 |
| | Stone | 2 | 1 |

[1] It is possible that some of the kidneys thus described in the earlier records were lardaceous, in which case the preponderance of lardaceous disease in the non-alcoholic series would probably be greater than is represented.

*Symptoms referable to albuminuria in the preceding cases.*[1]

| | Alcoholic. | Non-Alcoholic. |
|---|---|---|
| Number of cases in which renal symptoms of any kind were prominent . . . . | 20 | 26 |
| Renal œdema occurred in . . . . . | 14 | 18 |
| Internal dropsy, apparently renal, occurred in | 4 | 6 |
| Albuminuria found in . . . . . | 23 | 29 |
| Cerebral uræmia (coma, convulsions, &c.) . | 5 | 3 |
| Death from renal disease (directly)[2] . . | 10 | 14 |

Descending to details, and taking first changes which, so far as the descriptions allow us to judge, are in the tubes and their contents, we find on adding the kidneys described as enlarged and congested, and those which were fatty, to the varieties included within the tubal bracket, that in one or other of these ways the kidneys were diseased in 30 persons of the alcoholic series to 28 of the opposed class. Among these changes those only which had marked predominance in the alcoholic category were enlargement with either coarseness or congestion; of these were 14 in the alcoholic, to 6 in the contrasted series. The typical large white kidney was most common in the non-alcoholic class. Thus, it would seem that though certain alterations, which apparently have their seat within the tubes, prevail under an alcoholic trade, yet that they are not such as have marked symptoms or are generally discoverable during life. Under this influence tubal changes, though slightly more frequent, are less acute and obvious. It must be taken into account, however, as affording a partial explanation of their lessened liability to acute inflammatory affections of the kidney, that traders in liquor are less exposed to wet and cold, not infrequent causes of acute nephritis, than are the persons with whom they are compared. (See page 68.)

With regard to the intertubal or fibroid change we find distinct granulation in 31 alcoholic to 27 of the non-alcoholic series. If we look upon cysts (as with general truth we may) as evidence also of intertubal change, we

[1] The table comprises all the renal symptoms observed in the cases of the two series, excepting those referred to pyelitis or renal abscess, tubercle, or other morbid growths, or calculus. These are excluded for the sake of placing the results of albuminuria in a distinct form.

[2] Sanguineous apoplexy is not included here.

must place the total among the alcoholic at 35, among the non-alcoholic at 31. Thus, it would seem that the frequency of granular degeneration is slightly increased by a conversance with liquor, the inequality in the distribution of the disease between the two classes being nearly represented by the proportion of 8 to 7.

Lardaceous infiltration, like other diseases of the kidney, has been loosely ascribed to intemperance in liquor. This change, however, is decidedly the less frequent in the alcoholic series. The diminution may be due, wholly or in part, to the comparative rarity of surgical accidents in the class, with a correspondingly smaller share of such hurts of bone and joint as give rise to long suppuration. We may at least conclude that the excessive imbibition which accompanies habitual intimacy with liquor, though it may not prevent, does nothing towards engendering the change in question.

This evidence, relating solely to inspection of the kidneys, may be supplemented by collateral reference to other organs affected in dependence upon or association with them, as well as by a glance at the accompanying symptoms. Pathological facts as gathered after death and clinical observations made during life agree with each other and with the preceding statements in indicating under alcohol an increase of granular degeneration, a diminution of active tubal nephritis and of lardaceous disease.

As to the changes in the vascular system we find under alcohol a marked excess of common atheroma, of simple hypertrophy of the heart and of sanguineous apoplexy. These indications of vascular deterioration are so often associated with the granular kidney that they may be generally taken as signs of its prevalence. Simple hypertrophy, however, and cerebral extravasation predominate in the alcoholic series to an extent which would lead us to expect a larger excess of renal disease than is found. Possibly the lesser degrees of renal change may sometimes escape notice. But alcohol would seem, independently of renal disease, to have a damaging effect upon the arteries, whence the heart may become hypertrophied though the kidneys are natural. Thus, the deterioration of the coats of

the vessels which accompanies renal disease, more especially of the granular kind, is enhanced by familiarity with liquor.

Cerebral uræmia, which is a more constant associate of the granular kidney than of the other forms of renal disease, also predominates in the alcoholic series.

Symptoms which point to other renal changes tell with equal consistency a different tale. In the alcoholic series we note fewer cases in which renal disease was detected during life; there is here less discovered albuminuria, less dropsical effusion of every kind, and a smaller number of deaths attributable directly to renal mischief. These details concur with the facts derived from examination of the kidney itself in showing that traders in liquor, either from the operation of the drink or the circumstances of the trade, are less liable than their neighbours to lardaceous disease and to tubal inflammation of the active and prominent kind.

Diverging for a moment from the strict subjects of our inquiry, and looking at the state of the kidneys generally accompanying delirium tremens, we may find additional evidence on this point.

I subjoin a table[1] compiled from the same sources in which the post-mortem state of the kidney in subjects of *delirium tremens* is compared with that of men not known to have been intemperate who have been cut off by accidental violence.

*Kidneys after death with delirium tremens, and from accident without known intemperance. All of adult males.* (*From the year* 1841 *to* 1871 *inclusive.*)

| Conditions of kidneys. | Delirium tremens. 58 cases. | Accident. 58 cases. |
|---|---|---|
| Natural | 28 | 24 |
| Congested | 15 | 7 |
| Slight or uncertain change in cortex | 5 | 1 |
| Large, smooth, mottled | 3 | 1 |
| Granular surfaces | 6 | 8 |
| Cysts without other change | 1 | 7 |
| Average age of delirium tremens patients, 38 years. | | |
| „ accident | „ 41 „ | |

[1] This table is similar to one published in my work on 'Albuminuria,' but comprises the records of seven additional years.

Assuming, as we safely may, that most of the victims of delirium tremens have been habituated to alcoholic excess, we find in this fraternity of drunkards what to many persons may be a surprisingly small increase of renal disease. It must be observed, however, that the cases of accidental death are taken without exclusion as to trade, so that a certain proportion of lead workers, of whose calling granular degeneration is a necessary result, have been admitted.

Taking all the facts together, and allowing to the trader in liquor his full measure of protection from weather and violence, we must conclude—

1. That alcohol has no effect in causing lardaceous disease.

2. That it promotes granular degeneration, though not to an extent commensurate with the arterial change produced by the same agent.

3. That acute nephritis, though in exceptional instances, caused by intense alcoholic excess, is not generally increased in frequency by habits of drinking. In its comparative rarity under this influence the affection resembles others, save of the brain, of the acute inflammatory type.

4. Chronic and latent tubal disturbances, including fatty degeneration, are increased by the use of alcoholic liquors.

5. And that on the whole the kidneys are affected less injuriously by the popular poison than are the liver, the lungs, the blood-vessels, and the nervous system.

### *General conclusions.*

We are led to conclusions which are mainly with, though in some respects against, old views. In the present state of medicine it may be as useful to fortify old truths as to assault old errors. The results may be thus summed up.

Persons who trade in liquor drink on an average more than those who do not, and their morbid peculiarities are mainly due to that excess. Estimating the effects of alcohol on this basis by means of comparison between the class described and persons similarly situated save in relation to liquor, the following conclusions have been reached.

Alcohol shortens life; to trade in liquor costs three and a half years.

Reviewing the morbid results which the examination of each organ has revealed, they present a consistency which is in some sort their warrant.

Alcohol causes fatty infiltration and fibroid encroachment; it promotes tubercle, encourages suppuration, and retards healing; it produces untimely atheroma, invites hæmorrhage, and anticipates age.

The most constant fatty change, a replacement by oil of the material of epithelial cells and muscular fibres, though probably nearly universal, is most noticeable in the liver, the heart, and the kidneys.

The fibroid increase is little evident in the simply fibrous structures, such as fasciæ and tendons, but occurs about the vascular channels and superficial investments of the viscera, where it causes organic atrophy, cirrhosis, and granulation. Of this the liver has the largest share; the lungs are often similarly though less simply affected, the change being variously complicated with or simulative of tubercle; the kidneys suffer in like manner but in a more remote degree.

Alcohol also causes vascular deteriorations which are akin both to the fatty and the fibroid. Besides tangible atheroma there are alterations of the arterial walls, which show themselves by cardiac hypertrophy and cerebral hæmorrhage.

Drink causes or invites tuberculosis, which is evident not only in the lung, but in every organ which is amenable. It must be observed, however, that from the tendency of tubercle to dissemination it is only necessary that it be planted in one organ to be found in many.

Drink promotes the suppurative at the expense of the adhesive process. This is sufficiently seen in the results of pneumonia, of pleurisy, and of pericarditis, in diffuse inflammation after injuries, and in the sloth and insecurity of the healing process.

Descending from general conditions to individual organs, the effect of alcohol upon the nervous system must be looked

upon as special and taken by itself, since nervous matter presents to this agent a singular excitability of function, or in other words, a singular susceptibility of structure, for the purpose of acting upon which mankind in all countries and in all ages have sought and used alcoholic drinks.

Passing over those effects of intemperance which like delirium tremens are manifest rather during life than after death, we find that the brain pays a large reckoning in the shape of inflammation, atrophy, and hæmorrhage.

Alcohol multiplies inflammatory states of the brain of every kind, both of the substance and the envelopes, whether tubercular or not.

It occasions a gradual shrinking of the brain, as evinced by the accumulation of fluid in spaces once filled by cerebral substance.

It causes a liability to cerebral hæmorrhage by means of the arterial deterioration common to the greater part of the body.

With regard to other organs they are injured by alcohol much as they stand in its line of absorption. Passing over the stomach, the changes in which, however numerous, are, save when it is ulcerated, more prominent during life than after death, we come to the liver. This organ suffers more than any other, chiefly by way of cirrhosis and fatty impregnation. Next the stress falls upon the lung, probably less heavily than upon the liver, certainly more heavily than upon any other part of the body with the exclusion of the nervous system. The mischief in the lung takes apparently every shape of phthisis. It is probable that the change most often is of a kind to which the term tubercular would strictly apply, while there is evidence, not comprised in the present tables, of a fibroid overgrowth, which apparently may either accompany or simulate tubercle. The arterial deterioration need not be further mentioned than to assign its relative frequency. Judging by its tangible results—atheroma, hæmorrhage, and cardiac hypertrophy—we should assign to this change a large share in the pathology of intemperance, though unless the connection between alcohol and tubercle

has been much overrated, it would seem to play a less fatal part than disease of the tubercular kind. Lastly, the kidneys, more remotely exposed, have a smaller participation in the common hurt of alcoholism. They undergo congestive enlargement, fatty and fibroid change; they do not suffer, however, commensurately with the blood-vessels or with the same frequency as other viscera.

So far we have seen only the ill which alcohol produces; it may be asked is there none which it obviates? Apart from any medicinal or curative action, which the evidence before us does not touch, has it no *per contra* of prevention? It is difficult to answer this inquiry. Some active inflammations, such as pneumonia and endocarditis, are diminished in the alcoholic trades, but it must at once be seen that the increase of the alcoholic disorders must necessarily produce an apparent diminution of all which are unaffected by this agent. A man may be saved from pneumonia or acute rheumatism, not because alcohol antagonises such diseases, but because it kills him prematurely. He can die but once. Therefore, though under alcohol some forms of disease are comparatively infrequent, we must use much caution in concluding that it has a directly preventive influence. Nevertheless, it may be laid down as an axiom that any drug which can do harm can do good. Disease is most various, and may, or rather *must*, represent contrary conditions. It may be positive or negative, plus or minus. Too much or too little of any of the shapes of heat, food, and work may spoil the equipoise of health. If a drug promotes one change it may prevent its opposite. Alcohol certainly gives an asthenic type to disease; although we cannot as yet say with certainty that it defibrinates the blood, yet it retards adhesive and plastic processes. This influence may be beneficent if it hinder the development of acute inflammation, and obviate the formation of coagula, where, as in acute rheumatism, that action is harmful. It is possible that by some such antagonism to the sthenic and fibrinous type of disease we may explain the remarkable paucity of endocarditis in the alcoholic series. But, at the best, the

protecting is less certain and less effective than the deteriorating influence.

In brief and final enumeration, alcohol replaces more actively vital materials by oil and fibrous tissue; it substitutes suppuration for new growth; it promotes caseous and earthy change; and helps time to produce many of the effects of age.

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## *ON THE PATHOLOGY OF CHOREA.*[1]

In the present nascent state of the pathology of the nervous system what is most wanted is the accumulation of observations; general laws may hereafter become manifest and lines of classification show themselves; but as yet it is apparent that, putting aside the coarser injuries, our knowledge on this question is so far incomplete that it is hardly possible to trace, without intervals of conjecture, the progress of any disease belonging to this portion of the frame, from its origin in external or inherent influences to its close in fatal injury to necessary structures.

As a small contribution to a large want I propose to take the subject of Chorea, one which opportunity has brought in my way, and to describe the state of the nervous centres in a short series of cases, with such brief reference to other circumstances, as may help to throw light upon the pathology of the disorder.

The instances are related as nearly as could be judged in the order of their acuteness, beginning with those in which death ensued at the earliest stages of the disease.

Case I.—On the 12th of October, 1875, Margaret C——, aged 10, was admitted as my patient into St. George's Hospital, though my absence from town prevented my noting her later symptoms from personal knowledge. She had been attacked with acute rheumatism a month before, of which only the cardiac results were now apparent. The apex was lower than natural, and marked the position of a loud systolic murmur. She had no

[1] 1875. *Medico-Chirurgical Transactions*, vol. lix.

palpitation or dyspnœa, though there was some pain in the left side of the chest on deep inspiration, and pain also in the spinal region, which, however, as yet, did not receive much notice. Under small doses of iron and digitalis she seemed to improve, and on the 19th was able to leave her bed. When up, slight choreic movements were for the first time observed. These, slight as they were, terrified the child. A little friend of hers had lately died of chorea; she had watched and imitated the movements, and when she recognised them in her own person, made sure that she should meet the same fate. She was perturbed in mind, restless, and sleepless. On the 21st the movements much increased, and at the same time a little blood was found in the urine. In the evening she was seized with acute pain, which she referred to the lumbar region of the spine, and thought to be relieved by rubbing.

When this had lasted about an hour she somewhat unexpectedly died.

On post-mortem examination, about half a pint of clear fluid was found in each pleura, and two ounces in the pericardium. The heart was increased to the weight of eight ounces; it was partially contracted; the free edge of the mitral valve was thickened and on its auricular surface was a fringe of small fibrinous beads, easily detached; similar vegetations were found upon the edges of the aortic valves. The liver, spleen, and kidneys were tough and congested. No other morbid appearances were discovered save in the nervous centres. The brain was congested in membranes and substance. The puncta vasculosa were marked, and from many of the vessels of the oval centres and the medulla threads of black coagulum could be drawn. The spinal cord, in the superficial examination which was made in the fresh state, was not noted as unnatural.

Several parts of the brain, which it is not necessary to describe in detail, were examined in section with results such as in some of the cases related subsequently, though of earlier occurrence, are described more minutely.

The vessels were loaded with blood, those of the size above the smallest the most remarkably so; arteries and veins were both affected; when one only, then generally the vein. The corpora striata and the arbor vitæ were thus markedly affected; in the latter were one or two considerable holes which represented enlarged peri-vascular canals.

In the cord were more unusual deviations. The central

canal was dilated in a manner which I have not seen equalled.[1] The enlargement was great in the cervical region, where the transverse diameter of the canal varied from $\frac{1}{20}$ of an inch to half as much; less in the dorsal, where it diminished to about $\frac{1}{60}$; greatest in the lumbar, where it reached a seventh of an inch, occupying exactly one-third of the entire diameter of the cord. In the cervical and dorsal regions the cavity was mostly filled with granular blood-coloured material which evidently largely consisted of altered blood; in the lumbar region the canal was empty. In the dorsal region, where the contents of the canal were most abundantly sanguineous, the larger vessels were injected to the uttermost, distended and loaded veins lying in the grey matter on both sides of the canal and traversing the white columns.

In the upper part of the canal, particularly in the dorsal region, where the congestion was extreme, blood had been poured into it, and was of course found *in situ*; elsewhere the distending fluid had apparently been serum which had escaped when the cord was cut.

Beyond these changes, which may be briefly summed up as hyperæmia of the cord with distension of the canal by blood and serum, there were few others which call for notice. The anterior fissure, particularly in the cervical region, was eroded and contained blood-tinged effusion.

Case II.—Mary C—— died at the age of ten years under my care at the Hospital for Sick Children in her third attack of chorea. The first, four years previously, was attributed to her having fallen into a pond; she was treated for it in the hospital and recovered under sulphate of zinc. The symptoms were then nearly as severe as in the last and fatal seizure. She was ill five months, but recovered perfectly. The second attack, two years afterwards, was slight and transient. After it she remained well for eighteen months, until on the 21st of May, 1874, she was butted by a goat which a boy had set at her, pushed down, and much frightened, though but little hurt. On the 25th her legs were observed to twitch, and later the arms. The movements daily increased, speaking and swallowing became difficult, she became sleepless and feverish, complained of pain in the lower and middle parts

[1] I have observed a similar but less extreme dilatation of this canal in diabetes. See *Renal and Urinary Affections*, vol. i. p. 44.

of the back and in the thighs, and on the 10th of June was admitted for the last time. She had never had rheumatism.

When admitted the symptoms were severe; the movements of the limbs were so violent and continual that splints were required for their restraint—a measure nowise objected to by the child. There was much difficulty in articulation, but none in the choice of words. Her words were correctly selected, though uttered interruptedly, with much effort and grimace, and often only in a whisper. There was some difficulty also in swallowing, due apparently to the disturbed action of the mouth and tongue. She had sordes on the lips, a dry tongue, and much febrile disturbance, with a temperature gradually increasing up to 104·5. A faint systolic murmur was audible at the apex. She was sleepless and the bowels confined; conditions to the obviation of which the early treatment was mainly directed.

On the night of the 11th, having been but three days in the hospital, while in the act of taking some brandy-and-water she suddenly collapsed, became blue, and died. In the early part of the night she had slept from chloral.

On post-mortem examination it was found that, putting aside the nervous centres, all the organs were healthy except the heart. The pericardium was natural, the mitral valve only affected. Along its auricular surface, just above the free edge, was a straight regular line of small close hard vegetations, all firmly fixed, and without adherent fibrine. The condition of the heart was characteristic of the disease, just this amount of vegetation, without irregular fibrine, and thus limited to the inner surface of the mitral valve, being found in a considerable proportion of instances of death by chorea.

There was a slight excess of arachnoid fluid, but none in the ventricles of the brain. The grey matter was dark, the red points of the white numerous. The microscope showed a generally scattered loading of the vessels up to about $\frac{1}{50}$ of an inch in diameter, both arteries and veins. Distension with blood persistently after death is obviously more distinctly unnatural in arteries than in veins; in this instance both were conspicuously affected. The large superficial veins of the corpora striata were distended to the utmost; and some of the larger arteries passing through its substance were surrounded by translucent, structureless, or delicately granular material, apparently an exudation of liquor sanguinis, lying between

brain and vessel. In the pia mater and subarachnoid space were many extravasations of blood *in toto*; such were well seen in the anterior fissure of the medulla, where large distended arteries were involved in a loose mass of corpuscles, within the meshes of the subarachnoid, which had apparently escaped, not by rupture, but migration. The brain substance was nowhere broken by the extrusion.

In the cord many still loaded vessels entered the grey matter; there were no evidences of hæmorrhage, though many of perivascular change probably due to escape of at least the liquid part of the blood. Perivascular changes were found in the lumbar region, where dilated arteries traversed the grey horns separated from their substance by a wide interval filled with the globular translucent matter which so often marks the contact of nervous tissue with dilated vessels. Transudation from the vessel and erosion thereby of the channel in which it lies would seem to constitute the major parts of the process, of which the results have been described.

Case III.—A girl, 7 years of age, died at the Hospital for Sick Children in a first attack of chorea, under the care of Dr. West, to whom I am indebted for the opportunity of examining the nervous centres.

There was no history of rheumatism or fright. The only ostensible cause for the illness was that she was wet through the day before the first symptoms appeared. The attack was severe though uncomplicated. Death occurred on the 24th day with signs of prostration which had come on rather abruptly. She had had but little sleep, and the movements had been violent and general. She had been treated chiefly with antimony.

On post-mortem examination all the organs were found to be healthy except the heart and the nervous centres. The heart had a line of small recent vegetations along the auricular edge of the mitral valve.

The sinuses of the dura mater were full of blood, some of which had coagulated. The surface and substance of the brain were also injected. Portions of this centre, with the spinal cord, were remitted to me for further examination.

The peculiarities of the brain may be summed up in the word injection—arterial, venous, and capillary. The smaller arteries and veins, whether in the superficial pia mater, in the fissures,

or in the substance of the brain, had remained after death full of blood, which, solidified by the chromic acid, assumed the appearance of a general thrombosis. This loading of the blood-vessels affected all parts of the brain and all sizes of vessels; it was exceedingly marked in both arteries, and veins of about $\frac{1}{150}$ of an inch in diameter, and extended into the lesser ramifications until it reached the capillaries, many of which, even of the smallest size, were made conspicuous by the crowding of blood-corpuscles within them. The capillary injection was most marked in, though by no means confined to, the optic thalami. There were few evidences either of extravasation or of perivascular change; in one or two instances blood had escaped from the distended vessel into the surrounding sheath. The cerebellum, and conspicuously the neighbourhood of the dentate body, shared in the injection, particularly of the larger vessels.

The cord was congested, but to a less degree; a few large loaded vessels passed through the white into the central grey matter. In the dorsal and lumbar regions the grey matter showed the effects of hæmorrhage within its tissue. In certain tracts both of the dorsal and lumbar regions the grey matter had been broken or locally destroyed, with lateral symmetry, at the centre of each horn. The disruption at its greatest extent was nearly enough to cut the crescent through its middle and sever the anterior from the posterior cornu. The affected spot consisted of a somewhat irregular rending or crumbling of the grey matter, the broken-up nervous substance being more or less mingled with faded and altered blood, and the cavity fringed with the globular product of nervous disintegration. The result could only be ascribed to hæmorrhage some little time before death, simultaneously at several points in the grey matter, similarly disposed with regard to the two sides.

The morbid appearances in this case may be briefly described as those of injection of the brain and extravasation into the cord.

For the clinical facts of the following case and permission to examine the nervous centres I am indebted to my colleague Dr. Gee.

Case IV.—Clara W——, aged 8, who had had scarlet fever five years before, but never rheumatism, was, while apparently well, frightened by a boy who had hidden himself in a dark room, and agitated much out of proportion to the cause. Three days later

choreic movements showed themselves, and gradually increased until she was brought to the Hospital for Sick Children, fourteen days after their commencement. She then had violent choreic movements of the neck, trunk, arms, and legs. She could not stand or even sit without danger of falling. The face was little affected, but swallowing was difficult and speech hesitating. The embarrassment in the latter respect shortly increased, so that she became scarcely able to utter her name intelligibly. There was no cardiac murmur, though the sounds were not perfectly regular.

She was placed on a water bed and treated mainly with chloral. The choreic symptoms much diminished, but she became prostrate, lapsed into the condition to which the term typhoid is applied, displayed some ulcerated patches about the fauces, and finally sank on the thirty-ninth day of her stay in the hospital, the fifty-seventh from the fright from which the illness dated.

An objection to the examination of the body was anticipated by the prompt action of the house surgeon, Mr. Parker, as far as the brain and cord were concerned, and to him I am indebted for being able to examine the cord and portions of the brain, as well as for the use of sections which he himself prepared.

The appearances of the corpora striata did not indicate any changes beyond hyperæmia. The veins were mostly loaded with blood, the large superficial vein, which was similarly affected in the case of Mary C——, No. II., conspicuously so. The injection was more marked in the veins than the arteries ; in some cases where vessels of each sort lay together, the vein only was full.

There was no evidence of ante-mortem coagulation, of extravasation either of corpuscles or liquor sanguinis, or of erosion of tissue. Some empty dilated veins were seen which apparently had been loaded to distension at no remote date.

There were also evidences of hyperæmia in the medulla, which increased on reaching the cord and attained their climax in the cervical enlargement, where was a large mass of extravasated blood. This, which was obvious enough to the naked eye, lay in a torn cavity within the left horn, involving the outer margin of its central and posterior portion. The clot in transverse section measured $\frac{1}{12}$ of an inch long, and a third as much in width. The blood had evidently been effused long enough before death to have undergone change ; though still characteristic in colour, it had lost its corpuscular structure.

Beside the large mass described, there was evidence of

hæmorrhage—blood-coloured exudation mixed with products of the disintegration of adjacent surfaces—in the anterior fissure, the central canal, and one of the arterial channels of the commissure.

Some of the vessels of the cord of both kinds, more especially in the lateral columns and posterior horns, were distended. Some were strikingly so in the right posterior horn, in the spot corresponding to the hæmorrhage on the other side. Distended vessels were seen in the rest of the cervical region, and, indeed, to a less extent, throughout the whole cord. Vessels, chiefly venous, irregularly distended often to the utmost, were frequent in the white matter impinging upon the lateral aspect of the grey. These were numerous in the dorsal region, but there was no hæmorrhage save in the cervical.

Perivascular erosions, in which the products of destruction of tissue were mingled with those of exudation, were found in the lower portion of the medulla and in every region of the cord. These were placed chiefly at the bottom of the anterior fissure, and continuously with it in the transverse commissure. In this case, however, the destruction of tissue was less marked than hyperæmia and extravasation.

Case V.—John P——, aged 11, came under my care in St. George's on the 30th of December, 1874. Six weeks previously he had been attacked with acute rheumatism, the articular pains of which lasted three weeks. They were immediately succeeded by an habitual swinging of the arms and legs, which when remarked upon the boy said was "for exercise." Twitchings of the facial muscles ensued, with jactitating loss of command of the limbs and nearly complete loss of speech, apparently from difficulty in the articulation rather than in the choice of words. He had a loud systolic murmur at the apex, with evidence of hypertrophy of the heart. Under the valerianates of zinc and iron the choreic movements lessened, the use of the limbs and the power of speech were slowly restored, and on the 27th of January he was sent to Wimbledon as convalescent.

There the heart symptoms became more pronounced without any return of those of chorea. He had pain in the chest, blueness of the lips, and intolerance of the horizontal posture. In the night of February the 12th he expired with signs of cardiac distress.

On post-mortem examination it was found that the surfaces of the pericardium were universally adherent partly by old and partly by recent lymph. The free edge of the mitral valve was fringed with a row of fibrinous beads, which were most abundant upon its auricular surface. The edges of the aortic valve were similarly fringed. The lungs were slightly congested and œdematous, and the bronchial membrane vascular. All the other organs, including the brain and cord, were natural to the naked eye.

Subsequently sections from almost every region of the brain were examined microscopically. They were in most instances natural, the nerve-cells invariably so. Save some injection of the vessels not enough to be decidedly morbid, though the veins were much distended in particular about the dentate bodies of the cerebellum, the vessels and their canals were normal. There was no extravasation, effusion, or erosion.

In two situations, however, were remarkable exceptions to these statements.

In the deeper white matter of one of the cerebral convolutions were many conspicuous spots which consisted of accumulations of crystals of hæmatine mingled with indefinite *débris* probably of nervous origin, swelling the canals around arteries which still remained distended with blood.

The other region referred to as the seat of significant change is that of the corpora striata. These bodies were more minutely injected than the rest of the brain, the capillaries, as well as the larger vessels of both classes, being packed with blood-corpuscles; and numerous spots, striking objects under the microscope, were closely set in their substance. These consisted each of an artery in section, empty, crumpled, and collapsed, and surrounded with a mass of globular *débris* which had been formed at the expense of the surrounding tissue. They had evidently been produced by a solution or destruction of tissue around the vessel, consequent upon effusion from it, the result of injection, which had now ceased to exist.

The spinal cord displayed loaded vessels and eroded fissures, such as were seen in every other instance examined. In addition to these common changes the grey matter had undergone extensive transformation of the kind to which the term *sclerosis* has been given. This was slight in the cervical region, extreme throughout the dorsal, absent from the lumbar. The change

was confined to the grey matter, which it affected on both sides of the cord, nearly symmetrically. In the dorsal region it involved at least a third of the grey matter, as seen in section; the affected portions on each side being adjacent to the attachment of the transverse commissure and at the root of each posterior horn. In the cervical region, though the change was less extensive, its position was the same. The altered grey substance had become converted into a wool-like entanglement of curving areolar fibres, among which nerve-cells could be sometimes traced, especially near the edges, but from which all other nerve elements had disappeared, leaving a mere confusion of connective tissue. The nuclei proper to the healthy structure were present, but had undergone no increase, nor was there any other evidence of fibroid or connective new growth. The change seemed to consist essentially of a destruction and removal of the nervous elements, their fibroid skeleton only remaining. This was best displayed in glycerine.

Case VI.—On the 16th of March, 1874, Louisa W——, aged 13, came into St. George's, under the care of Dr. Ogle, with slight general rheumatism, affecting chiefly the knees and ankles, which had begun with a rigor six days previously. She had a loud systolic murmur at both base and apex, and slight movements characteristic of chorea. It was learned that she had, on two previous occasions, the date of the earlier not recorded, the later a year ago, had chorea together with rheumatism, the choreic in each instance having distinctly preceded the rheumatic affection. Her symptoms in the final attack were not apparently threatening, and the chorea in particular obtained little attention. She died suddenly in the night on the thirteenth day of her last attack, the seventh of her stay in the hospital. The treatment had chiefly consisted of small doses of citrate of potash.

On post-mortem examination the mitral valve was found to be thickened and narrowed, while a line of soft fibrinous beads traversed its auricular surface near the free margin. The aortic valves were thickened and puckered, and to a roughened spot some nodules of fibrine were firmly adherent. The left ventricle was contracted; the right contained firm, partly decolorised clot. The pleuræ each contained about a pint of clear fluid, the pericardium an ounce. The kidneys were congested; all the other organs were natural save the nervous centres. The

membranes of the brain and cord were congested; the puncta vasculosa of the brain were large and prominent; no other evidences of disease were apparent to the naked eye. Mr. Warrington Haward, to whom I am indebted so far for the post-mortem observations, kindly preserved for me the brain and cord in view of a more minute examination, of which I will now epitomise the results.

These were of two kinds—recent injection and its consequences belonging to the last attack, and ancient changes due probably to congestive processes associated with one of the earlier.

Beginning with the brain and taking the recent changes first, there was, especially between the floor of the lateral ventricles and the base of the brain, a remarkable injection, most conspicuous in the veins and capillaries, but sometimes involving also the arteries. The capillaries were exhibited diagrammatically by the blood-corpuscles within them. Large veins, in the optic thalami especially, remained thus obstructed and irregularly swollen, and in several instances blood-corpuscles had escaped from the veins into the surrounding sheath. Minute distended vessels, chiefly arterial or capillary, were here and there seen as the centre of a spot of colourless degeneration rejective of carmine, in which the nervous tissue had become transformed into a congeries of delicate "soap-bubble" globules sparsely intermingled with extruded blood-corpuscles.

The spinal cord was traversed throughout by enormously swollen vessels which were mainly venous, and were largest and most numerous in the dorsal region. Veins thus full and swollen occupied the lateral white matter and impinged upon the central grey, which itself was minutely injected, more especially in the posterior horns. Beyond these changes there was much erosion, especially in the cervical region at the bottom of the anterior fissure and in the course of the vessels in the commissure. In the same region a spot of recent destruction of the grey matter, allied to those found in the brain, existed in contact with a distended artery.

Case VII.—Mary O——, a widow, aged 54, came to me as an out-patient on the 23rd of July, 1873, and was at once admitted under the care of Dr. Fuller. She had the symptoms of chorea in a violent form. The movements of the head, face, and upper extremities were extravagant and with little interruption; the

face was in constant contortion and grimace; the left arm was somewhat more affected than the right; the lower extremities but little. She had much difficulty in putting out and keeping out the tongue, and in the articulation of words. These muscular disturbances were all increased by mental agitation. She was thin, worn, and haggard. The sounds of the heart were natural. The urine contained a trace of albumen. The movements had come on four years before without determined cause, two years subsequently to the cessation of the catamenia. There was no history of either rheumatism or fright. The condition of exhaustion in which she was admitted increased; difficulty of swallowing was superadded to the symptoms already mentioned; and she sank after having been in the hospital for a fortnight. With the increasing prostration the choreic movements diminished.

On post-mortem examination it was found that all the organs were practically natural, save only that the kidneys were somewhat shrunken and granular. The heart was perfectly healthy.

The cord in the cervical and upper dorsal regions was distorted and torn by the intrusion into its substance and fissures of masses of translucent matter. These pools of exudation, as they may be regarded, presented themselves with much regularity in certain situations; one in the anterior fissure, one smaller and less constant in the posterior fissure, and a large one within the grey matter of each posterior horn. Beside these were others, smaller in size and various in position. The anterior fissure in the upper part of the cervical region was filled from top to bottom, and to distension, with material for the most part structureless, though fringed with nervous detritus. At the bottom of the fissure the commissure was in some places deeply eroded. The pools in the posterior horns, which were evident to the naked eye by their size and transparency, were so disposed that a line connecting them would have fallen but little behind the commissure. They were situate, that is, towards the bases of the posterior horns, precisely in the spots which in some of the earlier cases were the seats of hæmorrhagic extravasation, and in one of the later of so-called sclerosis. The masses in question, though often bordered by the products of nervous decay, had evidently resulted, not from transformation of tissue, but intrusion into it. They were generally in the position of vessels, though these were not now congested; in some instances the structure of the cord was obviously broken and displaced by

them; their bulk was in many instances as structureless as glass; and it was sufficiently evident that they had resulted, not in the degeneration of tissue, but in the extrusion of liquor sanguinis.

These were not noticed below the middle of the dorsal region. There was no general congestion of the cord, nor any further changes beyond what have been alluded to.

To sum up the changes in the nervous centres, taking the seven cases as they ranged in duration from two days to four years, they amount to this:

Case I.—Two days. Injection of vessels of all classes in brain and cord; most marked in the corpora striata and arbor vitæ, and in the dorsal region of the cord. Traces of erosion widely distributed. Hæmorrhage into, and distension by serum of, the central canal.

Case II.—Twenty-one days. Similar injection of brain, with the addition of superficial hæmorrhages, and exudation around the arteries of the corpora striata. Injection of cord, and periarterial erosion in the dorsal and lumbar regions, marked in the grey matter.

Case III.—Twenty-four days. Injection of the vessels of the brain of every class, most numerously about the optic thalami; some extrusion of corpuscles. Injection of the cord and hæmorrhage into the grey matter of both dorsal and lumbar regions, symmetrical with regard to the two sides.

Case IV.—Fifty-seven days. Injection of the brain, chiefly venous, and of the corpora striata. Injection and erosion of the cord, with large hæmorrhage into the cervical grey matter and smaller elsewhere.

Case V.—Sixty-four days. Venous injection of the brain, especially of the corpora striata, wherein were also periarterial exudations. Arteries in the convolutions near Sylvian fissure surrounded by blood crystals and *débris*. Injection and scattered erosions of the cord.

Case VI.—Fatal attack thirteen days. Two precedent attacks (to one of which the older changes were apparently due), the last a year ago. Recent injection, such as in the

other cases, of the bodies at floor of the lateral ventricles, and of the cord. Older periarterial degenerations.

Case VII.—Four years. Large exudations into grey matter and fissures of cord, chiefly in cervical region.

Thus the changes throughout the series were remarkably constant in kind and place.

In kind they were all directly connected with vascular disturbance. The injection was general to all the vessels, most marked in the arteries. When the sources of hæmorrhage could be determined they were always arterial; the degenerations were usually periarterial. The first visible change would seem to be the injection or distension of the arteries, succeeded by extrusion of their contents to the irritation and injury of the surrounding tissue.

In place, the changes affected both brain and cord. Whether in the brain or the cord the changes on the two sides were generally, sometimes almost exactly, symmetrical, both being often affected at the same spot, in the same manner, and nearly to the same extent; and in instances where no such symmetry was obvious, a tendency to it was indicated, as for instance in the occurrence of vascular distension on one side corresponding to hæmorrhage on the other. The parts of the brain most amenable lay between the base and the floor of the lateral ventricles in the track of the middle cerebral arteries; the substantia perforata, the corpora striata, and the beginning of the Sylvian fissures.

Of the cord no region was exempt, but perhaps the cervical and dorsal regions were usually more affected than the lumbar. With regard to the vertical or physiological subdivisions of the cord, these all, whether white or grey, shared in the vascular distension; this condition, however, was usually most marked in the vessels belonging to or in connection with the lateral part of the grey matter about the root of each posterior horn. And it is to be observed that this also was the chosen situation of the more definite and special changes, whether hæmorrhagic as in two instances, sclerose or exudatory.

Speaking generally, the chosen seats of the choreic changes are the parts of the brain which lie between the beginning of the middle cerebral arteries and the corpora striata—the pars perforata; and in the cord the central portion of each lateral mass of grey matter comprising the root of each posterior horn.

The nature and steps of the morbid process, hyperæmia, exudation, and its consequences, are open to view, but not so the causes in which the series has taken origin. Arterial repletion seems plainly concerned in the development of the disease; why or at what bidding do these vessels thus gorge themselves?

To seek the origin of the process we may fancifully attribute the hyperæmia to causes of two kinds, the first belonging to the blood, the second to the tissue. Irritants may be introduced with the blood either solid as emboli or liquid like the hypothetical fluent poisons of disease. Or the irritation may be of nervous origin, and the vascular dilatation secondary thereto.

The nature of the lesions in the brain and cord is not consistent with the somewhat attractive hypothesis of embolism. The lesions are indeed determined in position by the course of arteries, in the brain notably by the middle cerebral—favourite routes of emboli—and the perforating branches which pass from these to the corpora striata; but in none of the instances described were decolorised fibrine, detached clots, or signs of impaction detected, and the erraticism of embolic accident was wanting; the constancy indeed with which the changes repeated themselves in certain positions and the equality with which they affected both sides of the body are conclusive objections to this hypothesis. The corpora striata, for example, were affected with almost absolute symmetry, notwithstanding that these bodies receive their blood respectively from the right and left carotids, and different parts of the aortic arch.

Observations of the concurrent circumstances of chorea may possibly assist us in understanding the nature and correlation of its lesions.

PATHOLOGICAL

*Particulars of 22 fatal cases. Those cases the names of which*

| Reference | Name | Age | Previous attacks | Cause of present attack | Its duration |
|---|---|---|---|---|---|
| Nov. 19—1842 | Emma L— | 17 | No history given | — | — |
| 1844 | Mary K— | 15 | Two | Amenorrhœa for 3 months before | 3 mos. and 3 wks. |
| 1845. No. 10 | Mary H—, M. | 26 | Jactitation 4 years before, after confinement | Chorea after rheumatism; has had menorrhagia | 3 wks. |
| 1850. 113 | George S— | 19 | Rheumatism before but not chorea | Rheumatism came on 4 days before chorea, slight, only of knees | 10 dys. |
| 1855. 309 | Mary W— | 17 | None mentioned | No fright; amenorrhœa | 9 dys. |
| 1860. 295 | Mary R— | 7 | None mentioned | Fright; pushed into a ditch | 7 wks. |
| 1863. 132 | Jane G— | 16 | None mentioned | Attributed to fright; has had rheumatism, not acute | 3 wks. |
| „ 167 | Mary C— | 15 | None mentioned | Fright; shouted at in sleep; ? uterine irritation; catamenia irregular, once in last 7 months | ? |

TABLE.

*are printed in italics are related also in the substance of the paper.*

| Rheumatism | Other diseases | Heart | Pericardium | Blocks or other evidence of embolism | Other organs |
|---|---|---|---|---|---|
| — | — | Healthy; valves healthy | Healthy | — | Ovaries large; uterus congested; bowels loaded; brain and cord congested |
| No rheumatism ever | Caries of clavicle | Minute rounded vegetation on auricular surface of mitral valve | Healthy | — | Uterus, vagina, and ovaries injected |
| Acute rheumatism 2 mos. before death; 10 days after recovery chorea began | Headache | Slender fringe of vegetation on auricular surface of mitral valve | Healthy | — | Old adhesions between the spleen and diaphragm; uterus large and hard; os ulcerated; old adhesions about ovaries |
| Rheumatic fever at 12 years of age, and also since more than once | — | Row of beads of firmly organised lymph along auricular surface of mitral valve (recent?) | Firmly adherent (old) | — | Brain congested; cord congested, diffluent opposite the third or fourth upper dorsal vertebra |
| None mentioned | None mentioned | Heart healthy; no murmur mentioned | Healthy | — | Cord 'appeared' softened in dorsal and upper cervical regions |
| None mentioned | Abscesses in chest-walls, &c. | Heart healthy; sounds natural | Healthy | — | Brain pale; abscess in chest-wall full of blood consequent on movements, &c. |
| Two months before chorea, rheumatic pains redness of joints, and amenorrhœa | Pneumonia | Recent vegetations on mitral and aortic valves | Old lymph on pericardium | — | Brain and cord congested; uterine organs injected; early pneumonia |
| None mentioned | ? Masturbation or fornication | Healthy; no murmur | Healthy | — | Intense injection of the brain and cord; vagina and os bathed with pus; vagina dilated; os uteri widely open |

PATHOLOGICAL

*Particulars of* 22 *fatal cases. Those cases the names of which*

| Reference | Name | Age | Previous attacks | Cause of present attack | Its duration |
|---|---|---|---|---|---|
| 1864. 249 | Leopold L— | 11 | None mentioned | No cause assigned or to be discovered | 5 mos. |
| 1867. 118 | Harriet S— | 17 | None mentioned (unmarried) | Pregnancy; 4 months, disease coincident nearly with its beginning | 4 mos. |
| „ 305 | Mary G— | 12 | None mentioned | No cause assigned | 13 wks. |
| 1869. 11 | CatherineT— | 15 | None mentioned | Excitement (mental or uterine?); catamenia present for the first time on admission | 2 wks. |
| 1874. 89 | *Louisa W*— | 13 | Two previous attacks of rheumatism and also of chorea—chorea in each having preceded rheumatism | Slight general rheumatism several days before the chorea begun | 13 dys. |
| 1875. 48 | *John P*— | 11 | None | Succeeded upon acute rheumatism, after he had it for 3 weeks and apparently recovered | 9 wks. |
| 1873. | *Mary O*— | 54 | None known (widow); no fright or rheumatism. | Unknown; catamenia ceased 2 years | 4 years |
| 1875. | *MargaretC*— | 10 | None known | Acute rheumatism, begun 5 weeks before chorea | — |
| Ch. Hosp. 27 | Maria P— | 11 | One | History imperfect; probably connected with rheumatism (pericardial adhesion) | 2 mos. |

TABLE.—*Cont.*

*are printed in italics are related also in the substance of the paper.*

| Rheumatism | Other Diseases | Heart | Pericardium | Blocks or other evidence of embolism | Other organs |
|---|---|---|---|---|---|
| None mentioned | — | Line of beads of lymph on inner edge of mitral valve (auricular) | Healthy | — | Cord and brain injected; grey matter, altered yellow in patches |
| None | Worms, pneumonia | Mitral valve slightly thickened, and on its auricular surface some fibrinous deposits, evidently quite recent, could be pulled off | Healthy | Fibrinous block in left kidney and in liver | Brain congested, pneumonia; one round worm in duodenum |
| "No history of rheumatism obtained" | Pneumonia | Minute beads of fibrine on auricular aspects of both mitral and tricuspid valves, also on endocardium; all quite recent | Healthy | — | Pneumonia; brain wet, clots in vessels at base, but not decolorised or apparently abnormal in any way |
| None | — | Beads of lymph on auricular surface of mitral, soft and fresh (no murmur) | Healthy | — | Brain highly injected; lungs congested; old tubercle in spleen; ovarian cyst; signs of recent menstruation |
| Rheumatism with each attack of chorea | Hydrothorax | Old and recent disease of mitral and aortic valves; rough murmur during life | Little fluid in pleuræ | — | Fluid in pleuræ (see notes of brain, in text, p. 104) |
| Three wks. of acute rheumatism, on cessation of which movements began | — | Mitral and aortic valves fringed with recent fibrine | Pericardium adherent | — | Lungs congested (see p. 102) |
| None | Trace of albumen | Healthy | Healthy | — | Kidneys granular (see p. 105) |
| Acute rheumatism absent when chorea begun, cardiac results only present ? | — | Recent vegetations on aortic and mitral valves; heart hypertrophied | Healthy | — | Dilatation of cord, &c. (see p. 96) |
| | Diphtheria | Murmur not evident after first 2 weeks; recent vegetations on mitral and tricuspid valves | Old adhesions | — | Diphtheria |

PATHOLOGICAL

*Particulars of 22 fatal cases. Those cases the names of which*

| Reference | Name | Age | Previous attacks | Cause of present attack | Its duration |
|---|---|---|---|---|---|
| Ch. Hosp. v. 2. 239 | Athalia W— | 9 | None; chorea, chronic, confined to left side | None known; scarlatina (caught in hospital) 3 weeks after admission, rheumatism on this, then diphtheria | 20 dys. |
| v. 3. 28 | *Amelia P—* | 7 | None | Wet through day before attack began; no rheumatism or fright | 21 dys. |
| „ 136 | Ruth J— | 10 | None mentioned | ? Stiff neck | 4 wks. |
| „ 182 | Mary A. H— | 8 | 5 or 6 attacks, first apparently caused by fright, but preceded by rheumatism 4 months before; fright, fell into ditch | Mental excitement? | — |
| „ 227 | *Mary C—* | 10 | Bad attack of chorea (from fright, fell into pond) 2 years before; had rheumatism, slight attack 18 months ago | Frightened by a goat | 15 dys. |

TABLE.—*Cont.*

*are printed in italics are related also in the substance of the paper.*

| Rheumatism | Other diseases | Heart | Pericardium | Blocks or other evidence of embolism | Other organs |
|---|---|---|---|---|---|
| Came on for first time after scarlatina caught in hospital | Scarlatina; rheumatism 4 days afterwards; diphtheria; tracheotomy | When admitted no murmur; afterwards systolic murmur, with rheumatism; granulations on auricular surface of mitral valve | Healthy | — | Results of diphtheria |
| None | — | Murmur came on subsequently to chorea; minute vegetations on auricular surface of mitral valve | Healthy | — | See p. 99 |
| Stiff neck 4 weeks before chorea | Pleuro-pneumonia | Enlarged; slight fibrinous deposit on aortic and mitral valves | Friction when admitted extensive; lymph in pericardium | — | Scattered tubercle, some in pleura; recent pleurisy |
| Acute rheumatism 4 months before first attack, none afterwards | Pleuro-pneumonia | Slight fibrinous deposit on all four valves | Pericarditis came on at Highgate, subsequently to chorea, endocarditis of all the 4 valves, slight | — | Pleurisy (came on at Highgate); pneumonia; liver and kidneys congested from cardiac lesion |
| None | — | Systolic murmur not present until 3 days after admission; line of small recent vegetations along auricular surface of mitral, other valves natural | Healthy | — | See p. 97 |

*Abstract of preceding Table, showing the distribution of Heart Disease among the subjects of Chorea, classed according to the apparent Origin of the Chorea.*

| Origin of chorea | Conditions of heart | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Healthy | Recent vegetations on mitral valve only | Recent vegetations on mitral, with old thickening | Recent vegetations on mitral and aortic | Recent vegetations on mitral and aortic, with old thickening of both | Recent vegetations on mitral and aortic, with pericardial adhesions | Recent vegetations on mitral and tricuspid | Recent vegetations on mitral and tricuspid, with pericardial adhesions | Recent vegetations on mitral and aortic, with recent pericarditis | Recent vegetations on mitral, with old pericardial adhesions | Total number of cases affected with recent endocarditis |
| Rheumatic (6 cases) | — | 1 | — | 1 | 1 | 1 | — | — | 1 | 1 | 6 |
| Mental (2 cases) | 1 | 1 | — | — | — | — | — | — | — | — | 1 |
| Uterine (3 cases) | 1 | 1 | 1 | — | — | — | — | — | — | — | 2 |
| Rheumatic + Uterine (1 case) | — | 1 | — | — | — | — | — | — | — | — | 1 |
| Mental + Uterine (2 cases) | 1 | 1 | — | — | — | — | — | — | — | — | 1 |
| Rheumatic + Mental (2 cases) | — | — | — | — | — | 1 | — | — | 1 | — | 2 |
| Unknown (6 cases) | 2 | 2 | — | — | — | — | 1 | 1 | — | — | 4 |
| Total (22 cases) | 5 | 7 | 1 | 1 | 1 | 2 | 1 | 1 | 2 | 1 | 17 |

The salient facts of the disease—those which must be fitted together in any rational view of its nature—are these; its origin in rheumatism; its no less frequent origin, without rheumatism, in circumstances acting on the mind; and its constant association, whether it has begun in one cause or the other, with mitral endocarditis.

I have tabulated the particulars of twenty-two cases, examined after death at St. George's Hospital, and at the Hospital for Sick Children; representing the experience of thirty-three years at the one hospital, fifteen at the other (see pp. 110–115).

The almost invariable association of endocarditis at least with the chorea of childhood, at once becomes prominent, and with it the pathological difficulty of the disease, the connection between the nervous and the cardiac disturbance, presents itself.

I subjoin an abstract from the preceding table which will show at a glance the frequency of endocarditis with chorea, be the cause of the chorea what it may.

Thus of twenty-two fatal cases of chorea the heart was found to be healthy but in five; and of these five exceptional cases only one was a child. In every instance making up the large tale of cardiac disease, there were recent vegetations on the mitral valve and often also elsewhere. Thus in childhood, at least, endocarditis is an almost invariable accompaniment of fatal chorea. Evidence of present or past pericarditis was found in six of the number; a large proportion due no doubt to the frequency of rheumatism in association with chorea. The cardiac characteristic of chorea, however, is not peri- but endo-carditis; or at least beading by lymph especially of the mitral valve. Why are the valves thus constantly affected? is a question which at once presents itself. Does the endocarditis cause the chorea by embolism or otherwise? does the chorea cause the endocarditis or what passes for it? or are the two disorders the independent results of a common cause? These questions are sufficiently intricate to demand a somewhat careful exami-

nation of the circumstances in which the concurrence is found.

Rheumatism is known to be a frequent antecedent of chorea; it must be asked first of all how often the endocarditis of chorea is rheumatic. Taking the seventeen cases of choreic endocarditis in the table, and looking at the antecedents of the disease in each instance, we find three in which there was no clue as to the cause of the disease. Putting these aside we find six in which the disorder was apparently due to rheumatism; three in which pains probably rheumatic had occurred at some time, though possibly other circumstances were more concerned in the production of the chorea; one in which an adherent pericardium without any other record of rheumatism was accepted as sufficient evidence of its occurrence. Thus it was not possible by any presumption to associate rheumatism with more than ten of these cases; leaving four in which the disorder was traced, to the definite exclusion of rheumatism, to mental or uterine disturbances, or a conjunction of the two. And so far the facts point to the inference that there is a pathological link between chorea and endocarditis with which rheumatism has nothing to do.

To throw further light upon the connection of heart disease with chorea, I have tabulated the clinical details in seventy cases, under my own care at the Hospital for Sick Children, all minutely recorded by a succession of competent registrars. The causes of each attack were more fully traced than in the post-mortem series, but the evidence relating to the heart is necessarily only stethoscopic. Thus the two series, the pathological and the clinical, in some sort supplement each other.

The proportion of cardiac disturbance as displayed by this table is very great. In fourteen only of the seventy cases were the sounds natural. They were merely irregular in eleven; reduplicated or unnatural but still without murmur in three; while in forty-two of the cases valvular murmurs were found, accompanied in sixteen instances

# CLINICAL TABLE.

## 70 *non-fatal cases treated in the Hospital for Sick Children.*

*Cases dependent upon, or possibly connected with, rheumatism. Those most markedly so are placed first. Doubtful or equivocal cases are marked with an asterisk.*

| Name | Age | No. of attacks, if more than one | Cause or concurrent circumstances of each | Rheumatism | Heart | Other diseases and remarks |
|---|---|---|---|---|---|---|
| Herbert H— | 5 | — | Succeeded immediately upon acute rheumatism | Acute | Murmur at apex | |
| Amelia S— | 13 | — | Succeeded immediately, but gradually, upon acute rheumatism | Acute | No murmur | |
| Louisa H— | 8 | — | Immediately succeeded upon 3 days of slight articular rheumatism | Slight in several joints, 3 days | Loud systolic murmur at apex, nearly ceased at last | |
| Louisa N— | 7 | 3 | First attack.—Scarlatina followed by rheumatism, followed by chorea. Second.—Following fright: a scald. Cause of third unknown | Slight rheumatism after scarlatina before first attack | The second attack, action irregular, soft systolic murmur at apex. In the third, action irregular, no murmur | |
| Joseph N— | 8 | 2 | First attack followed rheumatism; the second, mental shock: policeman came to say that his father was taken to hospital | Before first attack | Second attack only noted. At first natural; after a time the sounds became irregular, and slight murmur came at apex | |
| „ „ | 10 | 3 | Third attack two years after second, brought on by fright: bullock broke from slaughterhouse | None since first attack | In third attack the sounds weak, no murmur | |
| Ellen T— | 10 | — | Slight rheumatism in the knee just before | Slight local rheumatism just before chorea | Faint systolic murmur, most audible just above and to right of nipple | |

*Cases dependent upon, or possibly connected with, rheumatism.*

| Name | Age | No. of attacks, if more than one | Cause or concurrent circumstances of each | Rheumatism | Heart | Other diseases and remarks |
|---|---|---|---|---|---|---|
| Emily F— | 9 | 2 | First attack immediately after rheumatic fever; second without obvious cause | Acute rheumatism before first attack | Faint systolic murmur at base | |
| Ann B— | 9 | 2 | Rheumatic pains before first attack; cause of second uncertain | Rheumatic pains before first attack | In second attack natural; in first not noted | |
| Ann S— | 8 | — | Come on a fortnight after general rheumatism | Preceded, with an interval, by acute rheumatism | Irregular action, soft systolic murmur at base and apex | |
| Louisa S— | 3 | — | Chorea began 6 weeks after commencement of acute rheumatism | General articular rheumatism shortly before | Soft and slight systolic murmur at apex | |
| Florence N— | 10 | 2 | First attack followed scarlatina and rheumatism | Rheumatic pains preceded first attack, accompanied second | Rough systolic murmur at apex | |
| Eleanor B— | ? | — | Pain in finger-joints before chorea began | In fingers before chorea began | Slight systolic murmur at apex | |
| Arthur D— | 8 | — | Came on 6 days after subsidence of general articular rheumatism | Acute, subsided 6 days before | Systolic murmur at apex | |
| Arthur B— | 10 | 2 | First came on after pains in joints; second after excitement at school | Rheumatic pains before first attack | Slight occasional murmur at apex in second attack; state of heart in first not known. | |
| *Emily M— | 9 | 4 | First and second attacks after fright; cause of others unknown | Doubtful pain in ankles before first attack | Loud systolic murmur at apex and base | |
| William Q— | 9 | — | Mother and aunt insane; child restless, excitable, and subject to delusions; no ostensible cause | Slight in one joint after chorea had come on | No murmur, second sound sharp | |

| | | | | | | |
|---|---|---|---|---|---|---|
| * Jessie I— | 7 | 2 | First attack immediately preceded by 'low fever' of 3 weeks duration; cause of second unknown | None? at beginning of second attack; complained of 'something pinching her toes' | In first attack occasional systolic murmur at apex; in second ditto, with irregular action and doubtful old pericardial friction | Herpes |
| Henrietta D— | 10 | 2 | Father a lunatic; first attack, fright: knocked down by a cab; second unknown | Attack of rheumatism between those of chorea | Action irregular; prolongation of first sound to right of nipple | |
| Mary W— | 11 | — | Not decided; began with pain in right arm and thumb; possibly, but not apparently, rheumatic | Doubtful pain in arm and thumb | Systolic murmur at apex and base | |
| * Charles C— | 11 | — | No ascertained cause; pain, probably not rheumatic, in right arm when it began to twitch | Probably none; pain in arm | No murmur, sounds irregular | Very severe |
| * Isabella J— | 11 | — | Father epileptic; two years ago had scarlatina, followed by rheumatism; chorea, of 5 weeks date only, preceded by irritability | Scarlatina and rheumatism 2 years ago; none in connection with present attack | Slight systolic murmur at apex; afterwards only occasional | |
| Henry B— | 7 | — | Not to be recognised | Slight rheumatism 9 months before | Systolic murmur at apex | |
| * Ellen G— | 10 | — | None recognised; choreic movements, preceded by pain in limbs; choreic (?) | Pain in limbs, probably choreic | Irregular, faint systolic murmur at apex, at base, and in vessels | |
| * ElizabethG— | 12 | — | No immediate cause for chorea; two attacks of rheumatic fever, the last a year back | Two attacks of acute rheumatism previously | Action irregular, slight mitral murmur | |
| * Louisa M— | 10 | 2 | Indefinite pains in legs and arms, possibly rheumatic | Vague rheumatic pains | Systolic murmur at apex | |
| * Mary M— | 10 | — | Immediately succeeded very painful extraction of tooth; rheumatism after scarlatina 4 months before | Scarlatina 4 months before | Irregular, no murmur | |
| * Charles K— | 10 | — | Pains in limbs and shoulders at beginning of chorea (choreic?) | Doubtful pains in affected limbs (choreic?) | Soft blowing murmur at apex | |
| * Caroline P— | 9 | — | Came on after 'low fever' | Has had occasional muscular pains, thought to be rheumatic | Soft systolic murmur at apex, not constant | |

*Cases apparently entirely unconnected with rheumatism (from fright or other mental disturbance).*

| Name | Age | No. of attacks, if more than one | Cause or concurrent circumstances of each | Rheumatism | Heart | Other diseases and remarks |
|---|---|---|---|---|---|---|
| Arthur L— | 8 | — | Fright; chased by a cow; movements came on same night | None | Indefinite murmur at apex; subsequently ceased | Most severe |
| Emily E— | 9 | — | Frightened by drunken man; sleepless same night; illness immediate | — | Action irregular, no murmur | |
| Ann N— | 7 | — | Struck by her father when drunk; much terrified; chorea immediate | — | Action irregular, no murmur | |
| „ „ | 9 | — | Second attack 2 years later; came on after being 'smacked;' insanity in family | — | Action irregular, no murmur | |
| Annie S— | 7 | 2 | Much frightened by horse plunging; chorea immediate; cause of second attack not apparent | — | Natural with first attack; slight basic murmur with second | |
| Sarah E— | 11 | 2 | Repeatedly frightened by having to pass a certain dog; second attack also from fright | — | No murmur; intermittent pulse | Rickets |
| Georgina B— | 9 | — | Frightened by going into a room where was a corpse | — | Action irregular, no murmur | |
| Charlotte B— | 9 | — | Frightened by stories told her with regard to a hole in the ceiling; chorea immediate | — | Slight systolic murmur at apex, which went away | |
| Jeremiah C— | 10 | — | Father came home in the night drunk, and greatly alarmed him | — | Occasional prolongation or slight systolic murmur at apex | |
| Matilda M— | 9 | — | Frightened by a dog | — | Sounds irregular and muffled; first sound almost with murmur | |
| James F— | 11 | 3 | First due to fright; nearly run over; cause of two other attacks unknown | — | Action irregular, soft systolic murmur at apex; diminished with cessation of chorea | |
| Richard B— | 10 | 1 | Fright; horse ran away; chorea began with partial paralysis same evening | — | No murmur | |

| | | | | | | |
|---|---|---|---|---|---|---|
| Alice B— | ? | 2 | Both after being beaten | — | Irregular occasional murmur at apex, slighter at base | |
| Susan D— | 10 | 2 | First after bite of dog; cause of second uncertain | — | Action increased, but no murmur | |
| Matilda C— | 8 | — | Attributed to fright some months previously; uncertain | — | Irregular, slight prolongation of first sound | |
| Thomas M— | 9 | 2 | No ostensible cause for first attack; fright for second | — | No murmur, second sound loud and reduplicated | |
| Elizabeth B— | 6 | — | Frightened: boy played a trick upon her | — | Action irregular, no murmur | |
| Ann D— | 9 | 2 | Both attacks attributed to fright, but not very conclusively | — | Action irregular, slight systolic murmur | |
| Emily S— | 9 | — | Threatened to be beaten; immediately ill, but did not twitch for a fortnight | — | Murmur, with first sound at apex | Chorea ceased on access of severe tooth-ache |
| | | | *Causes various or not to be ascertained* | | | |
| Emily C— | 11 | — | Hysteria (?) no other or more definite cause ascertainable | — | Natural | Hysteria |
| Henry J— | 5 | — | Thread worms (?) | — | Natural | |
| Alice H— | 12 | — | Congenital syphilis (?) | — | No murmur | |
| Emma K— | 8 | 3 | No rheumatism or fright to be traced; thread worms (?) | — | The first attack.—Sounds irregular when admitted, but no murmur; loud systolic murmur at apex afterwards appeared. The second attack.—Sounds irregular, but no murmur. The third.—Sounds natural | All attacks very severe |
| Eliza C— | 10 | — | Attributed to hard place; no other cause ascertainable | — | Slight systolic murmur at apex | |
| Emma K— | 9 | — | Congenital syphilis (?) | — | No murmur | Corneitis |
| Charles C— | 9 | — | Came on after getting wet, but neither arthritic pains nor fright | — | Natural | |
| Henry P— | 10 | — | Of 4 children in family 3 had chorea; no ostensible cause | — | Loud murmur, with first sound at apex, afterwards diminished | Very severe |

*Cases apparently entirely unconnected with rheumatism (from fright or other mental disturbance).*—Cont.

| Name | Age | No. of attacks, if more than one | Cause or concurrent circumstances of each | Rheumatism | Heart | Other diseases and remarks |
|---|---|---|---|---|---|---|
| Ann C— | 9 | — | Cause not to be ascertained; no rheumatism or mental cause | None | Natural | |
| Alice G— | 6 | 2 | " " " | — | Systolic murmur at apex throughout both | |
| Ann J— | 9 | — | " " " | — | No murmur, misses a beat now and then | |
| Clara W— | 10 | — | " " " | — | At first action irregular only; afterwards faint systolic murmur at apex | |
| Thomas D— | 8 | — | " " " | — | No murmur | |
| Emily C— | 11 | — | " " " | — | No murmur | Hysterical cough |
| Frank W— | 6 | 2 | " " " | — | Action irregular, no murmur | |
| Harriet A— | 9 | — | " " " | — | Irregular; faint mitral murmur, persistent | |
| Emma S— | 8 | — | " " " | — | Action irregular; afterwards faint murmur at apex with first sound | |
| Mary S— | 9 | — | " " " | — | Prolongation of first sound at apex not amounting to murmur | |
| Horace B— | 9 | — | " " " | — | Systolic murmur at apex | |
| Emily H— | 12 | 2 | " " " | — | Natural | |
| Emily G— | 9 | 2 | " " " | — | Action feeble; no murmur | |
| Elizabeth P— | 8 | — | " " " | — | Natural | |
| Louisa W— | 11 | — | " " " | — | Action irregular; occasional prolongation of first sound at apex, also slight occasional murmur at base | |

with marked irregularity of action. Thus the total irregularity reached twenty-seven, or more than a third of the whole. In the forty-two instances in which valvular murmurs were found, these were systolic in every instance; at the apex only, in thirty-four cases; at apex and base, in six; at base only, in two. In one instance pericardial friction of some standing was audible.

Thus mitral endocarditis as characteristic of chorea is no less evident clinically than after death. It is to be remarked, however, as of common experience in chorea, that the murmur though mitral and organic is not always persistent; there are in this series six examples to the contrary. The evanescence of the murmur is no doubt due to the small amount of lymph usually deposited, and the nearly complete recovery possible to the valve. Passing from the murmurs of chorea in general to a particular consideration of their frequency in relation to its source, I append an abstract of the clinical table (p. 126).

This statement shows, as did the post-mortem series, that whatever be the origin of the chorea the heart is often—and it may be added is similarly—affected. Where the disease clearly succeeded upon rheumatism, valvular murmurs were present in a large majority of cases; in twenty-two out of twenty-eight. Where it was as clearly produced by fright, without rheumatism, valvular murmurs were still present more often than not.

In twenty cases in which the chorea was attributed to mental causes, while careful inquiry failed to elicit any history of rheumatism, there were eleven with valvular murmurs, and seven more with only irregularity of action. The circumstances in which the disease arose were in at least sixteen of the instances so definite, while the result was so immediate, that there was no room for doubt as to its veritable origin in fright. These facts would seem to show that endocarditis (for the mitral murmur of chorea, as post-mortem evidence shows may be taken as always endocarditic) is a consequence of chorea; since it is equally impossible to suppose that these hitherto healthy children

*Clinical Condition of the Heart in Chorea as produced by different Causes (in 70 cases).*

| | | Sounds natural | Sounds irregular or unnatural but without murmur | Valvular murmur |
|---|---|---|---|---|
| Association with rheumatism ascertained or presumed (28 cases). | Succeeded definitely and immediately upon rheumatism (13 cases) | 2 | — | 11 |
| | Rheumatism at same time, but not obviously connected with chorea (4 cases) | — | 1 | 3 |
| | First attack from rheumatism, second attack from fright (2 cases) | — | — | 2 |
| | Has had rheumatism; parent insane (2 cases) | — | 1 | 1 |
| | Has had rheumatism; fright the immediate cause; parent insane or epileptic (1 case) | — | — | 1 |
| | Equivocal pains—muscular, choreic, or rheumatic (?) (5 cases) | — | 1 | 4 |
| | Chorea succeeded upon getting wet (1 case) | 1 | — | — |
| No rheumatism known to have existed at any time (42 cases). | Succeeded immediately and definitely upon fright; never rheumatism (16 cases) | 1 | 6 | 9 |
| | Attributed to fright, but with less certainty; never rheumatism (3 cases) | — | 1 | 2 |
| | Immediately caused by fright; parent insane; never rheumatism (1 case) | — | 1 | — |
| | Associated with hysteria (2 cases) | 2 | — | — |
| | „ „ congenital syphilis (2 cases) | 2 | — | — |
| | „ „ thread-worms (2 cases) | 1 | — | 1 |
| | Cause unknown (16 cases) | 5 | 3 | 8 |
| | | 14 | 14 | 42 |

were the subjects of endocarditis when frightened, or contracted it subsequently, independently of the resultant nervous disease.

At the same time it is equally certain that endocarditis when associated with rheumatism is a frequent predecessor of chorea, and thus the connection between chorea and endocarditis is duplex, and our comprehension of the disease must be imperfect until we can unravel the mystery of this double relation.

The first question which presents itself is this. Chorea often follows rheumatic endocarditis; is the chorea caused by the rheumatism or by the endocarditis? When chorea succeeds upon rheumatism, endocarditis whether set up by the rheumatism or the chorea is so frequently present, that to ordinary and even somewhat extensive observation it might easily seem to be a necessary link between the two. But cases occur, though comparatively seldom, in which the chorea has clearly originated in rheumatism, and yet the heart when the patient comes under observation is found to give natural sounds.[1] It has never been my fortune to have watched such a case so closely from its origin in rheumatism to its ending in chorea as to be able to assert that no murmur was at any time to be heard; but rheumatic murmurs are so often persistent that the absence of one shortly after the attack renders it probable that the heart may have escaped from the first; and I think it may fairly be inferred from the evidence adduced that rheumatism may cause chorea without the intercurrence of endocarditis. And it may be further stated that there is no evidence that endocarditis apart from rheumatism has any share in the causation of the nervous malady. The embolic theory contradicted as it is by the minute pathology of the nervous centres is no less opposed by the rougher observations which the tables record. In the post-mortem series of twenty-two cases there was but one in which blocks or any of the recognised consequences of embolism were found; and it needs no large experience of

[1] See cases in table of Amelia S—, Ann B—, and Charles C—.

chorea to teach that its prevailing cardiac lesion—beading of the mitral valve, fine, close, and regular—is not such as is usually associated with the detachment and dissemination of fibrine. And to touch for a moment upon unrecorded experience, it may be added that chorea is only noticeable by its absence in cases where the occurrence of embolism has been ascertained. I have never seen an instance, though I have recorded one in which the well-known blocking as found after death had been conjoined in life with choreic symptoms.

Thus disconnecting, as we must do, chorea from mere clot-scattering, we may go a step further and say as experience warrants that heart disease except it be rheumatic is not an antecedent of chorea. Taking this with what has been already shown, namely, that chorea may follow rheumatism without disturbance of the heart's sounds, we can but conclude that the chorea is caused by the rheumatism, not by the endocarditis. The rheumatism, in short, directly produces the congestive or sub-inflammatory condition of certain parts of the brain and cord which has been shown to belong to the disorder; or in other words chorea of rheumatic origin is rheumatism of the nervous centres.

Thus chorea and endocarditis may concur as the common but independent results of rheumatism; but that there is some other association between the two is evident from the frequent succession of endocarditis upon chorea, however disconnected from rheumatic antecedents. That in such cases the endocarditis succeeds upon the chorea is certain; it is clinically evident that the murmur does so; and the cardiac change when presented after death is of a recency corresponding to this view of its origin. How often non-rheumatic chorea is thus succeeded may be seen by reference to the single cause of fright; in sixteen instances resulting from this influence, definite and uncomplicated murmurs existed in nine, and mitral endocarditis, as we are justified in inferring, as often. The conclusion cannot be avoided that the cardiac is caused

by the nervous disorder; the only doubt is how. The irregularity of cardiac action, by which choreic endocarditis is constantly preceded and accompanied, furnishes a suggestion in solution. It has long been observed that the muscle of the heart shares in chorea with other striped fibre. Of twenty-eight cases without murmur the action of the heart was irregular in eleven: of forty-two with murmur the heart was irregular in sixteen. Mere irregularity would seem to be the first change; irregularity with murmur the second. It used to be thought that the regurgitation of chorea was due only to muscular disturbance of the mitral valve; but the constant association according to post-mortem evidence of endocarditis with the murmur renders this explanation, to say the least, insufficient. But it may be suggested that regurgitation thus produced may possibly cause endocarditis, or, at least, the deposition upon the valve which passes for it. The beads are usually confined to the inner surface of the mitral valve, and arranged along the attachment of the thin edge, where a line of minute but abrupt prominences is presented to retrograde blood, but an arrangement of more gradual slopes to blood flowing normally. Thus possibly the collection of fibrine is the consequence not the cause of the regurgitation.

Whether this explanation be, or be not the true one, it must be held certain that chorea, be its origin what it may, causes the valvular beading which is commonly regarded as synonymous with endocarditis; and it must be believed that the cardiac has no share in the production of the nervous disease, large as is the influence of rheumatism in the relation.

It is not my purpose to dwell upon the causes of chorea, excepting so far as they assist in the interpretation of its lesions; but it may be remarked that its origin in the nervous system rather than as a vascular accident is consistent with a predisposition to it which can be recognised in bright and sensitive children, whose pink and white complexions and colour of hair give them what

is known as the Saxon type; and perhaps less markedly in the descendants of the epileptic and the insane.

To conclude then, we see in chorea a widely distributed hyperæmia of the nervous centres not due to any mechanical mischance, but produced by causes mainly of two kinds—one a morbid, probably a humoral, influence which may affect the nervous centres as it affects other organs and tissues; the other, irritation in some mode, usually mental, but sometimes what is called reflex, which especially belongs to and disturbs the nervous system, and affects persons differently according to the inherent mobility of their nature.

Given the irritant—mental, reflex, or rheumatic—the course of the disease has been sufficiently traced in hyperæmia and its results. The changes are widely scattered throughout that large region which lies inferiorly to the cerebral convolutions between the corpora striata and the lower end of the cord—the district of the motor and sensory as distinguished from the mental functions. The result chiefly in muscular excitement, rather than in paralysis or loss of sensation (though it is to be observed that a lesser degree of both is frequently present), may be associated with the character of the lesions which are points of irritation rather than planes of section, and as such calculated to produce irritative rather than paralytic effects; not so much to cut off as unnaturally to excite nervous function.

P.S.—In reprinting this paper—which was written twenty years ago—I have removed several references to what at that date was termed 'miliary sclerosis' and thought to be morbid, but is now recognised as a post-mortem change. The paper as first published contains some illustrations, not here reproduced, which display the dilatation of the canal, extravasations, and other congestive changes.

## *ON CHOREA, WITH REFERENCE TO ITS SUPPOSED ORIGIN IN EMBOLISM.*[1]

Dr. Angel Money, in a paper published in the current volume of the 'Medical and Chirurgical Transactions,' relates some experiments in embolism which have a bearing upon the origin of chorea. Dr. Money introduced minute artificial emboli, starch-globules, and grains of carmine into the hearts and arteries of rabbits, guinea-pigs, cats, and dogs, with the production of embolism of the brain and cord, and the supervention, when the cord was affected, of choreic movements—'involuntary movements indistinguishable from those of chorea.' The embolic theory of chorea, fathered by Kirkes and espoused by Dr. Hughlings Jackson, has thus received a sort of confirmation—a confirmation by synthesis, one to which it is impossible not to attach significance. This introduction of a new fact makes it necessary for those who, like myself, have been opposed to the embolic theory to reconsider their conclusions. Clinically, while the connexion of chorea with acute rheumatism is consistent with, and probably suggested, the embolic theory, the no less frequent origin of the nervous affection in mental emotion, particularly fright, is inconsistent with that hypothesis. It is a familiar observation that though the chorea of mental origin continually becomes associated with a cardiac murmur, yet that the chorea precedes any alteration in the rhythm or sounds of the heart, as if the cardiac were the result, not the cause, of the nervous disorder. Again, chorea displays a preference for families in which insanity, epilepsy, neuralgia, or other neuroses have declared themselves, as if nervous structure rather than vascular accident were in question.

I have a number of sections from the nervous centres in chorea which helped to form the basis of a paper in the fifty-ninth volume of the 'Medico-Chirurgical Transactions.' I have reviewed these with the help of Dr. Money's obser-

[1] *Lancet*, January 2, 1886.

vations and ten years' additional experience on my own part, and will proceed briefly to state the facts as they now present themselves, and collect the light which they throw upon the nature of the disease.

The facts of morbid anatomy may be thus summed up. There is in fatal cases of chorea a remarkable fulness of both arteries and veins in the upper part of the spinal cord and in the brain between the top of the cord and the lateral ventricles. The veins are more generally filled than the arteries, though the filling of the arteries, being more exceptional, is the more significant. Usually the vessels, whether arterial or venous, contain blood in a recent or unaltered state; but notably in one instance an artery under the corpus striatum, apparently a branch of the middle cerebral, contained decolorised clot, whether an embolus or a thrombus. This did not necessarily indicate more than a past stasis, whatever its cause. In many parts, both in the brain and cord, but most numerously in the cord, are seen the results of erosion, exudation, and hæmorrhage. Distension, erosion, exudation, and hæmorrhage, are distributed with some approach to regularity in the affected regions, with greater frequency in some parts than others, more in the grey matter of the cord than in the white, more in the lateral portions of the grey horns than elsewhere, and more in the lower parts of the brain than the upper. The changes frequently approach, and sometimes present with completeness, a condition of bilateral symmetry; they are often further advanced on one side than the other, but it is the rule rather than the exception to find that if there be a marked hæmorrhage on one side, as in one horn of the cord, a lesser degree of hæmorrhage, or possibly only vascular fulness, will show that the corresponding part on the other side is in a similar or allied condition.

I will dwell in a little further detail on this tendency to bilateral symmetry, because, though it is not unknown for embolic accidents to occur with some approach to similarity on the two sides of the body, yet, if this symmetry can be

shown to present itself with anything like constancy, a cause which acts with more regularity than embolism must be suspected. First as to hæmorrhage. In one case a great hæmorrhagic extravasation was found occupying a large portion of the grey matter on one side of the cord, while on the other side, in the corresponding position, were remarkably distended vessels. At the same level blood was extravasated into the anterior fissure, close to the commissure. In another case were considerable extravasations in the grey matter of the cord, in both dorsal and lumbar regions, similarly placed at the same level on both sides, at what may be considered the junction of the anterior and posterior horns. Blood was also found in the anterior fissure. Thus the extravasation especially affects the grey matter—I have never seen it in the white,—and is apt to concern both sides symmetrically. This points to selection rather according to function than by chance.

In other cases were symmetrical changes of other kinds, which were even more conclusive against embolism. In that of a boy who died with heart symptoms while convalescing from a severe attack of chorea, which had set in nine weeks previously, were found remarkable tracts of translucent degeneration in almost exactly the same regions on both sides of the grey matter of the cord, accurately bounded so as to exclude the white. The grey matter, which was changed chiefly by a destruction and removal of the nervous elements, the fibroid remaining unaffected—the degeneration would probably be called sclerosis, but I avoid the use of the word, as it has been applied to changes of a different and somewhat equivocal nature,—was affected with certain differences at different levels, but at each level in such a way that one side of the cord was almost exactly the counterpart of the other. In the paper referred to[1] I have given a drawing of one-half of the cord in this case, which drawing would serve, *mutatis mutandis*, to represent with almost equal accuracy the other half of the section. The parts chiefly affected were the lateral regions of the

[1] *Medico-Chirurgical Transactions.* Vol. lix. Plate ii., Fig. 2.

grey matter, together with the outer portions of the posterior horns. It is to be observed that the parts thus affected were nearly those which in earlier cases were the seats of hæmorrhage.

To conclude the evidence of bilateral symmetry in the lesions of chorea, and of their incompatibility with an embolic origin, I will briefly advert to a case related and figured in the 'Medico-Chirurgical Transactions'—that of an old woman who died of chorea, which had lasted for four years. In the cervical and upper dorsal regions of the cord were *masses* or *pools* of nearly structureless exudation, which formed striking objects under the microscope. I say 'masses' or 'pools' because, though the exudations presented themselves as solid after the action of chromic acid, it is possible that they might not have been so during life, but have consisted of serum, which the hardening agent coagulated. Solid or liquid, they were there, were symmetrical, and occupied much the places which were chosen by other choreic lesions. The intruded material was similarly disposed in each half of the grey matter, chiefly at its outer or lateral parts and at the beginning of each posterior horn; it was also found in the anterior fissure. The post-mortems upon which I have drawn were seven in number, of which, it will be observed, I have been able to appeal to four in evidence of a systematic placing of the lesions, which must be regarded as purposive rather than accidental.

In further regard to the pathology of chorea and of embolism, it is to be noted that, while the lesions of chorea are mainly in the cord, though not confined to it, this part of the nervous system rarely presents the known and admitted signs of embolism. We easily recognise the appearances which this accident gives rise to in the brain; they are often visible, even conspicuous, upon the pia mater; but we do not see them in the cord, excepting as pyæmia, and then infrequently. It is true that the cord is less often exposed to view than the brain, but often enough to enable us to say at least that spinal embolism is rare, while chorea is common.

Again, if embolism of the cord ever occur, and no doubt

it may without being found, it would probably be in those cases in which, together perhaps with evidences of embolism in many parts of the body, there is general and minute embolism of the brain. I have seen such cases, but never found chorea among their symptoms. Further, it may be stated as a general truth that where embolism is certified to after death by blocks, impactions, or other indubitable results of the process in one organ or many, there is no history of chorea. This statement will find confirmation in the experience of every physician who frequents the dead-house as well as the wards. But not to trust to impression or recollection, I will appeal to facts in detail as presented by the very complete post-mortem and case books of St. George's Hospital, together with those belonging to the Hospital for Sick Children, which are kept on the same plan.

I have examined the St. George's records from the time when I became curator in June, 1861, by which time embolism had become well understood, until October, 1885, comprising twenty-four years and 6,787 examinations. One hundred and forty-seven of these gave evidences of embolism in the well-known shapes of blocks in the kidney, spleen, and liver, and plugging of arteries chiefly in the brain. From this enumeration I have excluded embolism confined to the right side of the heart and the pulmonary vessels, as the left side of the heart, and the systemic circulation only, can be supposed to be in relation to the nervous system. Among the 147 cases were but two in which there was any record of chorea. The subject of one of these was a girl of seventeen, whose chorea began with and was apparently due to illicit pregnancy. There was no history of rheumatism. The endocarditis and embolism succeeded upon the chorea, and were apparently consequent upon it. There were blocks in the kidney and liver; there was no sign of embolism in the brain. The other case was that of a young woman who died at the age of nineteen, with heart disease and embolism, not of the nerve-centres. At the age of twelve she had had an attack of chorea, which, as far as was known, did not recur. These were the only instances of chorea among the 147 cases of embolism. The series

included, generally many times over, every recognised result of embolism. There were thirty-four examples of cerebral embolism, together mostly with embolism elsewhere, and of these thirty-four were several in which the brain was permeated with 'embolic dust'—emboli so finely divided and widely distributed as to promise any result which minute embolism general to the nervous centres could produce. To add to this the experience of the Hospital for Sick Children, I find that from January, 1860, to June, 1885, there were 1,867 post-mortem examinations and eleven of embolism. Embolism, by this showing, is less frequent in children than adults. Chorea, as is well known, is the more frequent in children. In the eleven cases of embolism were two in which chorea was simultaneously present; one in which it had occurred, along with acute rheumatism, three years previously. Thus, of 158 cases of embolism from both sources, there were three in which chorea co-existed, two in which there was a history of it; at most five cases of chorea in 158 of embolism. Thus, if chorea is a result of embolism, it must be a very infrequent one.

To take now another aspect of the question, and look at the coincidence of embolism and chorea from the standpoint of chorea, these three cases in which embolism and chorea co-existed—the chorea displayed in the last illness and the embolism found afterwards—were the outcome of 27 cases of chorea, of which the post-mortems were recorded in the same books at the same time, 14 from St. George's, and 13 from the Hospital for Sick Children. Thus, in 27 cases of death with chorea, embolism was recognised but in 3. Considering the frequency of heart disease in chorea, whatever may be the nature of the association, embolism strikes one as occurring less often than might have been expected. Among the 27 cases of chorea, endocarditis was present in a recent state at death in 22 cases, with which in 5 pericarditis was conjoined. I have elsewhere discussed the nature of the connection between heart disease and chorea. That embolism does not supply the link is shown by its infrequency when chorea

is present, and further by the exceeding infrequency of chorea when embolism is present. It is clear from the proportion of pericarditis that the endocarditis of chorea is often rheumatic; we do not as yet know how chorea is connected with rheumatism, though it is sufficiently obvious that it is so. In many cases where heart disease and chorea have concurred it has been manifest to clinical observation that the chorea has come first, the cardiac disturbance afterwards.

*Table presenting concurrence of Chorea and Embolism in* 8,654 *cases, with post-mortem examination:* 6,787 *from St. George's,* 1,867 *from the Hospital for Sick Children.*

| — | St. George's | | Hospital for Sick Children | | Total | | |
|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Both sexes |
| Embolism . . | 83 | 64 | 8 | 3 | 91 | 67 | 158 |
| Chorea . . . | 2 | 12 | 3 | 10 | 5 | 22 | 27 |
| Embolism with chorea . . | — | 1[1] | 1 | 1[2] | 1 | 2 | 3 |

[1] Besides this, there was one case in which a girl who died at nineteen with heart disease and embolism had had chorea at twelve.

[2] Besides this, there was a case of a girl in whom chorea and acute rheumatism had occurred three years before death, with embolism.

Human pathology, from whatever point of view we have as yet regarded it, gives no encouragement to the embolic origin of chorea; while it is almost conclusive against what is the same thing in a different light—chorea as a result of embolism. The clinical consequences of embolism are well known as gathered from cases in which the characteristic lesions have been found after death. Chorea has no place among them. Cerebral embolism often presents itself with sudden hemiplegia, which may be, but more often is not, briefly preceded by convulsive movements in the affected limbs, in no respect resembling the movements of chorea. The paralytic attack is usually without loss of consciousness; it is often on the right side, and with aphasia. Shivering, vomiting, pain in the head, sudden blindness, epileptiform convulsion,[1] drowsiness, delirium, and finally

[1] See report of two cases of Cerebral Embolism by W. H. Dickinson, *British Medical Journal*, May 21, 1881.

coma, are each occasionally present. Besides these, as the symptoms of the more chronic and localised shape in which the accident happens, there is an acute form which has been called infective endocarditis, in which 'embolic dust' is widely distributed, which is characterised by symptoms which resemble those of meningitis or of one of the specific fevers—headache, vomiting, repeated rigors, high fever, and active delirium, but no localised paralysis. In this form the wide and probably general scattering would lead us to expect chorea, could this symptom result in the human subject from such a cause; but neither in the limited nor in the general form of the disorder is any such consequence to be recognised.

I will now apply another test to the supposed association of embolism and chorea as cause and consequence. It has been shown that chorea and the recognised manifestations of embolism have little tendency to present themselves together in the same individuals; do they concur in the same classes of persons as to age and sex? If the disease is a result of embolism, it is but likely that there will be some correspondence in the incidence of the two conditions—that where one is abundant there will the other occur with some frequency. Whether this is so, the following facts will show. Of the 147 cases of embolism from the post-mortem books of St. George's Hospital, 83 were in male and 64 in female subjects. This is of value as the experience of a large general hospital. Male admissions at St. George's Hospital are more numerous than female in the proportion of three to two. This does not rest on any inflexible hospital arrangement, but on the greater frequency with which men apply, owing, no doubt, to their greater share of accidents and exposure. The post-mortem books at the Hospital for Sick Children give 8 cases of embolism in the male and 3 in the female. Thus it appears that at every time of life embolism is the more frequent in the male. It must at least be allowed that this disorder does not fall upon the female with any preponderance. Now as to chorea, the proclivity of which for

the female sex is almost too familiar to need illustration. A glance at the annexed summary of post-mortem results shows 22 deaths of females to 5 deaths of males from this cause; and clinical evidence tells in the same direction. At the Hospital for Sick Children I had charge for a time of both boys and girls—for a time of boys only, for a time of girls only. Putting aside the fatal cases which I have already spoken of, I was enabled to tabulate in the paper already referred to the particulars of attacks of chorea affecting 69 children. Of these, 50 were girls, 19 boys. To take the more recent experience of the same hospital, and as a whole, without distinction of physician, I have learned from Dr. Chaffey, the present registrar, that in the last seven years 472 patients were admitted with chorea—336 girls, 136 boys. Thus embolism and chorea differ in their incidence. One has no great sexual preference; what there is is for males. Of the other the preference for females is strongly marked.

Next as to age. Embolism is connected with antecedent disease—rheumatism or other. Acute rheumatism is less frequent in childhood than in early adult life, and the other changes to which embolism is due are mostly more chronic and of still later occurrence. Thus embolism belongs rather to adult life than to childhood, while chorea belongs rather to childhood than adult life. At St. George's Hospital, where patients of all ages are admitted, the results of embolism were found on an average once in 46 post-mortems. At the Hospital for Sick Children, where the patients range from two to twelve, these results were found once in 169 post-mortems. Thus embolism is nearly four times more frequent in a mixed community than in a community of children. At St. George's the patients admitted with embolism varied in age, the males from 15 years to 74 years, the females from 3 years to 66 years. The average age of the males was 38, of the females 29, of both together 34. As to chorea, the ages of the two male subjects was in each case 11 years. That of the females varied from 7 years to 54

years, giving an average of 17, raised thus high by what may be termed the accidental or at least the exceptional inclusion of the case fatal at the unusual age of 54. The average of the male and female cases together was 16. Thus from the experience of a general hospital, where there is no exclusion on the ground of age, persons die with embolism at a mean age of 34, with chorea at a mean age of 16, the last figure being no doubt higher than it would commonly be owing to the exceptional case to which reference has been made. Chorea thus belongs to an earlier period of life than embolism.

To recapitulate the evidence which has been adduced for and against the embolic origin of chorea. When chorea is associated with acute rheumatism, endocarditis often precedes the nervous affection. When chorea arises otherwise, as in fright, the nervous affection may precede any cardiac disturbance, the valvular lesion in such cases being, so far as clinical observation can show, not the first departure of the disease, but acquired in its course. Chorea is apt to occur in neurotic families. Pathologically, though the lesions of chorea are mainly vascular and perivascular, as are those of embolism, yet the parallelism does not extend into particulars. The distended vessels of chorea almost invariably contain blood-corpuscles, not fibrine; and the lesions have a bilateral symmetry, and proneness to repeat themselves in certain places functionally chosen—habits consistent with an origin in nervous disturbance, not with one in embolism and accident. After death with chorea the embolic process was found to have occurred in some part of the body in a tangible proportion of cases—3 in 27. But taking the frequent concurrence of valvular disease and chorea, however accounted for, the proportion of embolism is probably no more than would be reckoned on as due to the heart disease, even though it were presumed that the accident had no connection with the nervous disorder. That there is no necessary connection between embolism and chorea appears by the absence of the disease among the recognisable consequences of embolism. One hundred and forty-seven cases in which this lesion was found at St.

George's gave but one of chorea; among children chorea presents itself with more frequency, but, nevertheless, 3 in 158 represents the total concurrence. Next, embolism is distributed between the sexes not very unequally, the male taking the larger share. Chorea is about three times more frequent in the female than in the male.

Finally, it has been shown that embolism selects a later time of life than chorea, the average difference being probably understated by that between thirty-four years and sixteen. This selection by chorea of females and the young, while embolism displays no such partiality, if not absolutely conclusive against their relation as effect and cause, yet tells strongly in that direction. It is conceivable that female children may have a special susceptibility, in virtue of which they develop chorea from a cause which is insufficient to produce it in the male. Alcohol causes paraplegia in the female, seldom in the male; delirium tremens in the male, seldom in the female. It is not improbable that some of the causes of chorea may be presented equally to both sexes, but affect them unequally; and so it may be with embolism. But embolism is a coarse, mechanical accident, the consequences of which are not likely to be dependent on special susceptibilities. A storm of emotion may shake or shatter the more tender structure and leave the tougher unhurt; but we have no reason to attribute any such partiality of effect to the impaction of clots. The probability is that if chorea is a result of embolism, where embolism is there will be chorea. Putting all the evidence together, it appears to me to be conclusive against the embolic origin of chorea. Dr. Money's artificial embolism caused many varieties of abnormal or forced movement, of which some resembled those of chorea. If the right place be irritated, we have no reason to suppose that the irritant must always be the same. The irritation, for aught we know, may begin in one case in the vessel and affect the nerve, and in the other begin in the nerve and affect the vessel. The artificial process may be somewhat similar in its results to the natural one, and yet the *modus operandi* of nature and art be essentially different. In both we can recognise plugged

or distended vessels, and must infer the irritation of certain motor centres, but we need not assume a resemblance in all particulars or identity of origin. It by no means follows that because choreic movements are among the results of artificial embolism in animals, chorea as a disease is the result of embolism in man. Dr. Money, in his valuable and judicious paper, carefully abstains from drawing this conclusion, and I venture to think that the facts I have adduced justify his caution in this respect.

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## *CIRRHOSIS OF THE LIVER IN CHILDHOOD*[1]

In common and, indeed, just estimation cirrhosis of the liver is a disease of advancing years, the result of habits which are generally incompatible with childhood, and of which the cirrhotic action is mostly so slow that life is far spent before the process is concluded. But the exceptions to this rule are more numerous than perhaps is generally known, insomuch that it may be said, with truth, that in this country, with children, as with grown people, cirrhosis is, of all diseases of the liver, the most frequent. The causes of cirrhosis may be divided into two, alcoholic and non-alcoholic, and it may be shown that children are liable to both. In the first place, children are sometimes exposed to the effects of alcohol at a time of life which should be a sure protection from them, while there are other causes of cirrhosis, of which congenital syphilis may be one, and possibly some other form of heredity another, to which children are amenable. Moreover, there is reason to suspect that fibrotic inflammation of the liver, as of the kidney, may be left behind by some other disease, possibly by one of the exanthemata.

Fibrosis is most frequent in age, most active in youth. This is apparent both with the cirrhotic liver and the granular kidney; the earlier either of these presents itself the

[1] *Illustrated Medical News*, April 20, 1889.

more luxuriant the new growth, and the more bulky in proportion to the rest of the organ, the more conspicuous the symptoms, and the more quickly fatal the disease. With regard to the kidney, interstitial overgrowth, together with other inflammatory changes, is but too a common as a consequence of scarlet fever; besides which, fibrous hypertrophy, in a nearly simple form, is by no means unknown, whether from scarlatina or independently of it, in the shape of the precocious granular kidney, with its profuse renal growth, its marked arterial tension and cardiac hypertrophy, and the abundant secondary lesions with which it is attended. As to the liver, much the same may be said. The disorder may present itself in childhood, even in early childhood; its essential change, the morbid development of the portal fibrous tissue, then occurs at a pace and with a profusion which contrast with the tardy and scanty production of later life. I have seen the new tissue, both in the cirrhotic liver and in the granular kidney of childhood, so full of nuclei as almost to consist of them, like a rapidly growing sarcoma.

The liver, which is the present concern, increases under cirrhosis, the growth exceeding the subsequent contraction; this superabundance of new tissue is often clearly, even conspicuously, seen without the microscope upon the section, where superabundant fibroid tissue, recognisable by its whiteness, is seen thickly massed about the portal vessels, and sprinkled throughout the organ, no doubt, in the portal region, but often where no vessels are visible to the naked eye.

But though the new growth still retains so much bulk, evidence of contraction is not wanting in the nodulation of the surface.

I will briefly relate some cases in point.

Case I.—Philip Stock, aged three years, died under my care, at the Hospital for Sick Children, on March 12, 1870. His mother had been thought consumptive in her youth, spitting blood, sweating, and wasting, but since marriage she had had good health and borne healthy children. Beyond this no history of hereditary disease was discovered. The boy in question had

good health until eight months before death, when he was attacked by measles, which was followed by scarlatina, after which he had cough, with occasional difficulty of breathing; general anasarca, the urine being dark red and scanty; frequent feverishness with loss of appetite, and increasing pallor and fever. On admission, on January 20th, there was much anasarca affecting the face and eyelids, the trunk and legs. The urine was highly albuminous. Under the acetates of iron and potash, together with digitalis, the dropsy diminished, and for a time almost disappeared, allowing a somewhat enlarged liver to be felt below the ribs. There was much pallor and anæmia; the urine remained highly albuminous, and was very often lithatic.

Subsequently diarrhœa and vomiting set in, under which the dropsy disappeared. The tongue and mouth became ulcerated, and he gradually sank.

The post-mortem examination revealed general purulent peritonitis, of which no evidence had been recognised during life. There was no tubercle, nor was any condition local to the abdominal cavity detected as its cause. The kidneys were large, white, and fatty, with much increased cortices. They together weighed $6\frac{1}{2}$ ounces. The heart weighed $3\frac{1}{4}$ ounces, not specially hypertrophied. The mitral flaps were slightly indurated, and two of the aortic segments merged into one, but there was nothing to interfere materially with the action of the organ. The liver weighed $18\frac{1}{2}$ ounces; it was markedly hobnailed on the surface. On section, the most striking change was an enormous increase of the portal fibrous tissue, which was conspicuous to the naked eye. Microscopically, some fatty change was found in the hepatic cells.

Case II.—In the same year a case with similar antecedents occurred at the same hospital, under the care of Dr. West. A boy named Frank Boyd, aged one year and eight months, was admitted with hepatic ascites. He was the only surviving child of three. He had been healthy until he had measles, followed in three weeks by scarlatina, and that in five weeks by abdominal swelling without general dropsy. He was tapped three times, after which he died, death having been preceded for a few hours only by jaundice. The liver weighed $18\frac{3}{4}$ ounces. It was cirrhosed, the lower edge rounded almost to the segment of a circle, the surface approaching the hobnailed state, the section present-

ing, both to the naked eye and the microscope, an increase of the portal fibrous tissue. Many of the hepatic cells were degenerated, as was thought, from pressure.

Case III.—Thomas Forster died at the age of one year and nine months, under my care in the Hospital for Sick Children. He had been sickly from birth, and on that account had never been vaccinated. He had had no infectious diseases, nor was there any history of syphilis, phthisis, or drink. Two months before death it was noticed that his belly was getting large, which continuing at an increased rate, he was brought to the hospital. The belly was then tensely distended, twenty-five inches in its greatest circumference, while the chest was but seventeen. The swelling fluctuated, and was covered with full veins. There was no œdema. The child was somewhat rickety, but not ill-nourished. He was prevented from lying down by dyspnœa, which presently increased, and necessitated tapping, with the removal of 61 ounces of clear green fluid. After this, the liver came into notice, much enlarged, smooth, firm, and apparently normal in shape. The urine was free from albumen. Six days after the operation he was rather suddenly attacked with abdominal pain and tenderness, together with rapidity of pulse and much prostration, under which he died on the day following.

At the post-mortem it was noticed that the ribs were beaded and the legs bowed. Two pints of turbid orange-coloured liquid were found in the abdominal cavity, the wall of which, at the site of the puncture, was indurated, and occupied by a small abscess. The peritonæum was generally injected, the intestines rosy red, and glued together with recent lymph, with which also the liver and other parts were covered. The liver weighed 17 ounces. It was enlarged, reaching half-way to the umbilicus. It was cirrhosed, its lobules widely separated and compressed by white tissue. The whole organ was firm, tough, and leathery; sections were of a deep black colour. The spleen weighed 2 ounces; it was hard and of a damson colour. The kidneys each weighed 1 ounce; they were congested, otherwise normal. The heart was normal, of the weight of $1\frac{1}{4}$ ounce. There was no tubercle.

Case IV.—Frederick Gosling died at the age of ten and a half years, under my care at the Hospital for Sick Children, in the year 1878. He was one of nine children, of whom two were dead There

was no history of phthisis or syphilis. He had had whooping cough, but no other special illness, though never robust. About nine months before death he was jaundiced for a week, after which he was never so well as before, in particular having occasional puffing of the face in the morning. About a month before death the legs and abdomen became also swollen, and he was brought to the hospital. He was then pallid, with a large fluctuating swelling of the belly and œdema of the legs and abdominal walls. The liver was not to be felt. The heart was free from murmur. The urine displayed a trace of albumen. He had frequent bleeding at the nose. 48 ounces and 54 ounces of clear yellow fluid were at different times removed from the abdomen by means of a Southey's tube. Latterly he had had loss of memory, and ultimately screaming and unconsciousness.

At the post-mortem examination the lungs were found to be generally besprinkled with grey tubercle, as also was the peritonæum. Some of the bronchial glands were caseous. The liver was in a marked condition of cirrhosis, the lower margin drawn up above the edge of the thorax, the surface covered with irregular nodulations, which varied in size from that of a millet-seed to that of a hazel-nut. The left lobe was more affected than the right. The weight was $27\frac{1}{2}$ ounces. The spleen was large, weighing $8\frac{1}{2}$ ounces; the kidneys somewhat fatty; the brain practically natural; the heart natural.

Case V.—Elizabeth Offer died under my care at St. George's Hospital, at the age of twelve. The family history was good; there was no evidence of syphilis; she had had scarlatina, measles, and whooping cough. No alcoholism was traced. She had ascites to such an extent that the belly leaked at the umbilicus with much discharge of serum and relief. The liver projected downwards to below the umbilicus. The diagnosis of cirrhosis of the liver, which was obviously indicated, was confirmed post-mortem; the liver weighed 31 ounces; it was remarkably nodular on surface, and globulated in section with a conspicuous excess of fibrous tissue in the portal region.

Case VI.—Robert S., aged seven years, came under my care at St. George's Hospital on April 24th, 1886. His mother had died of heart disease. His father had good health, though there was phthisis in his family. The boy was one of four children.

There was no history of syphilis; he had had none of the diseases of childhood, nor was anything traced to throw light upon the origin of his disease excepting in relation to alcohol. The boy at first told us that he used to have beer on week-days and gin on Sundays, and that he preferred the latter. He withdrew this statement after an interview with his parent, but it was thought that more credit was due to the assertion than to the denial. It was said he had always been pale, and the abdomen large. Two days before admission his belly was found to be so much enlarged that his trousers would not button. On admission the belly was enormously distended with fluid, an enlarged liver could be obscurely felt behind the fluid, and the superficial veins were swollen. Between the 27th of April and the 21st of the following June, inclusive of both days, he was tapped nine times, with the removal each time of a quantity of clear serum, which varied from 150 ounces to 65 ounces. The fluid then ceased to be reproduced, and he left the hospital convalescent on August 18th. He was then free from dropsy: the liver was still large, reaching to about an inch below the umbilicus. I have omitted particulars of treatment, upon which it is not my present purpose to dwell. He remained free from dropsy to the end.[1]

On the 27th of December, 1888, he was brought back to the hospital with 'head symptoms' of recent date, pain in the head, left hemiplegia, occasional convulsions, and final coma, in which state he died on January 8th. The cause of death was a large abscess in the right hemisphere, which was not conclusively traced to its source, though it was hypothetically connected with the right middle ear, which contained muco-purulent fluid, and of which the bone was somewhat inflamed.

The peritonæum was uniformly occupied, and the cavity obliterated, by old adhesions. The liver was in a marked condition of cirrhosis. It no longer extended beyond its normal limits, and was obviously in process of contraction. The weight was 20 ounces. The surface was rough and coarsely granular. The section was traversed by numerous bands of fibroid tissue, which followed the portal vessels. The heart and lungs were healthy, the kidneys somewhat enlarged and congested, but not otherwise abnormal. There was no tubercle anywhere.

[1] The dropsy was removed under the influence of dry diet; the total amount of drink was reduced to 7½ ounces a day. The case, which was a striking one, may be found in detail in a lecture in the *Lancet*, July 27, 1895 (p. 192).

As an instance in which alcohol was clearly traced as an antecedent of cirrhosis in childhood, I may mention that of a boy of the age of eleven, who was my patient at the Hospital for Sick Children. The liver was enlarged to three fingers' breadth below the ribs; he had ascites, enlargement of the spleen, and I doubt not cirrhosis of the liver, though the evidence was only clinical. His mother acknowledged to having given him beer since he was five years old, while of late years he had drunk rum and gin with his father, a drunken cabman. I recall another example of the same kind at the same hospital. It was that of a little boy who contracted the disease from the effects of rum and water, which refreshment he used to convey to his father at a cab-stand, and in which he participated.

The cases which have been brought together will suffice to show that cirrhosis of the liver is by no means absent from among the diseases of childhood, and that, as I have already said, the new growth at this point is more rapid and profuse than in later life. The causes are not always clear; alcohol is prominent among them. This irritant tells upon the active and sensitive tissues of youth with more immediate and disastrous effect than afterwards; as in later life, it usually acts first and with the greatest effect upon the liver, which stands first in the course of its absorption. This has often presented itself to my mind as a warning not to give alcohol in children's diseases too liberally, and to withdraw it as soon as possible.[1]

I long ago showed that among the results of rickets was an increase in the portal tissue. In one of the cases now related rickets was apparent, but this concurrence did not present itself with such frequency as to suggest that it was more than accidental. From the after history of such visceral enlargements it must be inferred that the portal change is evanescent; not only may an organ enlarged by rickets resume its normal bulk, but also its normal action: so that the rickety change, unlike the cirrhotic, is not permanent.

As to the action of syphilis in causing cirrhosis of the

[1] 'On the Enlargement of the Viscera which occurs in Rickets,' p. 49.

liver in children, the cases now presented give no certain evidence. It is known that the liver is affected in congenital syphilis, and it is not improbable that cirrhosis may sometimes have this origin. But it would seem, from the details before us, that other causes must be more frequent than this.

A similar statement may be made with regard to valvular disease of the heart; this is little apparent during the instances of early cirrhosis which have been selected, besides which the condition of the liver after death, mostly pale rather than congested, was not such as to indicate a cardiac origin.

A question which some of the cases suggest is whether cirrhosis of the liver, like the corresponding change in the kidney, is ever a legacy of scarlatina or any other febrile disease. Scarlatina and measles are so frequent in childhood that they could not fail to present themselves as occasional antecedents of other diseases which occur at the same time of life, even though the connection were purely accidental. It is therefore necessary to wait for further evidence before formulating any other conclusion. But at least it may be pointed out that in two of the cases of early cirrhosis, those first in the list, the hepatic change came on in close sequence upon scarlatina, in both cases preceded by measles; while in a third case these two disorders had preceded the cirrhosis, though not closely. In the first case (Stock) the scarlatina had been followed also by renal disease, which at least suggests that the simultaneous disease of the liver may have had the origin which we could scarcely hesitate in assigning to that of the kidney. The affections of liver and kidney were apparently simultaneous; did the scarlatina which probably caused the one also cause the other? I leave this question to be answered by further experience, remarking only that if scarlatina in these cases had no part, direct or indirect, in the subsequent cirrhosis, nothing else is apparent to which it could be attributed. Among the *indirect* modes by which scarlatina might conceivably cause cirrhosis may

be the effect of alcohol used medicinally. But it is also possible that the exanthem may in some cases give rise directly to an inflammatory change in the liver, as we know it so often does in the kidney.

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## *HEREDITARY ALBUMINURIA.*[1]

I PROPOSE to bring before the Society a kidney which has especial, and I may say in one respect unique interest, for it is the only one which, so far as I know, has been minutely examined of those belonging to a family in which albuminuria has been hereditary for four generations. I will briefly sketch the morbid pedigree so far as I have been made acquainted with it. Some of the persons referred to have been under my own observation; as to the rest, I have been indebted for information to other members of the family and to their medical attendants. I may say that I have alluded to this remarkable piece of family history in my book on albuminuria; but the particulars I am about to adduce are more complete, and are crowned with the final observation which alone makes them proper for the consideration of the Pathological Society.

The first generation, of which the record is explicit, consists of a brother and four sisters. The brother died suddenly, after long wasting, 'of kidney disease in some shape,' at the age of 34. Of the four sisters two died at the respective ages of 49 and 48, each having had albuminuria for many years.

The brother left six children, two sons and four daughters, which constitute the second generation. Of the six four became the subjects of albuminuria. The second son died with it at the age of 26, having had it since the age of 12. The eldest daughter died of it at 39, having had it since 16; the second daughter, still alive, has it;

[1] 1889. *Pathological Transactions*, vol. xl. In the original paper are several illustrations which are not reproduced.

the third daughter died of albuminuria complicated with diabetes.

All the sisters left children, two of the four transmitting albuminuria. The eldest sister left six children, of which more hereafter; the third left five, one of which has albuminuria. These constitute the third generation.

Of the six children of the eldest sister five became albuminuric, the second son only escaping; the eldest son has it; the third son had it in an intermittin form from early boyhood, but lost it before the age of 23; the fourth son had it from 17 to his death at 25. Of the two sisters one displayed the disease at the age of 9 months, and died with it at 33; the other displayed it at 6 months, and has it at the age of 19.[1]

The disorder presented itself in the fourth generation in the person of the only child of the elder sister, whose urine was found to be albuminous within five hours of birth,[2] and so remained.

This pedigree is not yet complete in its morbid relations; to make it so I must revert to the first generation and to the brother suspected of albuminuria, but not convicted of it, from whom all the subjects of albuminuria in the succeeding generations hitherto mentioned are descended. The wife of this ancestor was not free from the suspicion of possessing herself, or at least transmitting, an independent strain of renal disease. Her father had 'died young from

[1] This patient has since died.

[2] The presence of albumen in the urine at birth, or soon after, does not appear to be exceptional, or necessarily an indication of disease. In the case in question more importance must be attached to the continuation of the albuminuria than to its occurrence at the early period of life at which it was first observed.

Dr. Leonard Remfry, then Obstetric Assistant at St. George's Hospital kindly undertook at my request to inquire into the condition of the urine of new-born children in this particular. He found the inquiry beset with difficulty, but with perseverance and the occasional use of a catheter was eventually able to procure samples passed during birth or within a few minutes of it in seventeen cases. The quantity passed seldom exceeded two drachms.

Dr. Remfry thus states his results:—All the specimens are colourless, looking like water. Reaction various, generally neutral; sometimes faintly

some form of kidney illness.' After the death of her first husband, the ancestor in question, she married again, and had a daughter, who has had albuminuria more or less for twenty years; this daughter has a son, who is also albuminuric. It is therefore probable that the morbid proclivity under notice may have descended upon the second and subsequent generations from two sources.

It is possible that this morbid tendency may have existed for a longer time than is recorded. The family is an ancient one. Portraits have been preserved from as far back as the time of Edward IV., and I am informed that they generally display, whether from time or disease, a peculiar transparent pallor, like that which prevails among the living members. I can imagine one of this gallery of pallid ancestors addressing another, *mutato nomine*, in words which were used at a scene in which one of them bore a part—

acid or alkaline. Sp. gr. not determined. In none of the cases of albuminous urine has that of the mother been found to contain albumen. All the tests have been made with cold nitric acid, except when otherwise stated.

*Condition of Urine at Birth, as ascertained by Dr. Remfry, in seventeen cases.*

| Sex. | Albumen. | Sex. | Albumen. |
|---|---|---|---|
| Male. | Trace. | Female. | Trace. |
| Male. | Trace. | Female. | Trace. |
| Male. | Large trace. | Female. | One tenth (by boiling). |
| Male. | Trace, two hours after birth contained yellow lithates. | Female. | Large trace. |
| | | Male. | None. |
| Female. | None. | Male. | Trace; urine opalescent, contained epithelium in large quantity. |
| Female. | Much. | | |
| Female. | None. | Male. | Decided trace. |
| Female. | Faint trace. | Male. | Trace. |
| Male. | Large trace, a few granular casts. | | |

Thus of the total of seventeen observations, fourteen gave the reaction of albumen with nitric acid in the cold. This test is of course not absolutely conclusive, but the quantity of urine generally obtained did not suffice for as exhaustive examination as would otherwise have been employed.

Dr. Remfry states that the specimens, with one exception, were obtained through the kind co-operation of Dr. Daniell, House Surgeon of Queen Charlotte's Hospital.

'Look I so pale, Lord Dorset, as the rest?
Aye, my good lord; and no man in the presence
But his red colour hath forsook his cheeks.'

As regards nine of the members of this family group I am able to annex particulars which will illustrate the nature of the disease. These will be briefly given, the case which eventuated in a post-mortem examination, of which the results are before the Society, being related in somewhat fuller detail.

The numbers refer to the position of the individual in the generation as regards seniority, and correspond with those in the annexed genealogical table (p. 155).

*Second generation.* 3. *Female.*—Had albuminuria since the age of 16, and possibly earlier. Dropsy of the legs first appeared during pregnancy, at the age of 26, and continued afterwards intermittently. Up to the age of 29 the urine passed at night was said to be free from albumen; afterwards the urine was albuminous at all times. Latterly the patient became blind as the result of albuminuric retinitis, and died with uræmic symptoms at the age of 39.

*Second generation.* 5. *Female.*—When between 40 and 50 suffered much from bronchitis, while under treatment for which the urine was found to contain albumen, and subsequently sugar. The patient was gouty. I saw her about two years after the albumen had been discovered. The urine then contained 4 per cent. of sugar, albumen only to opacity. Numbers of vegetable spores were seen, such as were found in numbers 4 and 6 of the third generation. Before the diabetes became manifest the patient had had much anxiety and been disturbed by nocturnal illusions. There was occasional swelling of the ankles at night; no signs of over-arterial tension. The symptoms of diabetes latterly preponderated over those of albuminuria. Death occurred under circumstances with which I was not fully acquainted.

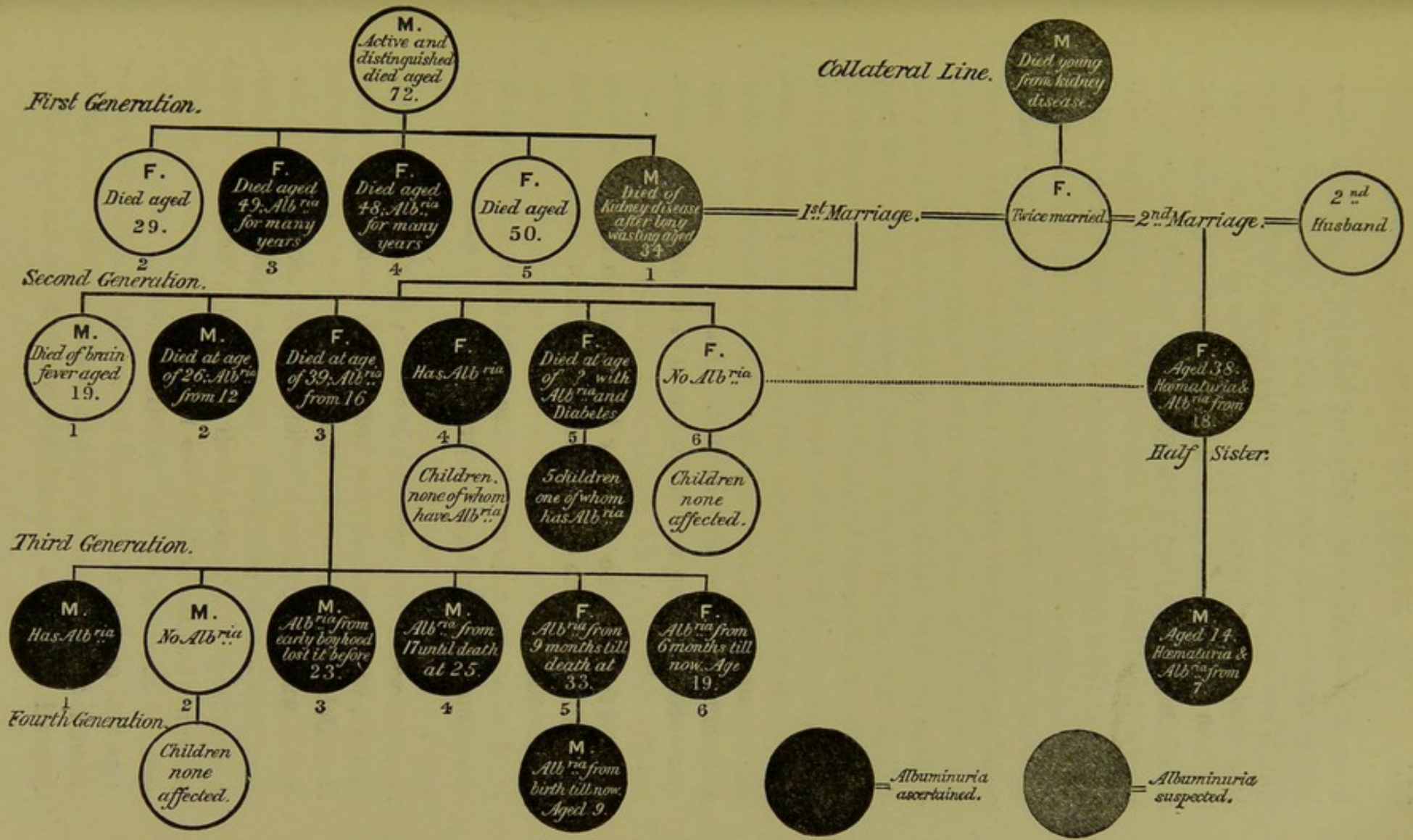
*Third generation.* 3. *Male,* aged 31.—Formerly had albuminuria rather severely, but for the last nine years, during which the urine has been frequently examined, no

albumen has been found, nor any sugar. Spores like those of the yeast-plant have sometimes been seen, and often amorphous lithates, uric acid, and oxalate of lime. He is a strong man, rather pallid, but apparently in perfect health, with no excess of arterial tension nor any evidence of renal disease.

*Third generation.* 4. *Male.*—Albuminuria first detected at the age of 17, after some excess in drink, from which time it continued until death at the age of 25. He was generally pallid. There was usually no dropsy, though occasionally a trace of œdema was found on the tibiæ. The heart's dulness was somewhat increased; the sight remained clear. About two years before death he went to travel, suffered shipwreck, with much exposure, and was severely attacked with malarial fever. On reaching home he had a return of this, the temperature going up to 103·5°; this was immediately succeeded by acute pulmonary symptoms, apparently of the nature of congestion or inflammatory œdema, agonising dyspnœa, with crackling sounds and dulness, chiefly at the left base. He sank rapidly, remaining conscious to the last.

The urine was usually pale and abundant. On one occasion when it was measured it amounted to 5½ pints in the twenty-four hours. The specific gravity ranged from 1007 to 1024; the albumen from a trace to one third, excepting on one or two occasions, when none could be found. The albumen was generally more in the evening than the morning. The urine was usually acid, sometimes highly so; once or twice it was alkaline, and was found to be ammoniacal and to contain triple phosphate. No sugar was ever found, though torulæ often were. Numerous casts were found, granular, epithelial, and hyaline. Blood corpuscles were occasionally seen. No crystals were found, excepting once triple phosphate, probably the result of changes outside the body.

The corpse was extraordinarily pallid. There was no œdema. Only the abdomen could be examined. The spleen was hard, and of about twice the normal size, probably




Genealogy of Albuminuric Family.

from malarial disease. The two kidneys weighed together just over 9 oz., the right 4, the left 5. The smaller was preserved entire, and is now before the Society; the larger was used for examination.

The capsule was much thickened, but with force came off cleanly, displaying a surface in a typical condition of granulation like that of the ordinary contracted granular kidney. The prominences were of light buff colour divided by delicate vessels. The section was characteristic; both cortices and cones of a light buff colour; the cortex somewhat diminished, so that the cones approached the surface somewhat more nearly than in health, but not so closely as is sometimes seen. The cortex was streaked with opaque yellow lines, which to the naked eye looked like what they afterwards proved to be, distended tubes. The cones were also pale in colour; in the central part of the organ they had the appearance of having been narrowed by pressure of the intermediate cortex. The whole organ was remarkably tough and fibrous in feel. One or two small cysts were seen. There was some excess of fat about the pelvis. To the naked eye the kidney had exactly the appearance of that form of the granular kidney which succeeds upon scarlatinal or some other form of nephritis. There was no lardaceous reaction.

Under the microscope, the epithelium was generally free from fatty change. There were many elongated fibre-cells.

The organ was examined in section by Dr. Delépine and myself, with the following results. I have also to thank Dr. Delépine for one of the sections, and the drawing which was shown to the Society. The results were these. In some places, especially under the capsule, which was abnormally thick, was much vascular injection. Of the more persistent changes, the most conspicuous was a great general increase of the interstitial fibrous tissue at the expense of the Malpighian bodies and tubes. The older fibroid tissue was increased; beside which there was a general profusion of delicately nucleated fibroid tissue

apparently of more recent growth. The Malpighian bodies were diminished in number, and many of them atrophied and contracted. The tubes were wanting or obscured in many parts, while in others, particularly in the cones, they were dilated, and occupied by casts which formed conspicuous objects. These were sometimes hyaline and translucent, often strongly marked, and of a coarse granular character. Besides these tubal casts were a large number of small concretions many of which were intratubal in position, and so nearly resembled the casts in texture and size that it was difficult to draw an abrupt demarcation between them. Other concretions were larger and less obviously associated with the tubes, so that it was a matter of inference rather than observation that they had taken their origin in changes within them. Some of the concretions were but little acted on by acids, and were evidently organic; others were as obviously calcareous. Dr. Delépine satisfied himself and me that some dissolved in hydrochloric acid with effervescence, and were probably carbonate; others dissolved in nitric acid without effervescence, and were probably phosphate or oxalate.

The arteries were generally thickened, as is usual with the granular kidney. The only exceptional point in the morbid anatomy was the abundant collection of concretions. There could be little doubt that these were of tubal origin, though with many all traces of their anatomical position were lost. They sometimes presented themselves as rounded groups of opaque dots which appeared to be in the interstitial tissue. It is probable that these concretions were secondary to other changes rather than the primary mischief. Dr. Delépine suggests that they may have been consequent upon the atrophy of the Malpighian bodies, and consequent want of wash in the affected tubes.

*Third Generation.* 5. *Female.*—Was for many years the subject of albuminuria, and died with uræmic symptoms at the age of 33.

*Third Generation.* 6. *Female,* aged 19.—Has had albuminuria since she was an infant at the breast. Has a

remarkably pale delicate complexion. Has been generally free from dropsy excepting occasional traces of œdema on the tibiæ. The cardiac dulness has been thought to be somewhat increased, and the pulse rather tense. The retinæ have been examined and found to be normal. Eczema of the skin has been occasionally present. From its first detection albumen has been constantly found in the urine, occasionally only a trace, but as a rule in tangible quantity ranging up to two-fifths, the amount being greater latterly than in earlier years, and usually greater in the evening than the morning. The presence of fungoid vegetations frequently suggested search for sugar, generally with a negative result; once reduction of copper gave evidence of a small amount. The quantity of the urine was not noticeably abnormal; the specific gravity varied from 1007 to 1026. It was generally acid, free from crystalline deposit, excepting once or twice when it was alkaline, probably from decomposition, and displayed triple phosphate. Large granular and epithelial casts [1] were often found, and occasionally blood-corpuscles.

*Fourth generation. Male*, aged 9 years.—Within five hours of birth the urine was found to contain albumen to the amount, as I was informed, of about one-twelfth. The urine remained albuminous, the albumen varying from a cloud to one-eighth; the specific gravity from 1020 to 1027. The urine was generally pale and highly acid. Uric acid was often deposited in considerable quantity. Blood-corpuscles were occasionally found, and once a well-marked finely granular cast. Sugar was always absent. There was never any œdema or any recognised constitutional symptom of renal disease.

*Collateral Branch.*

*Second generation. Half-sister*, aged 38.—Became subject to attacks of hæmaturia at the age of 18, and has since repeatedly passed blood with the urine, and often albumen,

[1] Since this report was drawn up this case has ended fatally, with renal symptoms.

when there was no blood. The albuminuria has not been constant. On some occasions no trace of albumen could be found; at other times the albumen was considerable and the blood often enough to give a strong colour. The urine was more bloody and albuminous during pregnancy, and it was thought to be made worse by cold. It frequently contained lithates, and often large granular casts. There has never been any œdema or constitutional sign of renal disease unless pallor be so looked upon. The habitual health was regarded as vigorous.

*Third generation. Half sister's son*, aged 14.—Only son of half sister. I first saw him at the age of 7, when I learned that he had been liable to attacks of hæmaturia for several years. These, in which the urine became deeply smoky and displayed abundant blood-corpuscles, came on at regular intervals, and were often preceded by severe rigors and attended with pain in the loins and over the pubes. Hæmoglobinuria was suggested by the rigors, but negatived by the uniformly corpuscular character of the blood. The bladder was sounded for stone, with a negative result, and a hypothesis of tubercular disease of the kidney, which was advanced by one of his advisers, was set aside by the persistent absence of pus in the urine and the progress of the case. The attacks were not brought on by exercise. It was thought that cold sometimes induced them, but more often they were without ostensible cause. The albumen was generally slight, often only a trace, excepting when the blood was present. The urine was often lithatic, and sometimes displayed crystals of uric acid and of oxalate of lime. Large epithelial casts were sometimes found. There was usually no dropsy, though once or twice under the attacks the face and eyelids became puffy. The boy had formerly a delicate and anæmic appearance, but of late has gained more of the aspect of health.

The general clinical resemblance in the group of persons affected makes it probable that the complaint was of the same nature in all, though not always to the same degree. Casts were found in five of seven individuals whose urine I

examined, besides which there is a general resemblance in history and symptoms. The albuminuria shows itself early, in one case at 9 months, in another at 6, in a third at birth. The disorder is of long duration, often lasting from childhood to middle life; the symptoms of it have usually been long in abeyance or little conspicuous, pallor and perhaps an occasional trace of œdema being in many cases for long the only ostensible results. In one instance, in which the uræmic characters were strongly declared, the disorder was complicated with repeated pregnancy. Although usually persistent there is at least one case in the group in which the albuminuria, after lasting many years, passed off.

If we may apply to the rest the morbid anatomy of the one case in which it was displayed, it would seem that the disorder is a chronic form of diffuse nephritis which affects the tubes and Malpighian bodies, and usually (but perhaps not invariably, if we may judge by the one instance of recovery) the interstitial tissue.

As to the cause of the renal inflammation, or irritation, we may first ask whether any habitual state of urine, or urinary diathesis, which could underlie and bring about the organic change is to be traced as a family possession. Such an inquiry is discouraged at the outset by the early period of life at which the albuminuria has presented itself, in one instance at least so early that it was manifest that the conditions on which it depended must have existed before birth. Of the seven cases in which I had occasional opportunities of examining the urine, sugar was present in two, in one to the amount of diabetes, in one only a trace, and that but seldom. In all the urine was generally acid, in one highly so. Uric acid crystals were sometimes present in three, amorphous lithates in three, crystals of oxalate of lime in two. In none were there any phosphatic deposits, save once or twice when triple phosphate was found, the urine then being ammoniacal, probably from decomposition. Blood-corpuscles were often present in four instances, in two abundantly. Diabetes was therefore

not to be regarded as a general antecedent, nor did the urines generally display any such frequency or constancy of crystalline deposits as to suggest any calculous process as the general origin of the renal change. Gout was a marked concomitant in one case. I am not aware that it was so in others. The effect of alcohol was not to be entirely excluded from all, but the frequency of the disease in females and in early life absolutely excluded this as a general cause of the disease. What, then, was it due to? The facts appear to convey no other suggestion than that of hereditary tendency, bearing directly on the kidneys; some special susceptibility or vulnerability belonging to these organs as part of their original formation. It is known that the granular kidney is sometimes a matter of inheritance, and were this a clinical society I could adduce other examples, though none so striking.

I ventured some years ago to refer to this group of persons as exemplifying the hereditary transmission of the granular kidney, though at that time there had been no post-mortem within my knowledge. What was then only a surmise now becomes a pathological fact, and as such I thought it might be worthy of the notice of this Society.

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## *ON ALBUMINURIC ULCERATION OF THE BOWELS.*[1]

I PRESUME to bring before the Society an accompaniment and result of renal disease to which I have formerly briefly referred, but which has not yet received the notice which is due to it, whether as regards its pathological interest or its vital importance. I am not aware that the concurrence of ulceration of the bowel with albuminuria found mention until 1876, when, in the Croonian Lectures of that year, I ventured to adduce two cases of this enteric lesion in connection with the granular kidney, unexplained by either typhoid or tubercle, and not to be accounted for otherwise

[1] 1894. *Medico-Chirurgical Transactions*, vol. lxxvii.

than as related in some manner to the renal disease.[1] The late Dr. Greenhow presently enabled me to add a third, which, with the two before mentioned, was cited in my second edition of 'Albuminuria' in 1877, with the recognition that the intestinal change resulted from the renal, though at that time it was not apparent how the association was brought about. In the following year I called the notice of the Pathological Society to a case of 'Ulceration of the Bowel as a Consequence of Renal Disease,'[2] with the particulars of a microscopic examination of the bowel, made by my former clinical clerk, Mr. A. Shann, which revealed the hæmorrhagic nature of the process.

During the considerable time which has passed since these observations were published I have been on the watch for the enteric complication of albuminuria, which has long taken its place—at St. George's at least—among the things to be looked for in post-mortems relating to renal disease. I now propose to ask the attention of the Society to the experience which has accumulated, making use not only of my own cases, but also of those which have occurred in the practice of my colleagues, to whom I tender my thanks for their permission to do so. To avoid the length and tediousness of complicated narratives, I have condensed the records into a tabular form, which will present in brief the essential particulars. These have been for the most part gathered from the hospital post-mortem books, though I have occasionally been able to refer to my own notes in addition. The observations extend over forty years. The first case came under my notice when I was clerk to the late Dr. Bence Jones, though the connection of the ulceration with the renal disease was not recognised until later instances had thrown a backward light upon it, and enabled it to be recovered from a remote past to illustrate comparatively recent pathology.

I have brought together twenty-two cases of ulceration

[1] *British Medical Journal*, April 22, 1876, p. 1560.

[2] *Path. Soc. Trans.* vol. xxix. p. 117.

of the bowel, concurrent with albuminuria and not with any other condition by which the enteric lesion could be explained. In two of them there was also ulceration of the stomach, possibly of the same nature, and a single instance will be found concerning the same organ which must be admitted to be of doubtful value, though of some interest in relation to the conditions under which it occurred. I have appended, with design to throw light upon the morbid process, eight instances of extravasation of blood without ulceration, but similar in place and circumstances to it.

With regard to the bowel, the ulceration was confined to the small intestine in eleven cases; confined to the large intestine in five cases; in five both were affected. In one the position of the ulceration was not more exactly defined than as 'near the cæcum.' Within the small bowel the region most frequently affected was the ileum, though no part was exempt. In the large bowel the colon suffered with the greatest frequency, though the cæcum was affected alone or with other parts of the bowel in three instances.

The ulcers were not associated in particular with any of the glandular structures of the intestines, but affected the mucous membrane without any obvious preference excepting in general terms for the lower part of the ileum. Peyer's patches were usually exempt, though in one instance (that of Williams) they were involved. The solitary glands of the cæcum were recorded in one case as intensely congested but not ulcerated. In several the valvulæ conniventes were found to be ulcerated or congested, so that, if the morbid process have any special choice, however rarely exercised, it would seem to be for these structures. In size the ulcers varied from two inches by one to spots no larger than the marks of raindrops. The ulcers were usually regular in shape, more or less circular, without thickening, and with 'punched' edges. In some instances the ulcerated surface had become covered, as if by a process of healing, with a delicate membranous film (Jackson, Hissgen).

The most conspicuous characters of the morbid change were derived from hæmorrhage, old and new, in the neigh-

bourhood of the ulcers. Pigmented stains and recent ecchymoses were common—the latter in situations, such as the edges of the valvulæ conniventes, where the ulcerative process has been known to declare itself. The most important characteristic of the ulcers is their tendency to perforate and set up peritonitis, in other words their deep origin or penetrating character.

This introduces the minute anatomy of the ulcers and the morbid process to which they are due. The most constant factor, and that which presents itself as of the greatest significance, is extravasation of the blood. The earlier observations which led to this conclusion have been corroborated by more recent; I may refer especially to some microscopic examinations which Mr. Fenton has recently undertaken at my request. These relate to two preparations in our museum which had not hitherto been examined in section. They belonged to the cases of Hissgen and Parker; and though they had been for fourteen years in spirit, there was no difficulty in recognising the hæmorrhage connected with the ulcers. It is probable that the effusion of blood in the submucous coat, which has been microscopically demonstrated in three instances, is the primary and essential lesion upon which the ulceration is consequent. The general fact of effusion of blood in connection with these ulcers rests on a much wider foundation than the few cases where it has been described with microscopic minuteness. Not only are ecchymoses and pigmentations continually associated with the ulcers, but intestinal hæmorrhages, particularly in the submucous coat, are not infrequently observed without ulceration in connection with advanced renal disease, hypertrophy of the heart, and retinal hæmorrhage. I have annexed a table which displays eight cases of this nature.

Besides the ulceration which has been described as belonging to the intestine, I have brought together three cases in which the stomach presented a similar change in similar circumstances. In two of these the stomach was ulcerated together with the bowel, and possibly by a similar process. One of the subjects was a child of eight, an age at which

ulceration of the stomach is exceedingly rare. In this instance the gastric ulcer was surrounded by traces of old hæmorrhage, while similar signs were abundant about the ulcers in the intestine. Putting aside the evidence of hæmorrhage in the stomach in this instance, the gastric ulcers were not noticeably different from those of common experience.

Putting all the observations together, we recognise in connection with chronic albuminuria a peculiar form of ulceration of the bowel which is associated with, and probably produced by, submucous hæmorrhage; while similar hæmorrhage also presents itself in the same circumstances unattended by ulceration. There is reason to believe that the stomach may, however rarely, participate in the same process.

I will take brief note of what may be called the foreign relations of the intestinal lesion and the association of hæmorrhage in the bowel with hæmorrhage elsewhere. In the total of twenty-two cases where the intestine was ulcerated, with or without the stomach, the retinæ were the seat of albuminuric changes, hæmorrhagic or otherwise, in nine. In the same series hypertrophy of the heart was recorded in nineteen, as if the intestinal lesion were but one of the cardio-vascular series.

Looking at the conditions of kidney found together with this ulceration in the twenty-two cases referred to, advanced fibrosis was present in all but two; in fourteen cases the kidneys were described as granular; in four as fibrotic, or in a state of interstitial nephritis; in two as well-marked examples of the large white kidney of nephritis. Assuming, as we may, that the large white kidneys were affected with interstitial as well as tubal inflammation, it is to be concluded that twenty of the twenty-two were in some phase of advanced renal fibrosis. Reverting to the two exceptional cases where renal fibrosis was not recognised: in one (Powell) the kidneys were certainly the seat of old disease, and were probably fibrotic, though not assuredly so. They were irregular in shape,

had adherent capsules, and one presented a cicatricial depression. The other (Louisa Booth) was a case of lardaceous disease which has especial interest in relation to the ulceration of the bowel. The lardaceous disease was not the cause of the ulceration, but its result. There had been general dropsy, presumably renal, at the age of twelve. This appears to have been succeeded by the special ulceration, perforation, and a circumscribed abscess within the peritoneal cavity. The consequent profuse discharge by the bowel led to general lardaceous disease which superseded or masked the renal mischief, and proved fatal at the age of twenty-one.

The symptoms of this complication of renal disease present themselves usually towards the fatal close, though there is evidence in old cicatrices found after death that the ulceration is not always immediately fatal. The symptoms are those of bowel irritation, sometimes such as might belong to dysentery, with which are often associated those of peritonitis and perforation. In the course of these there may be certain reminiscences of uræmia; it is difficult to say how far the occasional obstinate vomiting may have this origin, though other manifestations of the uræmic state appear to be much in abeyance when those of the intestinal lesion are prominent.

The post-mortem observations before us show that peritonitis was present in about a third of the cases, in seven of twenty-two: and that perforation had occurred about half as often, in four of the number. The symptoms of irritation of the bowel are diarrhœa and griping. The diarrhœa is by no means always present; there may indeed be constipation and a need for purgatives. The diarrhœa is sometimes profuse, and the chief cause of the prostration which in these cases is apt to be the mode of death. The motions are often liquid, and like those of typhoid; rarely they contain mucus. The griping pain is often prominent, and is most marked after food. A boy (Charles Dodd), who suffered much in this way and died with peritonitis and perforation, furnished his own diagnosis as 'a twisting

of the guts.' He had served a butcher and acquired the rudiments of anatomy.

One of the most noticeable symptoms is vomiting, which is often frequent and either spontaneous or readily provoked. A woman (Maria Jackson), under Dr. Bence Jones, had so much vomiting that for a time her disorder was attributed not to the kidneys but to the stomach ; sickness had been habitually induced by the sight of food and even by the sound of the dinner-bell. In some of the cases recorded it was a matter of doubt how far the vomiting was dependent on the state of the bowels and peritoneum, and how far uræmic. This symptom appeared most prominently when peritonitis was present. Hiccough was occasionally noticed.

Death is usually brought about by prostration and collapse, due either to the diarrhœa or to the peritonitis with or without perforation. Delirium was present towards the close in three of the twenty-two cases. The advent of the abdominal symptoms is usually succeeded by death in no long time ; in five cases where this interval was recorded it varied from two days to over two months. In one which has been already referred to (Louisa Booth) there was evidence of albuminuric ulceration a year before death, which led to perforation, a circumscribed abscess, profuse purulent discharge, and lardaceous disease. In a minority of the cases (nine of twenty-two) no bowel symptons were noted.

Without occupying the time of the Society with considerations which must be obvious, I will rest content with having invited attention, and that in somewhat more detail than before was possible, to a result of chronic renal disease which has hitherto received little notice, but which apparently belongs to a well-known class, for it is presumably of hæmorrhagic origin, is akin to the retinal alteration, and has relation to the cardio-vascular changes of which hypertrophy of the heart is the most constant indication.

The enteric manifestation has interest clinically as well as pathologically, for it commonly foretells the approach of

the fatal ending, and often brings it about in modes which have been sufficiently denoted.

In conclusion, I have to record my obligation to Mr.

## TABLES

*Cases of Albuminuric*

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| Maria Jackson. Dr. Bence Jones, 1853. Croonian Lectures, 1876 | 30 | — | 6 months | Highly albuminous, sometimes bloody; low sp. gr.; casts | Granular, contracted; arteries thickened | L.V. hypertrophied | Much dimness of sight |
| Charles Dodd. Dr.Dickinson, 1872. Croonian Lectures, 1876 | 14 | Stone? lithotrity at age of 3 | 3 years | Trace of albumen; casts; pale; low sp. gr. | Granular, contracted, fibrotic; right atrophied from stone | L. V. much hypertrophied; weight 8 oz. | Advanced albuminuric retinitis |
| Thomas Wilding. Dr.Whipham, 1878 | 39 | A great drunkard, scarlatina in infancy | Œdema 2½ months | Highly albuminous, sometimes bloody | Granular, contracted; weight 8½ oz. | Hypertrophied; weight 18 oz.; recent pericarditis | — |
| Francis J. Williams. Dr.Dickinson, 'Path.Trans.,' vol. xxix. p. 117, 1878 | 20 | Scarlatinal dropsy at age of 6 | 14 years (?); polyuria 2 years | Highly albuminous; casts; pale, copious | Granular, contracted; fibrotic; weight 8 oz. | L.V. hypertrophied; weight 15 oz. | Advanced albuminuric retinitis |
| William Shrapnell. Dr.Whipham, 1878 | 24 | — | 2½ months | Highly albuminous; pale | Very granular; weight 11 oz. | Enormous hypertrophyof L.V.; weight 39 oz.; recent pericarditis; pulmonary apoplexy | — |

William J. Fenton for making the microscopic observations to which I have already referred in connection with his name, and to Dr. Rolleston for assisting him.

## OF CASES.

*Ulceration of the Bowels.*

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| No dropsy; obstinate vomiting throughout, thought to be uræmic; epistaxis | Obstinate vomiting; latterly griping abdominal pain and diarrhœa. Died of peritonitis | Turbid fluid in peritoneum; intestines glued together by recent lymph. Lower half of ileum thickened; showed red and dark patches of discoloration, and patches of ulceration of no great depth covered with thin membrane. Colon natural | None |
| No dropsy; frequent headaches; obstinate vomiting regarded as uræmic; convulsions | Obstinate vomiting; griping pains in belly: abdominal tenderness; diarrhœa | Recent peritonitis. Lower two feet of ileum contained many ulcers, some of which were linear and corresponded with valvulæ conniventes; these often exposed peritoneum. Mucous membrane between swollen | None |
| Œdema, ascites, vomiting | Latterly diarrhœa and vomiting, with epigastric pains, especially after meals | Small ecchymoses, especially on valvulæ conniventes. In colon, for two feet below valve, small star-shaped and elongated ulcers, the latter transverse; edges congested | None |
| No dropsy after original attack; great arterial tension, epistaxis; dyspepsia, vomiting | Nausea, vomiting, griping abdominal pain, tenderness; pain, especially after food; constipation, needed purgatives; mucus in motions | Purulent fluid in peritoneum. Many small ulcers in ileum, mostly in Peyer's patches, clean cut as if punched out, three of which had perforated. Lower half of ileum congested and pigmented. Colon pigmented and ecchymosed. Microscopic sections of ileum showed dark pigment mixed with blood crystals in submucous coat | None |
| No dropsy; hæmoptysis, dyspnœa | No local symptoms; constipation, needed purgatives | Numerous ulcers in lower part of ileum and upper part of colon. In colon ulcers numerous near valve. In the middle of the ileum two old, practically healed ulcers, large as Peyer's patches. Recent ulcers small, roughly circular, and with raised edges. Hæmorrhages and pigmentation about ulcers. No peritonitis | None |

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| Frederick Hissgen. Dr. Dickinson, 1879 | 27 | — | 2 years | Highly albuminous : sp. gr. 1008 | Granular, contracted; cortices shrunk; hæmorrhages in Malpighian capsules | L.V. hypertrophied; weight 16 oz. | Albuminuric retinitis |
| Philip Parker. Mr. Holmes, 1879 | 58 | A painter | — | — | Very granular; weight 10 oz. | L.V. hypertrophied; R. V. thin; weight 10 oz. | — |
| Edward Powell. Dr. Dickinson, 1878 | 21 | — | Abscess of hip at age of 8 years; diarrhœa 2 months | Trace of albumen; scanty, high coloured | Both congested; capsules adherent; left irregular in shape withdepressed fibrous patch | Healthy; weight 10 oz. | — |
| Benjamin Cotton. Dr. Dickinson, 1881 | 46 | Had dysentery in India | Œdema 4 months | Albumen $=\frac{1}{3}$; scanty, latterly bloody | Large, white: weight 21 oz. | L.V. hypertrophied; weight 22 oz. | — |

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| No œdema; headache, vomiting | Much pain in belly for 40 days before death, especially about umbilicus, where was tenderness; constipation; hiccough; no diarrhœa; latterly delirium | Peritonitis and extravasation of fæces. Several perforations in ileum and many ulcers in various stages. Ulceration extended from middle of jejunum to middle of ileum. Ulcers generally clean cut; mucous membrane in their neighbourhood covered with thin filmy layer. Tips of valvulæ conniventes apparently first ulcerated. Peyer's patches not especially affected. Colon exempt | None |
| *Examination by Mr. Fenton*, 1893.—The submucosa is œdematous and swollen from the presence of recently extravasated blood. Around the hæmorrhages are signs of inflammation, *i.e.* small-cell infiltration, probably the result of the hæmorrhage. The vessels are engorged with blood, and the walls of the arteries are thickened. There are some pigment masses in the submucosa, probably evidence of former hæmorrhage. The peritoneum has recent lymph on its surface. The mucosa has entirely gone in parts, with, in some places, part of the submucosa; the removal has the appearance of having been effected by a process of digestion rather than by active inflammation | | | |
| Admitted with fracture of thigh; no renal symptoms recognised | Bowels confined at first, then profuse diarrhœa with abdominal pain, tenderness, vomiting, and delirium. Sank from abdominal disturbance, which began 5 days before death | Peritonitis. Descending colon and sigmoid flexure thickened and 'worm eaten.' Ulceration more or less continuous in these regions, with many recent hæmorrhages. Bowels nearly perforated in several places<br>*Postscript*, 1893.—After keeping in spirit punched-out appearance well shown. In many places round, sharp-edged hollows like indentations made by raindrops<br>*Examination by Mr. Fenton*, 1893.—Submucosa œdematous and swollen by recently extravasated blood. Small-cell infiltration around hæmorrhages, as in case of Hissgen, and probably from same cause. The vessels, especially the veins, distended with blood. Pigment masses present. The mucosa has in places been removed as in preceding case. | None |
| None beside diarrhœa | Obstinate diarrhœa for over 2 months, with exhaustion, delirium, and death | Several small ulcers of mucous membrane, surrounded by slate-coloured zones of old hæmorrhage, on valve and just above it. Congestion of small intestine and stomach | None |
| Œdema; some ascites | No bowel symptoms | Ulcer of the size of a sixpence in small intestine 2 feet above valve; ulcer clean, regular, pigmented. Congestion 6 inches above, but not about, ulcer | None |

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| Sarah Greenfield. Dr. Dickinson, 1882 | 39 | Repeated pregnancies (?) | Headache 6 months | Highly albuminous; sp. gr. 1012; polyuria | Granular, contracted; weight 8 oz. | L.V. hypertrophied; weight 16 oz. | Albuminuric retinitis |
| Louisa Booth. Dr. Dickinson, 1883 | 21 | General dropsy at age of 12, which lasted 12 months; œdema recurred 4 months before death | 11 years (?); 4 months (?) | Latterly albumen = $\frac{1}{3}$: polyuria | Lardaceous; weight 9 oz. | Weight 6 oz. | — |
| Ann Harffery. Dr. Wadham, 1885 | 19 | Scarlet fever in 1871; nephritis in 1873 | 8 years | Albumen = $\frac{1}{3}$; casts | Left kidney contracted and fibrotic; right tubal nephritis | L.V. much hypertrophied; weight 10 oz. | — |
| Ann Mackay. Dr. Wadham, 1885 | 15 | Scarlatina in 1873 (?) | — | Albumen = $\frac{3}{4}$; smoky, scanty; casts | Fibrotic, contracted, pale; capsule adherent; weight 4 oz. | L.V. hypertrophied; weight 8 oz. | — |
| Ann Shaw. Dr. Champneys, 1888 | 49 | Stricture of urethra (?) | — | Contained blood and pus; alkaline | Both granular; right atrophied; left enlarged and contained abscesses | Old mitral disease; weight 16 oz. | Hæmorrhages in both |
| George Windover. Dr. Dickinson, 1889 | 58 | Painter; gout | — | Albumen = $\frac{1}{6}$; pale; sp. gr. 1012 | Granular, contracted, cysted; weight 6 oz. | L.V. greatly hypertrophied: weight 22 oz.; recent pericarditis | Albuminuric changes in both |

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| No dropsy: headache, vomiting | Acute abdominal pain with looseness of bowels 2 days before death | Many pink injected patches on peritoneum. Many prominent congested villi on small bowel, some of which are beginning to ulcerate | None |
| General dropsy at age of 12. One year before death showed symptoms of circumscribed abscesses in peritoneum, due (as was subsequently ascertained) to perforation of intestinal ulcers, no doubt due to former albuminuria; then constant discharge of pus from bowels, and consequent establishment of lardaceous disease, which affected liver, spleen, and kidneys. Recurrence of albuminuria and dropsy, with diarrhœa, vomiting, and prostration | | Irregular ulcers in large intestine, some of which, in the sigmoid flexure, had perforated the bowel and were in connection with the abscess cavity outside; ulcers irregular in shape, with thin edges. General lardaceous disease, which was apparent during life. | None |
| No œdema latterly. Died with uræmia and convulsions | Pain in belly 10 days before death, which recurred in severe paroxysms; formerly constipation; diarrhœa latterly, abdomen resonant and tight | Ileum for 6 feet contained ulcerated patches stained with fæcal matter. Ulceration connected with congestion and insterstitial hæmorrhage. Some of the vessels plugged by coagula | None |
| No œdema at any time; epileptiform fits; urinous smell in breath. Died with convulsions | None noted | Ulcers 'near cæcum'—the largest 1 inch by $\frac{1}{2}$ inch. Cæcum and colon present many black patches of the size of pins' heads, due to intense congestion of solitary glands. Congestion present in other parts of bowel, but not to same extent | None |
| Œdema, vomiting; Died of uræmia | Vomiting (uræmic?). No distinct bowel symptoms | Large ulcer in middle of transverse colon | None |
| Œdema, ascites | None recognised | Ulcers in transverse colon | None |

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| Henry Webb. Dr. Ewart, 1890 | 52 | Painter | — | Loaded with albumen | Large, white; not lardaceous; weight 16 oz. | Weight 14 oz. | — |
| Thomas Spaul. Dr. Cavafy, 1890 | 24 | Calculi (?) | 3 months | Cloud of albumen; pale; sp. gr. 1010 | Both granular; stones in left; left weighed 1 oz.; right 7 oz. | Both ventricles hypertrophied; weight 18 oz. | Albuminuric retinitis; hæmorrhage in right |
| Henry Slemon. Dr. Ewart, 1890 | 56 | Whiskey drinking (?) | — | Albuminous; scanty, bloody | Granular, contracted; weight 4 oz. | L.V. hypertrophied; weight 12 oz. | — |
| Jane Moss. Dr. Cavafy, 1890 | 48 | — | 3 weeks | Albuminous and lithatic | Fibrotic; contained old infarcts; weight 8 oz. | Both ventricles hypertrophied; weight 16 oz. | Normal |
| William Jones. Dr. Cavafy, 1891 | 66 | — | 5 years | Slightly albuminous; copious, pale | Granular, red, contained caseous masses; weight 12¼ oz. | L.V. hypertrophied; weight 29 oz.; pericarditis | — |
| *Ulceration of Stomach possibly Albuminuric,* | | | | | | | |
| Jessie Bevan. Dr. Dickinson, 1888 | 17 | — | 2 years | Albuminous; low sp. gr. | Contracted, granular, fibrotic; weight 5 oz. | Great hypertrophy of L.V.; weight 16 oz. | Albuminuric changes |

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| Œdema, headache, vomiting, dyspnœa, drowsiness | None noted | Two sharply cut ulcers in first part of duodenum. Mucous membrane of duodenum injected | None |
| Headache, vomiting, dyspnœa; œdema latterly | None observed | Ulcer in commencement of duodenum. Cæcum extensively ulcerated, with blood in mucous coat. Old ulcers in sigmoid flexure. Tar-like fæces in colon | None |
| Œdema. Died of uræmia. Had also cirrhosis of liver and ascites | None observed | Ulcer in ascending colon. Hæmorrhage in mucous coat of sigmoid flexure | None |
| Œdema; delirium | Diarrhœa; motions like those of typhoid | Intestines discoloured externally at intervals. Internally discoloured patches to a marked extent, together with ulceration in the duodenum. A few patches of ulceration in the jejunum. Hæmorrhagic patches in ileum and sigmoid flexure. The hæmorrhages were in the submucous coat, and evidently the primary condition, the ulceration being secondary | Scars at apices of lungs |
| Much œdema; mitral systolic murmur; dyspnœa; epistaxis | None observed | One large and two small shallow ulcers in the first part of the duodenum; no thickening; no trace of new growth | None |

*with or without Ulceration of the Bowel.*

| | | | |
|---|---|---|---|
| Œdema, headache, vomiting | Epigastric pain after food (ulcers of stomach); much abdominal pain and vomiting | Several small ulcers in *stomach*, the largest about size of threepenny piece. Numerous ulcers from upper part of ileum to lower part of descending colon, in early stage. Mucous membrane thickened and ulcers 'punched out.' Numerous small brown extravasations beneath peritoneum of ileum | None |

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| Emily Potter. Dr. Dickinson, 1881 | 8 | — | 7 months | Albumen = $\frac{1}{8}$; alkaline | Interstitial nephritis; unequal in size. Bladder hypertrophied | L.V. greatly hypertrophied; weight $5\frac{1}{2}$ oz. | — |
| EmmaKillick. Dr. Dickinson, 1891 | 53 | — | 3 weeks | Albumen = $\frac{1}{3}$; lithatic | Red, granular; weight 10 oz. | Hypertrophy and dilatation of L.V.; weight 17 oz. | — |
| *Cases of Intestinal Hæmorrhage* | | | | | | | |
| Charlotte Rayner. Dr. Cavafy, 1887 | 40 | Pregnancies (?) | 7 months | Blood and pus | Granular; contracted; weight 6 oz. | L.V. hypertrophied; weight 20 oz. | Albuminuric retinitis |
| Edward Heath. Mr. Pick, 1890 | 42 | — | — | — | Highly granular; cortices shrunk; weight 10 oz. | Enormous hypertrophy of L.V.; weight 20 oz. | Hæmorrhages in both |
| Elizabeth Lucy. Dr. Dickinson, 1890 | 47 | — | 12 years (?) 2 months (?) | Solid with albumen; scanty, bloody | Congested; cortices swollen; weight 16 oz. | Weight 12 oz.; recent pericarditis | — |
| HenryFowler, Dr. Whipham, 1891 | 43 | Painter; gout | 3 months | Albumen = $\frac{1}{10}$; pale; sp. gr. 1010 | Granular; cysted; cortices shrunk; weight 9 oz. | Hypertrophy of both ventricles; weight 23 oz. | — |

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| Swelling of face; no general œdema; wasting; much cardiac tension; gums spongy and bleeding | Vomiting, diarrhœa. Day before death sudden agonising pain in abdomen (perforation) | Recent peritonitis. Local extravasation of fæces. Small intestine from about the middle shows extensive erosions of mucous membrane, with several well-marked ulcers opposite the mesentery; three of these in the last foot of the ileum perforated; no thickening about them. Similar ulcers in cæcum and colon. Intestines deeply stained with extravasated blood; fæcal contents also blood-stained. *Stomach* shows an ulcer near the pylorus, of the size of a sixpence, surrounded by traces of old extravasation of blood | None |
| Much œdema; systolic murmur at apex; hæmoptysis; fluid in pleura | 'Indigestion' for 3 years before death | Several small ulcers in *stomach*, with several cicatrices of healed ulcers. Surface of stomach congested and covered with mucus | None |

*in connection with Albuminuria.*

| | | | |
|---|---|---|---|
| Vomiting | None noted | Small intestine congested, and small patches of hæmorrhage in mucous membrane. No ulcers | None |
| Repeated epileptiform fits after a fall | None noted | Numerous hæmorrhagic patches throughout large and small intestines, and also on stomach | None |
| Acute œdema; suppression of urine latterly | None observed | Extensive ecchymoses in cæcum. No ulcers | None |
| No œdema; asthma; epistaxis. Died of dyspnœa | None observed | Numerous submucous hæmorrhages in ileum. No ulceration | Cretaceous tubercle in right lung |

| Name, physician, date | Age | Cause of renal disease | Duration of renal symptoms | Urine | Kidneys, P.M. | Heart, P.M. | Retinæ |
|---|---|---|---|---|---|---|---|
| William We by. Dr. Whipham, 1891 | 61 | — | 7 months | Nearly solid with albumen; scanty | Small, white, smooth; not lardaceous; weight 12 oz. | Hypertrophy of L.V.; weight 25 oz.; recent pericarditis | Advanced albuminuric degeneration; hæmorrhages |
| Richard Seymour. Dr. Dickinson, 1892 | 46 | — | 5 months | Albumen = ½; scanty | Granular, cysted; cortices shrunk; weight 12 oz. | L.V. hypertrophied; weight 30 oz.; recent pericarditis | Small hæmorrhage in left |
| John Read. Dr. Whipham, 1892 | 37 | Drink (?) | — | Albumen = ¼; pale | Small, red, granular, cysted; weight 6¼ oz. | Great hypertrophy of L.V.; weight 18 oz. | — |
| John Reardon. Dr. Dickinson, 1892 | 28 | — | 1 month | Albumen = ⅓; scanty | Tubal nephritis; congested; weight 13 oz. | Hypertrophy of L.V.; weight 16 oz. | Hæmorrhages and inflammatory effusion |

## *PLACES AND COMMONPLACES IN RENAL DISEASE.*[1]

I PROPOSE to bring together, perhaps in a somewhat unconnected manner, some of the impressions which have been left upon my mind as the results of experience in regard to renal disease, taking those especially which would seem, I will not say to have practical value, but at least to have a practical bearing. I shall introduce some considerations with regard to climate which may furnish useful guidance in the treatment of the granular kidney and the more chronic varieties of nephritis in persons who belong to what may be called the movable classes.

[1] *Lancet*, February 10, 1894.

| Renal symptoms | Bowel symptoms | Ulceration, P.M. | Disease bearing on ulceration |
|---|---|---|---|
| Œdema; bronchitis; dyspnœa; uræmic smell | None observed | Small submucous hæmorrhages throughout small intestine. A pigment mass in sigmoid flexure. No ulceration anywhere | Old vomica at apex |
| Œdema; arterial tension; dyspnœa | None observed | A few submucous hæmorrhages in ileum. Small pigmented area in ascending colon. No ulcers | None |
| No œdema; alcoholic paralysis | None noted | Several ecchymosed areas in ileum. No ulcers | None |
| Much dropsy; tense pulse; bronchitis; uræmia | None observed | Lower part of ileum and cæcum show numerous small submucous hæmorrhages. No ulceration or pigmentation | None |

First, as to acute nephritis or acute renal dropsy. The disease has a tendency to recovery qualified, especially when scarlatinal, by a tendency to fibrosis. To assist recovery warmth in bed and liquid diet are essential. The food should consist not only of milk, but should also comprise thin beef-tea or light animal broths. Water and aqueous drinks should be given freely; alcohol prohibited. Bread and butter may be generally permitted, and farinaceous food in some variety may be early introduced. After a calomel purge it will often suffice to give a little alkalising saline such as potassio-tartrate of soda (a great favourite with me) or tartrate or citrate of potash. These are laxative and mildly diuretic, and beneficially reduce the acidity of the urine, which in such cases is apt to be excessive. If anything more is needed to keep the bowels moderately loose, a morning potion of sulphate of magnesia should be

superadded. Digitalis ought to be given only if there is dropsy or if the urine is very scanty. With the exception of digitalis and such mild salines as have been mentioned, the whole class of diuretics may be avoided as either useless or injurious. Pre-eminent among the latter are cantharides. Though the urine may contain blood, it is seldom advisable to give medicines with the object of stopping the discharge, which depletes the kidney, generally to its advantage. If the loss is profuse and persistent, and there is much anæmia with it, some astringent form of iron, the sulphate or the perchloride, may be given after a time with an aperient, such as a little sulphate of magnesia, or in an extreme case iron alum in the same company. Should the cerebral indications of uræmia present themselves, they ought to be treated on principles to which I shall presently refer.

Next, with regard to chronic albuminuria, I will put aside for the present that of lardaceous origin and speak with regard only to the granular or fibrotic kidney. Many are the reminiscences which testify to the lengthy and almost indefinite quiescence of the disease. A man may suffer from it for a quarter of a century and be practically none the worse, though such cases are not common. The heart will be hypertrophied as a salutary adjustment, and the dropsy which so often marks the beginning of the end may be indefinitely postponed. For tens of years there may be no obtrusive symptoms or anything to prevent the patient, if he be one, from pursuing his usual way of life, presuming that this is one of ordinary moderation and care, and that he does not wholly ignore his disease. It may even be better for him to remain in ignorance of it than to be over-treated or over-restricted. The physiological agencies, diet and climate, first present themselves for consideration. As to diet, in a quiescent case I commonly advise temperance rather than austerity —one meat meal, one fish meal, and one meal without meat or fish. Farinaceous and vegetable food without restriction, milk without stint but without insistence,

watery drinks freely, and the less alcohol the better. I believe a purely milk diet is not generally advantageous. One of the hardest pulses I ever felt belonged to a man who conscientiously restricted himself to a milk diet and ultimately died from apoplexy. It is a matter of importance, and even of life, that in certain forms of renal insufficiency the free drinking of aqueous liquids should be permitted and even enjoined. When the urine is of low specific gravity and poor in quality it can provide sufficient exit for the urinary elements only when it is in large amount. It is a condition of life that quantity should make up for quality. The quantity is maintained by that of the water introduced as drink. In such a case as I am supposing the solid food should be light and the liquid abundant. I have seen fatal consequences follow the reversal of this rule in the treatment of the advanced granular kidney.

As long ago as 1867,[1] I took upon myself to advocate the employment of a warm climate in the treatment of chronic renal disease—a therapeutical measure which has since been widely resorted to. If it be said that the results are disappointing, this must be taken as implying no more than that too much is expected. No climate can turn fibrous tissue into tubes or a granular kidney into a healthy one; but the crippled organ may be relieved of work, the insufficient gland may be made sufficient, and the easily irritated structure may be saved from inflammatory attacks by the influence of some place where cold winds cease from troubling and the kidneys are at rest. The desiderata may be said to be warmth, uniformity of temperature, and dryness of air. I think that the first consideration should be the temperature, regulating as it does the action of the skin and of the kidneys. The mean temperature, as expressing broadly the general attitude of this overruling physiological influence, is perhaps the most important guide in the choice of a climate. The daily range, or variability within each twenty-four hours, is of obvious importance, but probably less so than is the daily mean temperature.

[1] *Pathology and Treatment of Albuminuria*, first edition.

A great daily range implies a corresponding fall in some part of the twenty-four hours, which must be of paramount importance to persons exposed. But seekers after health are not like shepherds who watch their flocks by night, nor need they be much out of doors after sunset. The low temperatures are at night, or at least after sunset, when the careful valetudinarian will not be fully exposed to them. It, is therefore, probable that the daily range is of less importance to a person who is his own master than either the mean temperature or the relative humidity. The latter, it is needless to observe, does not mean the absolute amount of moisture in the air, but its degree of saturation, or, in other words, its capacity for water, which varies with the temperature. Upon this depends the rate of evaporation, whether from the wet-bulb thermometer or from the skin and lungs. A low relative humidity encourages evaporation and vicariously relieves the kidneys. A human being is virtually a wet-bulb thermometer, by which instrument the relative humidity is ascertained. When the wet-bulb evaporates much, so do the skin and lungs. Hence it may be laid down that the chief atmospheric conditions to be desired in renal disease are a high mean temperature and a low relative humidity.

I here introduce a tabular statement, giving the three essential particulars for the cold months January, February, and March at the following health resorts. I fear they are not all equally trustworthy, nor do all relate to the same years; but the observations may serve as a guide where no better ones are available.

The table may be of assistance in the choice of a winter resort, but it must be fully allowed that many items have to be regarded which are not here expressed. Exposure to wind is one, sunshine and rainfall are others. Experience of the place is the best guide, and other things besides climate have to be considered. I have known an exacerbation of chronic nephritis to be attributed, and with apparent reason, to foul odours. We have to put one thing against another and make the best compromise possible. I have

*Winter Climates (Mean of January, February, and March). After London the Places are arranged according to Mean Temperature.*

| Place | Authority | Mean temperature | Mean daily range | Mean relative humidity |
|---|---|---|---|---|
| London (Norwood) . | Mr. Bayard, Meteorological Society (ten years) . . | 39·7° F. | 10·7° F. | 86 |
| Margate . | Mr. Bayard, Meteorological Society (ten years) . . | 39·9 | 9·1 | 87 |
| [1] Worthing . | Mr. Bayard, Meteorological Society (ten years) . . | 40·1 | 9·9 | 87 |
| [1] Brighton . | Mr. Bayard, Meteorological Society (ten years) . . | 40·6 | 10·2 | 83 |
| Bude . . | Reports of Meteorological Society (ten years) . . | 41·5 | 10·6 | — |
| Bournemouth | Dr. Compton, quoted by Dr. Dobell (eighteen years) . | 41·6 | 9·7 | 85 |
| Torquay . | Dr. Symes Thompson, Medical and Chirurgical Society (ten years) . . . | 41·6 | 9·9 | 83 |
| Ventnor . | Mr. Bayard, Meteorological Society (ten years) . . | 42·2 | 9·3 | 85 |
| Falmouth . | Royal Cornwall Polytechnic Society (ten years) . . | 42·9 | 7·1 | 83 |
| [1] Penzance . | Mr. W. Hosken Richards (ten years) . . . | 43·2 | 6·0 | — |
| [1] Ilfracombe | Mr. F. C. Bayard, Meteorological Society (ten years) . | 43·4(?) | 7·9 | 85 |
| Guernsey . | Mr. F. C. Bayard, Meteorological Society (ten years) . | 43·7 | 7·6 | 87 |
| [1] Scilly Islands | Mr. W. Thomas, Cornwall Polytechnic (ten years) . | 45·3 | 6·4 | 87 |
| Cannes . | Dr. Marcet, Meteorological Society (three years) . | 49·2 | 12·0 | 73 |
| San Remo . | Meteorological Observatory, quoted by Dr. Hassall (one year) . . . . | 50·0 | 13·0 | 71 |
| Algiers . | Dr. Tothill (nine years) . | 54·6 | 12·6 | 66 |
| Cairo . . | Mr. F. M. Sandwith (five years) | 57·8 | 17·5 | 64 |
| Alexandria . | Mr. F. M. Sandwith (nineteen years) . . . | 59·4 | 11·0 | 65 |
| Las Palmas (Canary) . | Mr. Morley Douglas (one year) . . . . | 61·8 | 11·2 | — |
| Madeira . | Dr. Grabham (ten years) . | 62·0 | 8·6 | 73 |
| Luxor . . | Mr. F. M. Sandwith (one year) | 62·0 | 31·2 | 49 |
| Calcutta . | Report of Sanitary Commissioner for 1893 (one year) | 69·7 | — | 66 |
| Maritzburg (Natal) . | Dr. Mann (one year) . . | 70·3 | 17·6 | 73 |
| Cape Town . | Dr. Mann (one year) . . | 72·4 | 15·9 | 67 |
| Bombay . | Annual Report of Sanitary Commissioner for 1893 (one year) . . . | 75·2 | — | 63 |
| Madras . | Annual Report of Sanitary Commissioner for 1893 (one year) . . . | 79·0 | — | 78 |

[1] In estimating the climatic differences between English places, which are necessarily small, it has to be borne in mind that something may occasionally be due to the placing of

sent many patients to the Riviera, and have on the whole been disappointed with the results. There the daily range of temperature is considerable, the evening fall is abrupt, and the difference between sunshine and shade is great. These drawbacks belong to other subtropical places, but at some of these there are greater compensations. As compared with the Riviera, the South of England has the advantage of greater uniformity and the disadvantage of greater humidity. I believe Algiers to be preferable to the Riviera and Egypt to be preferable to both, notwithstanding the great daily range of temperature. The special advantage of the Egyptian climate would appear to be best obtained at Luxor or some similar place. The great evening fall of temperature must of course be specially guarded against. Theoretically, Cape Colony should be an excellent winter resort in renal disease, the English winter being exchanged, after no very laborious journey, for the African summer; but some who have been there do not report favourably. Dr. Grabham, who has kindly supplied the figures in the table which relate to Madeira, tells me that he has there witnessed good results in cases both of post-scarlatinal nephritis and advanced Bright's disease. I have myself had no experience of Madeira as a resort in cases of renal disease, but the particulars of its climate testify to its suitability in regard to mean temperature and equability. Anyone interested in the climate of Madeira may refer to an article entitled 'The Island of Madeira' that is being published in 'The Lancet.'[1] I think that patients who cannot travel as far as Egypt or Algiers, or at least further south than the Riviera, may be content with a selected spot in the South of England. I have

[1] *The Lancet*, January 6, 13, 27, 1894.

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the instruments in an exceptionally advantageous position rather than in one which fairly represents the neighbourhood. It is possible that the high temperature records from Ilfracombe may have their explanation in some such accident. One may hesitate to accept as a general truth that Ilfracombe is warmer than either Falmouth or Penzance. Brighton, again, is represented as being slightly warmer than Worthing; common experience indicates a difference in the opposite direction. Finally, it is probable that as between Penzance and the Scilly Islands the mean daily range is greater at the former than it is at the latter, notwithstanding that the figures quoted present Penzance as having a slightly less range than the Scilly Islands.

known many patients to do well at Bournemouth and Torquay; Ventnor deserves more consideration in this respect than it has received; while Falmouth and Penzance promise better than any one of the three. As regards the last two places, it is to be regretted that at present there are no data enabling a comparison of their relative humidity, though, under the energetic influence of Mr. Silvanus Trevail, the County Council of Cornwall have originated observations which will supply the needed information.[1] I have an impression that Falmouth will be found to be less humid than Penzance; should it prove so it will be the better resort in renal disease, but on this point exact information is required. I have never found a patient who was willing to spend the winter in the Scilly Islands; the humidity of the air and the exposure to storms are objections, while social interests are wanting, though an artist or student of Celtic antiquities may employ his time pleasantly enough.

The importance of warm clothing is almost too obvious to mention. The skin and the kidneys alternate in function; when the skin is active the kidneys are at rest. A man may breathe the air of winter and carry the temperature of summer under his waistcoat, and provide himself, without leaving his home, with one of the advantages of travel. He should be cased in woollen from head to heel, and should wear a greatcoat which is worthy of the name.

Next as to treatment by medicines. The first necessity is to keep the bowels regular and loose, or at least free.

[1] Since this was written the County Council for Cornwall have established a number of Meteorological Stations which promise to make this county one of the best observed in England. From the reports which have been already issued I extract the needed particulars for the year 1894. Taking the averages for the three months, January, February, and March, as in the table, they are as follows:—Penzance—Mean temperature, 46·2; mean daily range, 8·7; relative humidity, 90. Falmouth—Mean temperature, 45·1; mean daily range, 9·0; relative humidity, 84. Thus, so far as the year 1894 may be taken as a sample, Falmouth presents a considerable advantage over Penzance in point of humidity, and should be the best renal resort in England from which we have definite meteorological observations.

Speaking now only of chronic and quiescent cases, a good practice is to give a ferruginous laxative on going to bed and on getting up, say, a drachm of aloes wine, a drachm of sulphate of magnesia, ten minims of the tincture of the perchloride of iron, and perhaps two or three minims of liquor strychniæ; or the decoction of aloes and tartrate of iron may be substituted for the wine and perchloride of iron, and sulphate of soda for sulphate of magnesia. The doses should be adjusted so that the bowels may act twice a day, or at least three times in two days. If the urine is over-acid a suitable mixture may be made of tartrate of iron with tartrate of potash, together with sulphate of potash or soda. Iron must be given with caution, and omitted if the patient is full-blooded, over-fed, or constipated; anæmia is, of course, an indication for it.

The normal termination of the granular kidney is by uræmia, though there are many pitfalls by the way. In order to obviate this tendency to a fatal issue sweating should be enforced where the tendency is declared by headache, vomiting, or otherwise. A Turkish bath at regular intervals, every ten days or fortnight, may long ward off what would otherwise happen. When from the distance of the bath or the state of the patient this is not available, the lamp bath should be used at home; and I may speak especially of a modification of it which for some years past I have been in the habit of employing. When a patient is in mortal peril one may hesitate to enforce so powerful a measure as the enclosure of the whole body in hot air: his hold of life may be feeble, and there may be a fear of disturbing it; but if the heated air is applied only to the feet or below the knees much good may be done, and, so far as I have seen, never any harm. One wicker arch only is used instead of several; the hot air thus limited will make the lower extremities perspire freely, while the whole body will do so to a less degree. I have found this partial bath—'the leg bath,' as I call it—to be invaluable. If this procedure does not cause the patient to perspire, as is sometimes the case, pilocarpine may be injected subcutaneously before the

bath is applied. Another preliminary measure which is useful in such cases is to place the patient for two or three minutes in a bath of very hot water, say, at 109° F., before the hot-air bath—which in such a case would, of course, involve the whole body—is applied.

As dropsy is one of the most prominent symptoms of albuminuria, I will say a word or two about its treatment. Nature's cure for renal dropsy is hypertrophy of the heart.[1] This may be presumed to act by way of the veins ; the increased suction power of the thickened left ventricle must draw upon these channels and so pump out the water-logged tissues, while the hypertrophy of the right ventricle, slight but real, must likewise help on the venous circulation. Renal dropsy, therefore, tends to recovery, but the process is slow and needs to be expedited. Œdema of the legs, whether renal or cardiac, is relieved by posture—not merely moved, but removed. Beyond this, measures of two kinds tend to benefit renal dropsy—those which lessen the contents of the vessels, and those which increase the force of the heart. Digitalis, I think, is almost invariably indicated ; it probably acts upon the heart and vessels in the first place, and upon the secretion of the kidneys only secondarily. I do not think that the way this drug acts in renal dropsy is entirely clear ; it is known to increase the contractile force of the left ventricle, but does it increase its expansile force ? It is not known that it does so. No doubt the increase in the contractile force of the right ventricle must tend to relieve dropsy, but beyond that one does not see very clearly. However, whatever obscurity there may be about the *modus operandi*, there is no uncertainty as to the beneficial result. Most reputed diuretics are useless, and some, like cantharides, are injurious. I have learned to distrust and avoid the whole class, with the reservation of digitalis and the vegetable salts of potash, which, in addition to their other properties, are slightly diuretic. Hydragogue purgatives and hot-air baths have their use, and, I may say also, their

[1] See paper on 'Renal Dropsy' in the *Medical and Chirurgical Transactions* for 1892, vol. lxxv.

abuse. Depletion may be carried too far and a condition of anæmia maintained by which dropsy is promoted rather than relieved. Exhausting measures ought, therefore, to be used with moderation and counterbalanced, according to circumstances, by food and iron. Among the purgatives which are useful in such cases the mercurials are not to be rigidly excluded, though they ought to be given seldom and in small doses. In considering the benefit to be derived from purgation and diaphoresis in renal dropsy, it is not to be forgotten that the initial causes of this form of the disease are probably the contamination of the blood and its altered relation to the capillaries, and the circumstance that evacuation tends to carry off the peccant materials. The abdomen may be tapped with safety, by which means not only it, but the legs will be relieved ; punctures of the legs by any process, whether with tubes or needles, should be avoided as far as possible, as being fraught with danger, however apparently trivial the operation.

Renal asthma, distressing and alarming as it is, admits of relief, and even of cure, by treating the uræmia in which it takes its rise. Ethers, alcohol, and nitrite of amyl are of temporary use ; but the mainstay is in the evacuants, hot-air baths, calomel, and the hydragogues. The pulse under these attacks often shows extreme over-tension, on lessening which they will abate or cease to recur.

With regard to the over-tension of the advanced granular kidney, a caution may not be out of place. We should not attempt to lower the pulse-tension to an ideal of health, as the result would probably be death ; the circulation is carried on under difficulties and only by means of increased force. The hypertrophy of the heart is a measure of salvation and the increased tension is a necessary condition. We should consent to a moderate increase of tension and seek to modify it only when it is excessive.

Mental disturbance is rare, but not unknown, in connection with the advanced granular kidney. Depression or melancholy may long precede death. More immediately foretelling the fatal issue are the lesser degrees of wander-

ing or delirium, and what I suppose may be called 'chronic mania,' insanity with delusions. I have learned to regard the latter conditions as of evil omen.

To bring to an end these trivialities of practice I will say a word or two as to the treatment of the lardaceous disease and the relation of the alkalies to the morbid deposit. There is much to suggest potash as remedial. Apart from syphilis, the only ascertained cause for the disorder is suppuration, a process which necessarily entails a loss of potash. The relation of the discharge to the deposit is so direct as to suggest some such arithmetic as this: take pus from blood, and the lardaceous matter remains. This matter is distinguished from the normal constituents of the body by its deficiency in potash and by its ready solubility in alkalies. The suggestion cannot fail to present itself that the substance is deposited in consequence of a deficiency in the blood of the normal alkaline solvent; but there are in the living body complications which the test-tube cannot reveal, and therapeutics based only on chemistry are apt to miss their mark. The loss of pus which causes the lardaceous deposition is not only a loss of potash but of the white corpuscles in which it is contained; the loss of the organisms cannot be ignored, nor can potash by itself replace them. Then, again, it has to be considered that, however readily the lardaceous material can be dissolved out of an organ by liquor potassæ after death, it is not quite so easy to get that reagent to bear upon the tissue during life. The material is dissolved only by the caustic alkalies, not by their salts, and the action of the blood upon these alkalies has to be considered. It is obvious that the blood cannot convey liquor potassæ as such. Nevertheless, I think it is wise, and I am sure it is harmless, to give a little liquor potassæ upon an empty stomach when suppuration is present; but general restorative treatment is what ought to be mainly relied upon—viz. a liberal diet and sea air, with iron, quinine, and cod-liver oil. I have seen more good result from a sojourn at Margate than at any other place. When the lardaceous condition is due to syphilis the benefit produced by the

long-continued use of iodide of potassium, possibly conjoined with iodide of iron, is remarkable. In order to obtain the greatest good from such medicines they should be given for years; I often propose two years as the duration of the course, and have many times continued it for longer than this with advantage.

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## *LECTURE ON THE CARDIO-VASCULAR CHANGES OF RENAL DISEASE, WITH SOME OBSERVATIONS ON THE LARGER ARTERIES.*[1]

Gentlemen,—Excepting what has to do with bacteria it may be said that no observations in pathology have been so fruitful in results as the great discovery of Bright. I do not now propose to deal with this as regards the kidneys themselves, but only to touch upon some of the systemic consequences of renal disease which Bright partly, I may say in large part, made known. Bright noticed the hypertrophy of the left ventricle, and attributed the muscular increase to abnormal resistance. He inferred that there was an obstacle to the outward course of the blood, and assigned the capillaries as its probable situation. The immediate cause he considered to be in the impurity of the blood, which in some way interfered with its transit through these channels, which expose to the blood so large a surface and maintain with it such intimate relations. This theory held its ground without modification or question until Dr. (now Sir George) Johnson observed the thickening of the muscular coat of the arterioles, and was led to the belief that the hindrance was not in the capillaries, as Bright had supposed, but in the minute arteries thus thickened. Johnson argued that the capillaries were not muscular, and therefore not contractile, while the arterioles were both. In this view the arterioles became hypertrophied in their efforts to keep the impure blood out of the

[1] Baillie Lecture. *Lancet*, July 20, 1895.

tissues; the heart became hypertrophied in its endeavours to overcome the resistance thus occasioned. The muscularity of the arterioles is not to be disputed, and it is known to physiologists that under certain influences, connected particularly with the nervous system, these vessels have the power of contracting, and so cutting off the blood from the tissues; but whether this stopcock action, as Johnson called it, is the essential factor in the cardio-vascular change of heart disease remains to be seen. The capillaries have been shown to be contractile, though not muscular, and the observations of Johnson do not disprove the theory of Bright.

Leaving this contest as yet undecided, I must note the appearance of another combatant, or rather hostile alliance, upon the field. Sir W. Gull and Dr. Sutton, neither of whom, I grieve to say, is with us now, came down in force upon the position of Johnson, and a great battle ensued, which was waged with obstinacy and even with asperity. The allies disputed the muscular thickening of the arterioles, and maintained that the only thickening was of the fibrous tissue. Johnson, in upholding the muscular increase, denied the fibrous, the appearance of which he held to be delusive and the result of reagents.[1] The contest was the more obstinate because there was right on both sides, and the more embittered because on both sides there was wrong. Without staying to recall the fortunes of the fight, I may say that I took then, and have taken since, some pains to satisfy myself of the actual condition of the arterioles, and will briefly repeat, what I said at an early stage of this controversy, that the muscular thickening as described by Johnson is a fact which does not admit of doubt. On the other hand, it is equally certain that with this there is fibroid overgrowth, however brought about. The champions of fibrosis did not attribute the vascular change to the renal or acknowledge any steps in the pathological process, but they regarded both as the parallel and simultaneous results of a baneful tendency which pervaded the whole body like

[1] Sir George Johnson now admits the fibroid thickening.

original sin, affected at the same time the vessels and the viscera, and produced a general deterioration, which they likened to the effects of age. This comprehensive theory is probably not entirely without foundation; there *is* such a thing as general fibrosis, particularly as a form of senile decay, and of this general fibrosis the granular kidney may sometimes be a part. But it is abundantly clear that this explanation suffices but for a small minority of the cases in which cardio-vascular changes are associated with renal. By far the greater number begin with disease local to the kidney, and proceed from step to step through the blood to the vessels and the heart. Inflammation limited to the kidney, produced by cold or scarlet fever, perhaps in a child where there can be no question of general fibrosis, except as a result of renal disease, is constantly and rapidly followed by the special cardio-vascular hypertrophy, and that in its most extreme and characteristic shape.

That the granular kidney is essentially a local matter and not a mere subdivision of a general fibrosis, as Gull and Sutton tried to make us believe, is borne out by what happens to the liver. Of all the organs in the body, the kidney and the liver are most liable to fibrotic change. If the kidney is affected thus merely as part of a general change, the liver surely ought to participate. But it does not do so. I found that in 250 cases of granular degeneration of the kidney, the liver displayed the corresponding change—that is, cirrhosis—in only 37 instances, a proportion of 1 in 7.[1] Granulation of the kidney and cirrhosis of the liver do not go together, as they should do if the fibrotic change in each were due to a common cause. The fact is that, putting aside the general effects of valvular disease of the heart, the liver and kidney are each influenced by causes proper to themselves, not common to the whole body. The liver is made fibrotic chiefly by alcohol, the kidney by climate, lead, gout, heredity, and as the sequel of acute nephritis. It has always seemed to me that the morbid independence of these organs is a strong

[1] *Albuminuria*, second edition, p. 171.

if not a conclusive argument against the theory of general fibrosis as propounded by Gull and Sutton. On every ground, therefore, we must accept that as a *general rule*—I do not say there are no exceptions, but I am sure they are few—the renal change is the initial mischief, the *fons et origo* of which the cardio-vascular changes are the results.

The only question that presents itself as admitting of doubt is by what process the renal change brings about the cardio-vascular. The impurity of the blood due to the imperfect action of the kidneys may be accepted as an intermediary, but how does it produce the effects in question? There is presumably an obstruction which gives rise to increased blood pressure, and this to hypertrophy of both muscle and fibre. But where is the obstruction? Is it in the capillaries, as Bright supposed, or are we to place it with Johnson in the arterioles? It seems unlikely at first sight that the heart and the arteries should become hypertrophied by acting against each other—it looks like a want of the common consent which we usually find in nature; but how shall I say it is not so? There are many things which are too wonderful for us, four of which have been mentioned by a royal and inspired author. The action of the heart and vessels may be, like these, beyond our comprehension; but it is not beyond our inquiry. I have ever found, when a difficulty presented itself, that the best way was not to argue or even to reflect, but to seek for additional facts. Being able to get them is the great advantage we have over our brethren the theologians. The facts at their command cannot be added to by observation, whatever talent may be expended in reconsidering the stock in hand. With us the main endeavour is to collect fresh particulars, and nothing further is necessary than to let them speak for themselves. Unlike the students of theology, we are able to push our inquiries beyond death, and take for our motto, 'Nec silet mors.'

As possibly throwing light upon the rival theories of obstruction in the capillaries and in the arterioles, I thought it worth while to look more widely than had hitherto been

done at the distribution of the arterial hypertrophy. If this should prove to be limited to the smaller vessels, which alone can be credited with the stopcock action, then the stopcock or inhibitive action may be the cause of the hypertrophy, or of as much of it as concerns the muscular coat; if, on the other hand, the large arteries should be found to partake of the same alteration, then we must look for some cause which acts alike on large and small. With this view I have examined a series of arteries, including some of the largest: the aorta, the innominate, the common femoral, and the renal. Sections of these vessels made carefully in the same respective situations were placed under the microscope and outlined on paper with the camera lucida and a uniform magnifying power of 6·35 diameters. The tracings thus obtained were then measured both as to the thickness of the wall and the circumference of the vessel. The number of vessels thus treated amounted to 139, which were supplied by forty-nine individuals mostly chosen as the subjects of marked renal disease or as presenting the type of health. The conclusions are based upon the whole number of vessels of each kind and not derived from selected specimens. As a standard of health I have taken eighteen post-mortems, generally after accident or acute disease—ten of males and eight of females. In the male series I have added two cases of acute nephritis, in which, as there was no time for, nor any evidence of, cardio-vascular change, the vessels may be taken as healthy and as furnishing a second standard of comparison. The granular kidney is represented by sixteen male subjects and seven female. In the latter series I have thrown together, as exhibiting virtually the same condition, and that the fibrotic kidneys described as *granular* and as *white contracting*. The male series shows that with the granular kidney the aorta is thicker than in health as 42 to 39, and thicker than with acute nephritis as 42 to 31. The innominate with the granular kidney gives a thickness of 0·41 in., that with health 0·35 in., and that of acute nephritis 0·30 in. Here I am speaking of

course of the vessels magnified as I have stated. The common femoral gives with the granular kidney a thickness of 0·33 in., with health that of 0·26 in., and with acute nephritis that of 0·24 in. The renal gives with the granular kidney a thickness of 0·25 in. in health, and with acute nephritis one of 0·18 in. Both coats are thickened, as will be seen on reference to the accompanying table. The muscular shows the change with more exactness than the fibroid, for it is not exposed in removal, or liable, as the outer coat is, to have any part left behind. The circumference of the vessels of each kind is also increased with the granular condition, that of the aorta giving $13\frac{3}{4}$ in., as compared with $11\frac{3}{4}$ in. in health and $10\frac{3}{4}$ in. with acute nephritis. The innominate, femoral, and renal showed similar increase under the same circumstances, as is displayed in the table. The comparison, as far as relates to the female sex, will be seen to give similar evidence. The aorta, the innominate, and the femoral are all increased under the renal change in question in the total thickness of the wall, in the thickness of the muscular coat, and in circumference. The only exception is in the case of the renal artery in the female, which presents an increase of circumference, but none in the thickness of the wall; only eight specimens, however, of this vessel in this sex were examined, and the number is too small to give weight to the exception.

I have added as collateral illustrations two instances which show the absence of arterial thickening, as of cardiac hypertrophy, with the large white and the lardaceous kidney; and a third which presents a large amount of aortic thickening in connection with atheroma without renal disease. This aorta, which displayed a large endo-arterial deposit, gave a total thickness of 0·57 in., which was greater than those in any of the renal cases, the thickest aorta with renal disease measuring 0·55 in. With the aorta thus thickened by atheroma the change did not extend to the innominate, which was thinner than the

*Thickness and Circumference of Arteries (Magnified 6·35 Diameters). Mean Measurement in Inches.*

*Male Adults.*

| State of kidney | Number of cases observed | Aorta | | | Innominate | | | Common femoral | | | Renal | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Total thickness | Muscular thickness | Circumference | Total thickness | Muscular thickness | Circumference | Total thickness | Muscular thickness | Circumference | Total thickness | Muscular thickness | Circumference |
| Healthy | 10 | 0·39 | 0·29 | 11¾ | 0·35 | 0·27 | 8⅝ | 0·26 | 0·13 | 4¼ | 0·18 | 0·10 | 3 |
| Acute nephritis; no cardio-vascular changes | 2 | 0·31 | 0·26 | 10¾ | 0·30 | 0·22 | 7¼ | 0·24 | 0·14 | 4⅛ | 0·18 | 0·08 | 4 |
| Large white kidney | 1 | 0·35 | 0·25 | 12 | 0·40 | 0·30 | 8 | 0·30 | 0·20 | 5 | — | — | — |
| Granular | 16 | 0·42 | 0·31 | 13¾ | 0·41 | 0·29 | 8¾ | 0·33 | 0·21 | 6¼ | 0·25 | 0·14 | 3½ |
| Lardaceous kidney | 1 | 0·30 | 0·27 | 12 | — | — | — | 0·35 | 0·25 | 5½ | — | — | — |
| Highly atheromatous aorta | 1 | 0·57 | 0·38 | 15½ | 0·30 | 0·23 | 6½ | — | — | — | — | — | — |
| *Female Adults.* | | | | | | | | | | | | | |
| Healthy | 8 | 0·34 | 0·26 | 11 | 0·26 | 0·24 | 7 | 0·24 | 0·15 | 4½ | 0·21 | 0·13 | — |
| Granular or white contracting kidney | 7 | 0·38 | 0·30 | 12½ | 0·36 | 0·27 | 9 | 0·29 | 0·16 | 5 | 0·19 | 0·11 | 3½ |

average of health.[1] The above measurements will, of course, be understood to apply not to the actual dimensions of the vessels, but to those of the representations uniformly magnified as stated.

It has been well known since the observation of Sir G. Johnson—which represents a great truth, however it is to be interpreted—that with the chronic granular kidney the arterioles, even to the smallest, become hypertrophied. For the purpose of this inquiry I thought it would be of interest to ascertain as far as practicable how the degree and kind of thickening here compared with that of the larger vessels. To this end I have examined afresh a number of minute arteries belonging to the pia mater and median fissure of the brain, which are in my possession as the results of former work. Some have been preserved in Canada balsam, others in glycerine. I have dealt with these specimens graphically, making outlines of them with the camera lucida, as in the case of the larger vessels. I may say that I have made no selection—by judicious selection it is possible to prove anything—but I have taken all that came to hand as either typically healthy or connected with typical granular kidneys. The results are before you. The outlines show that under renal disease there is generally, but not universally, thickening of the wall, and that this affects both the muscular and the fibrous coats. The total thickening, and that of each coat, is, judging by the eye, generally greater in the small vessels than in the larger, though demonstrably present in both.

[1] I may introduce a word as to the methods employed. Sections of the arteries were cut with the microtome and mounted on slides for the microscope. These were reflected on paper with the camera lucida and carefully outlined, all with exactly the same microscopic and optical arrangements. With the magnifying power used the vessels were presented as large enough to be accurately measured. The thickness of the wall was ascertained with a rule graduated to hundredths of an inch. The circumference, taken along the outer edge of the muscular coat, was measured with a rotating instrument such as is used for estimating distances on maps. This gave the circumference in inches and fractions of an inch. The enlarged figures to which I have referred were displayed in the course of the lectures. I believe the measurements may be accepted as accurate.

Putting all the facts together, and including what is common knowledge as to the left ventricle, we may formulate the general statement that in connection with the chronic granular kidney we have hypertrophy of the muscle and of the fibrous tissue belonging to the whole arterial system connected with the left side of the heart, and of the muscle of the heart itself. It is probable that had the inquiry been carried into the pulmonary vessels similar changes might have been found there. We see indications of them in the hypertrophy of the right ventricle, which in these circumstances is almost invariably associated with that of the left, though on a smaller scale. Looking at the import of the changes which have been recorded, we recognise, first, the general application of the ancient dictum that muscle becomes hypertrophied when resistance is opposed to its contraction. We next see the influence of another law, which may act together with, or independently of, the first—namely, that tissues, whether epithelial, fibrous, or muscular, become over-nourished and overgrown as the result of habitual congestion or over-pressure, or undue retention, of blood within them. As regards renal disease, it is impossible to separate the question of obstruction ahead from that of over-pressure by their contents upon the arterial walls. Some of the forms of renal disease are attended from their outset with increased blood pressure, others are not. When the pressure is increased, then, sooner or later, there is hypertrophy; when it is not increased there is none. This state of pressure or tension precedes the hypertrophy, for it is evident to the finger, the sphygmograph, and the stethoscope long before there has been time for any structural change. The granulating kidney and the lardaceous may be instructively contrasted. The granulating kidney—beginning, say, in nephritis—is attended from the very first with increased tension and followed as surely by hypertrophy. The lardaceous condition begins with no increase of tension or even with a lack of it; the heart remains small to the last, or, at least, until other renal changes are superadded. It is clear

that there is a necessary connection between the over-tension and the hypertrophy; and as the cause precedes the effect, it must be supposed either that the tension causes the hypertrophy, or else that both are the results of some common cause.

Searching for the immediate cause of the increase of tension and the hypertrophy which follows upon it, we can only attribute one and both to a difficulty in the emptying of the arterial system caused by an obstruction at or near its outlet, due to the morbid condition of the blood. So far all seems clear. The increased retention of blood within the arterial system is an adequate cause for vascular and cardiac thickening, on the principle which has been already adverted to, by which congestion causes hypertrophy. This would seem to be the essential and simple cause of the fibroid thickening. With regard to the muscular there are other considerations. The arteries are alternately filled by the ventricle and emptied by their own contractility. Peripheral resistance will oppose their contraction and thus occasion muscular hypertrophy. With the large arteries this rule seems of obvious application; they are in the same case as the heart and owe their hypertrophy to similar means. With regard to the minute and terminal arteries, other modes of action are to be taken into view. These vessels, which are known under certain circumstances to exert a special contractile power and thus cut off the blood from the capillaries, have been supposed to do this in renal disease and to owe their hypertrophy to their efforts not to accelerate, but to retard the current. I will not argue for this view or against it, but content myself with stating the facts, or what appear to me to be such, which bear upon it. I think it must be allowed that the smaller vessels are generally more thickened than the larger—at least, I have seen some examples of extravagant thickening among the smaller which I have failed to find in the larger vessels.

It often appears that the calibre of the small vessels is lessened in comparison with the thickness of the walls,

whereas with the larger there is dilatation as well as parietal thickening. It looks as if the smaller vessels had some special cause of hypertrophy beyond that which affects the larger. There may be a special inhibitory mandate addressed to their muscular tissue, but against this we have to place the fact that the fibrous tissue, to which no such mandate could be held to apply, is at least equally thickened. Whatever be the cause of the hypertrophy, it must relate equally to both coats.

In searching for the primary seat of obstruction, however our attention may be arrested by the arterioles, we must not disregard the capillaries. These have been proved to be contractile, though they are not muscular.[1] These vessels are in more intimate relation to the blood than any others, and may be supposed to be the first to be influenced by its abnormal conditions; and as evidence that they are so influenced we have the appearance of dropsy—or, in other words, of abnormal capillary transudation—at the outset of renal disease. This process is capillary and not arterial, and at least points to a morbid condition of which the capillaries, not the arterioles, are the seat. Arterial obstruction, indeed, must be prohibitive of dropsy by cutting off the blood from the vessels which are its source. These considerations appear to indicate that, whatever be part of the terminal arteries, the capillary system is the point of departure of the series of changes which have been described. Whether these terminal arteries have any special sympathy with the capillaries with which they are so immediately connected is a question which suggests itself. Whatever be the explanation of the vascular changes upon which I have dwelt, it must include the fibrous thickening as well as the muscular.

Without attempting to pursue these questions to the end I will content myself for the present with having demonstrated the fact that under renal disease, putting aside the

[1] Paper by Dr. Roy and Dr. Graham Brown on 'The Blood-pressure and its Variations in the Arterioles, Capillaries, and smaller veins.' (*Journal of Physiology*, 1879-80, Part 2, p. 323.)

pulmonary department to which the same rule probably applies, there occurs a hypertrophy of the cardio-arterial system which is universal from its origin to its termination, and comprises not only the ventricle and the arterioles, but affects also the intermediate arteries of every size.

In conclusion, I have to thank Dr. Rolleston, Dr. Lee Dickinson, and Dr. Cyril Ogle for procuring the specimens of which I have made use. My especial thanks are due to Dr. Rolleston—and to a larger extent to Mr. Fenton—for cutting and mounting the sections.

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## *THE PRACTICE OF MEDICINE AT ST. GEORGE'S HOSPITAL FORTY YEARS AGO.*[1]

It was my good fortune, between the years 1852 and 1854, to serve as clinical clerk under Drs. Wilson, Page, Bence Jones and Pitman respectively, added to which, Dr. Wilson and Dr. Page, on their retirement from the Hospital, presented to me, unasked, their entire collections of clinical books. The notes were then kept in books, not on detached sheets as at present. With these at my command, together with the post-mortem books, I will try to present the practice of the time, avoiding, as a rule, any personal reference to the practitioner. As regards one series only, which belongs to an earlier period than that of which I shall chiefly speak, shall I identify the physician.

Among the books which I received from Dr. Wilson is one which records the practice of 'the great Thomas Young,' who was physician to the Hospital from 1811 to 1829. The volume, which relates to the years 1828 and 1829, was passed on to Dr. Wilson, together with Dr. Young's hospital patients, on Dr. Wilson's appointment. This book appears to me to have especial interest, as exhibiting the state of medical practice in the early part of the century, and as exercised by the most comprehensive genius and greatest man of science who ever held the office

[1] *St. George's Hospital Gazette.* May and June. 1893.

of physician to St. George's, or, indeed, to any other hospital. Dr. Young's notes comprise those of 145 cases. They are ample and minute in treatment, but without any clinical particulars excepting an occasional word of diagnosis, and here and there a brief note of the post-mortem appearances. In the list of diagnoses we miss some which in the present day we are but too familiar with. There is no mention of albuminuria or of any renal disease excepting 'nephritis calculosa'; only one of disease of the heart, in the terms 'pleurisy or carditis.' Strange to say, there is no reference to anasarca or any kind of dropsy excepting ascites; there are frequent notes of fever with ulceration of the bowels (the typhoid of to-day); some of pneumonia, pleurisy, hydrothorax, phthisis, rheumatism, scarlatina, erysipelas, chlorosis, and ague—the last with some frequency. There appears to be no reason to think that the diseases in this Hospital then were very different either in kind or character from what we have now; though, as there were fewer means then of tracing disorders to their organic origins, they were named more superficially. I find that of the 145 patients, 18 were bled and 11 cupped, besides which many were leeched. Thus, every eighth person suffered phlebotomy, every eleventh cupping, but the amounts were not enormous. The average of venesection was ten ounces, of cupping eight ounces. The most striking feature in Dr. Young's practice was his constant use of blisters. Of the 145 patients of which I have records, 89 were blistered. The total number of blisters employed was 158, so that Dr. Young used more blisters than he had patients. The largest number applied to any one patient was eight. This man, apparently suffering from rheumatism, had one to the sternum, one to the nape, one to the temple, two to the head, one to the hip, and two to the heel. I dare say some may have done good, especially if locally applied, for local rheumatism—a practice which has since been largely resorted to. But Dr. Young appears to have used blisters somewhat indiscriminately, applying one over any part which was thought to be diseased, and often one

to the nape of the neck—I suppose on some general theory of derivation. Sometimes the blisters were kept constantly open, and Dr. Young frequently employed a plaster under the name of Emplastrum Carui. This, I presume, is what was called Emplastrum Cumini, which contained cumin, caraway, and resin, and probably had irritating properties. Dr. Young evidently had great faith in counter-irritation. In this respect he may have caused some unnecessary discomfort, but probably did little other harm. He gave small quantities of wine not infrequently and cinchona frequently. Of the 145 cases, 51, or more than a third, had this bark at some time or other, besides a few who took quinine.

It may be of interest to look at Dr. Young's practice in regard to one or two individual diseases, selecting some which can be clearly identified. One of these is 'Fever'—certainly in most cases typhoid, though not so named.

Nine cases of this kind are recorded. Seven were 'cured,' two died, in both of which the small bowel was found to be ulcerated. The treatment comprised general and local blood-letting, blistering, calomel and antimony, cinchona and wine. One was bled to 10 ounces; one was cupped on the nape to 12 ounces; one had 12 leeches to the head; another, 32 leeches in three instalments, of which 20 were to the head. Three patients were blistered, two upon the nape. Six had calomel and antimony, one calomel without antimony, and one antimony without calomel. The calomel was usually given every four hours in doses which varied from half-a-grain to two grains; the antimony, as James' powder, as often, in doses of from two to five grains. Cinchona was given in five cases; wine in two.

A fatal case of pleurisy was treated with leeches and blistering, and the administration every four hours of James' powder, Dover's powder and calomel, the last only in half-grain doses. One, not fatal, had only antimony and ipecacuanha. A case described as pneumonia was first blistered, then treated with haustus salinus, squills, ammonia and wine.

In the whole of Dr. Young's practice of which I have record there is no instance of the internal use of iodide of potassium. In two cases tincture of iodine was given in doses respectively of eight and six minims; in one case iodine ointment was used, and in another an ointment of iodide of potassium.

Iodine was discovered as far back as 1812. Iodide of potassium had become known as a sorbifacient, and had even been credited with the power of absorbing the mammæ and testicles, but whether from neglect or distrust, this remedy found no place in Dr. Young's pharmacopœia. We can scarcely realise the exercise of medicine in this sinful world without it.

We discern in Dr. Young's practice much moderate blood-letting, much counter-irritation, a general but not excessive use of mercury and antimony in most febrile and inflammatory affections, a liberal administration of bark, and occasional recourse to wine. Dr. Young's methods were more lenient, less exhausting and more supporting than much which came after; and I can scarcely doubt that they gave better results. I have been told that he was regarded by some of his contemporaries as a feeble practitioner, and described by the students as 'a great philosopher, but a bad physician.' But the Apothecary of the Hospital, who in those days had a subordinate charge of all the medical cases, appears to have thought differently; for it is recorded as his experience that a greater proportion of Dr. Young's patients were discharged 'cured' than of those who were subjected to the more energetic treatment of his colleagues. And Dr. Wilson speaks of his predecessor in terms which suffice to show that both these learned physicians were in advance of their age. Dr. Wilson writes thus in 1845:—'For many years past, by a system of mock-energy in the treatment of disease, reckless in its means because opposed to reflection, and pretending to facts from the absence of principles, the study of physic has been discouraged in this country, and its practice degraded'; and subsequently, looking back upon the

practice of Dr. Young, and his want of popularity as a physician, his successor thus explains the position of the elder philosopher:—'He lived in an age when what is called *vigorous practice* was very generally prevalent; when the use of calomel and the lancet was in the ascendent; when symptoms were rudely interfered with and combated without any proper study of the causes in which they originated.' Dr. Young had the caution of a philosopher, Dr. Wilson the temper of a sceptic. The public asked neither for philosophy nor scepticism, but like Christian and Hopeful, preferred to follow Mr. Vain Confidence, too often, like them, to find themselves in the clutches of Giant Despair.

I will now put together some particulars of treatment belonging to the four years from 1851 to 1854—roughly speaking, forty years ago. My aim will be to present not so much the practice of individual physicians as the fashion of the time in the treatment of particular diseases.

To take first, 'Fever.' This was mostly typhoid at the time of which I speak, and was then beginning to be described by that name. Typhus was less frequent, but nevertheless was well known in the wards. To take typhus first, two fatal cases were treated with haustus ammoniæ acetatis every six hours, and mercury—in one, two grains of grey powder, in the other, three grains of calomel—three times a day. In one case a little wine was given towards the end, in the other none.

Of the treatment of typhoid I could adduce numerous examples. The same 'antiphlogistic' principles were carried out mainly by means of salines, mercury and antimony. Haustus potassæ tartratis with perhaps twenty minims of antimonial wine, every six hours, was common, sometimes varied by the omission of the antimony and substitution of citrate or acetate of ammonia for the potash-salt. It was usual to give also mercury, sometimes two or three grains of grey powder three times a day, sometimes either Dover's powder or calomel and opium at

night only. Wine was for the most part withheld or given but sparingly, or only at last.

Dr. Wilson's practice was somewhat exceptional in his usual avoidance of mercurials in typhoid. An ammoniated saline with a little chloric ether was his ordinary prescription, with perhaps two ounces of wine and 'Fever diet.' Whey and Wenham Lake ice comprised the rest of his dietary. 'Fever diet' was emphatically a low diet, consisting only of a pint of tea morning and night, with a quarter of a pint of milk, twelve ounces of bread daily, and barley water *ad libitum*. The diet employed by the other physicians was not very different. In the treatment of fever at this time we see the same belief in mercury and salines which ruled practice in all febrile and inflammatory diseases. With our present notions we can but regard the mercury, at least, as wholly injurious in fevers, which, like typhoid, kill by depression, and believe that the results would have been better had no medicine been given. A contrary system was introduced at this time, experimentally used, and soon abandoned. Large doses of quinine were given in some cases of typhoid—in one case 20 grains every six hours; in another, 10 grains every two hours. Both were fatal, and this method of treatment was discontinued.

Especial interest attaches to the treatment of pneumonia, in consequence of the violent oscillations of medical opinion in this matter. At the time of which I speak there was little difference of opinion, at least at St. George's Hospital. Bleeding, calomel, antimony, blisters, and starvation were the means employed with more or less severity by all. To give a few instances:—Pneumonia of the lower lobe of the right lung was recognised in a previously healthy man of 27. He was at once bled to 12 ounces, next day to 8 ounces, and on the third day 16 leeches were applied to the chest. In the meanwhile, he had every six hours two grains of calomel, a quarter of a grain of antimony, and a quarter of a grain of opium, which combination was continued to salivation. Erysipe-

latous inflammation then presented itself in both arms, starting from the punctures, and he died, says the medical registrar, 'debilitated by his illness and loss of blood.' Post-mortem there was found partial consolidation of the lower lobe of the right lung. Had he been left alone he might have been alive now. A girl of 18 was admitted with pneumonia. She was in a condition of great depression, faint, and 'evidently unable to bear active treatment'; the pulse was weak and rapid. Wine was ordered by the apothecary. On the advent of the physician there was 'much hesitation as to the propriety of bleeding,' but the breathing was greatly oppressed, and 'a small bleeding was practised with great care'; a large blister was placed upon the chest, and calomel and opium were given every three hours. She died next day with hepatisation of the right lung. Perhaps she would have died in any case; but if her tendency to death was, as it seemed, by exhaustion, the treatment could not have done much to obviate it. The treatment was conducted by a physician of great ability, and evidently with the most anxious consideration. This case displays in a remarkable manner the faith of the time in blood-letting and mercury. The physician fully recognised the constitutional indications against them, but he dared not in any circumstances withhold remedies which he held to be so essential. *Now* we should have had regard only to the general condition.

To present the treatment fairly I must give that of successful as well as of unsuccessful cases. A man who recovered of pneumonia under Dr. Wilson was bled in two consecutive days to a total of 22 ounces, was blistered, and took every four hours a draught of tartrate of potash with a drachm of antimonial wine. Dr. Page's practice was not very different. A man was admitted with pneumonia, having had shivering one week before, which probably marked its beginning. He was cupped to 12 ounces, had 'fever diet' without wine, and a pill containing three grains of calomel, a third of a grain of antimony, and a quarter of a grain of opium, three times a day. He was

made out-patient on the seventh day after his admission. Another, who did well, was cupped to 10 ounces, and dosed every six hours with three grains of calomel and one of opium. He was discharged convalescent fourteen days after admission. In the light of many such cases similarly treated with a successful result, I have sometimes asked myself whether the modern practice in pneumonia is wholly right and the old treatment wholly wrong.

No doubt our progenitors often, perhaps generally, pushed their 'antiphlogistic' method too far, and had too little regard to the general condition. But was there no truth in their theory, however extravagant their practice? Under the influence chiefly of Todd, pneumonic patients have since been drenched with alcohol, as if this were a specific for the disease instead of sometimes its cause. But may not the dispassionate mediciner look on both sides of the question, and say here I perceive a divided duty?

The vascular system of the lung is in a condition certainly of leakage into the air-cells, and presumably of over-pressure in the vessels; the general powers of the system are apt to fail under the process, so that there is a tendency to death by asthenia. We must have an eye to both these opposed but associated conditions. The morbid products in the air-cells have come out of the blood-vessels, and have to go back into them. They are got rid of more by absorption than expectoration. If we lessen the pressure within the pulmonary vessels we presumably lessen the exudation from them, and facilitate its return into them. Thus depleting drugs or depleting measures may, and even *must*, benefit the local state of the lung. But the pulmonary disorder is attended with general asthenia and failure of vital power, which is the chief immediate danger and the usual mode of death. If we deplete or depress too much, we promote this fatal tendency; and no doubt our predecessors often did so. If we deplete too little or charge the vessels too much, as perhaps is sometimes done now, we prolong the morbid process or retard that of recovery; allow matters to remain in the lung which might otherwise

be got rid of; and, by protracting the disease, invite the occurrence of secondary changes.

Of late I have reverted with caution and abatement to parts of the old practice in pneumonia—and I think with advantage. I have always been accustomed to begin the treatment of this disease with a mercurial purge; I now carry on the mercurial afterwards, with a grain of calomel or blue pill two or three times a day. I give no opium, for which I see no indication. Without it the small mercurials keep the bowels loose, which cannot but be an advantage when there is so much morbid material to be got rid of, and it may be fairly presumed that they lessen exudation and promote absorption. As to diet and stimulants, I endeavour to keep a rational course, free from the falsehood of extremes, taking for my daily guide the constitutional state of the patient. Many do well without alcohol; some require a good deal. My only experience of the 'fever diet' is what I have gathered from witnessing the practice of my predecessors; my own rule of food has always been somewhat less parsimonious.

To look next at Acute Rheumatism, and first at fatal cases; a man of 45 had it in a severe form as regards the joints, the heart escaping. He was afterwards found to have granular kidneys. On the day of admission, April the 7th, he was bled to 14 ounces; on the 8th, again to the same amount; to 12 ounces on the 10th. On the 13th he became subject to epistaxis, which recurred frequently and with severity until his death. On the 21st he had some confusion of intellect, and was cupped on the back of the neck. His medicines were at first five grains of calomel with one of opium three times a day, which were afterwards commuted for frequent five-grain doses of calomel followed by haustus sennæ. He died on May the 4th, which he surely would not have done if he could have been saved by depletion.

A man, aged 45, was admitted with acute rheumatism and pericarditis. He had a draught of tartrate of potash

with a quarter of a grain of antimony every four hours, and calomel and opium three times a day to salivation. Fluid was then discerned in the abdomen, which was accordingly rubbed with blue ointment, and a pill was given containing mercury, squills, and digitalis. He sank, with diarrhœa.

Dr. Wilson employed blood-letting and mercury in the treatment of rheumatic fever, often with the addition of alkalies and colchicum. I may adduce the following among many instances. A case at first without heart mischief, after a purge of calomel, opium, and senna, was ordered haustus potassæ citratis with a scruple of bicarbonate of potash, and 15 minims of colchicum wine every six hours. Three days afterwards, on the occurrence of pericardial grating, there were ordered in addition three grains of calomel and two-thirds of a grain of opium every six hours, and these continued to salivation. The epigastrium was twice leeched. The patient recovered. A man with rheumatic fever, pain in the left side and difficulty of breathing, was at once bled to 12 ounces, and the operation repeated in the evening to the same amount. Two and a half grains of calomel and two-thirds of a grain of opium were given every six hours, together with haustus potassæ citratis and 40 minims of antimonial wine. Next day the patient displayed pericarditis, and ultimately recovered. I need not multiply examples to show the general belief which at this time prevailed in the uses of blood-letting, mercury, and opium in the treatment of rheumatic fever. The general impression which I have derived in looking through a large number of cases is of the frequency of pericarditis among them. For particulars with regard to the frequency of heart affections under the treatment which was employed for acute rheumatism at the time of which I speak and later, I may refer to p. 236.

To show the belief in counter-irritation which still prevailed, I will relate a single instance:—A woman with chronic albuminuria and many complications, was blistered for each of them. For broncho-pneumonia, two blisters

were applied to the sternum; for pericarditis, one to the precordium—and the sore afterwards dressed with savine ointment; a blister was applied to the epigastrium, to relieve vomiting; and one to the abdomen, in consequence of pain and tenderness, which were traced ultimately to enteritis, now recognised as among the albuminuric sequelæ.

The trust in mercury in almost every affection of the liver was frequently displayed. A man had jaundice associated with a large round hepatic tumour, which proved to be a hydatid cyst. He had five grains of blue pill three times a day, to salivation, together with a draught containing nitre, nitric ether, nitric acid, and taraxacum. I suppose now the aspirator would have been used.

To give our predecessors their due, I may mention a disorder in which their practice was more consistent with the latest views, and probably more beneficial than that which a short time ago was generally employed. They, with one consent, treated hæmoptysis with small repeated doses of sulphate of magnesia in infusion of roses. The infusion of roses was a survival of the mediæval doctrine of signatures. The sulphate of magnesia is in conformity with the latest considerations with regard to arterial tension and the usage based upon them. Judging by recorded experience, the early practice was far more successful than the ergot, by which it was superseded.

The tendency which prevailed in the middle of this century to cast blame upon the liver, and the belief that mercury would put it right, if anything could, were emphatically declared in the treatment of cholera. I refer to the epidemic of 1854. The process of purging to death, which is the way of the disease, very soon empties the liver and bowels of bile, so that the evacuations become bileless and colourless. This appears to have suggested the liver as primarily concerned in the disorder, and as a necessary consequence, calomel as the remedy. At the beginning of the epidemic, until experience had demonstrated its futility, this drug was generally employed,

and with an energy which was commensurate with the severity and rapidity of the disease. The patient, commonly blue and cold, was first comforted with a mustard emetic; he then took calomel, usually in scruple doses and at short intervals; he drank eagerly and vomited abundantly, bringing up drink and medicine together with the characteristic rice-water vomit. Thus it is to be hoped that much of the calomel was got rid of without injury. That some was is certain. I cannot forbear relating an experiment which was not perhaps in the best taste, but which was conclusive as to the calomel. Much of this was vomited scarcely changed, and, in one instance, was collected and subsequently converted into black-wash, and used as such—rather an unclean application of the *reductio ad absurdum*.

To adduce a few illustrations:—A patient in the blue stage had three scruples of calomel in the course of an hour—exactly a grain a minute—then a scruple every hour for six hours. Another, whose hands and feet were blue to blackness, had a scruple of calomel every half-hour from two o'clock until ten, after which, strange to say, he appeared to be somewhat better, and was allowed a respite, after which the drug was resumed at the rate of 10 grains every two hours. Ten grains of calomel every hour was a not uncommon mode of administration. In one instance, the same drug was given every ten minutes, but I am unable to state the dose. With the calomel was often given a draught containing chlorate of potash and carbonate of soda. Perhaps the chlorate of potash was meant to impart oxygen and relieve the cyanosis. Sometimes the attendant draught contained sulphate of magnesia and antimony. Why the antimony, I cannot guess. The sulphate of magnesia may have been intended as an eliminant, though the cholera poison is an eliminant which would seem to require little assistance; it eliminates itself and the patient too. Many other drugs were tried, among which opium, acetate of lead and sulphuric acid may be mentioned. In one instance half-an-ounce of the dilute acid was injected into the bowel.

The foregoing experiences are all taken from the post-mortem book, and therefore necessarily relate to fatal cases; but others similarly treated might have been introduced where the result was different. Experience was rapidly gained in the epidemic, and with this practical result. The best treatment of cholera, and that which was finally adopted, was this :—First a mustard emetic, then iced water *ad libitum* (which was drunk with avidity and vomited copiously), and nothing in the shape of medicine.

As giving some information with regard to the results of treatment at different dates, I have selected one or two fairly definite diseases and ascertained their mortality, forty years ago, and recently, as displayed in our medical registers. For the earlier date I have consulted those kept by Dr. Barclay; for the later, I use numbers supplied by Dr. Lee Dickinson. It is necessary, in the first place, to bear in mind that the number of beds from 1851 to 1854 was less than that from 1888 to 1891, with the average of 331 to 353. First, with regard to fever, we meet with the difficulty that the old term 'continued fever' included typhoid, typhus, and other slighter and indeterminate febrile affections.

*Number of Cases admitted into St. George's Hospital and Number of Deaths from several Diseases in separate Periods of Four Years.*

| | Cases | Deaths | Percentage of mortality |
|---|---|---|---|
| *From* 1851 *to* 1854. | | | |
| Continued fever | 574 | 68 | 12 |
| Pneumonia | 168 | 48 | 28 |
| Acute rheumatism | 307 | 11 | 2·9 |
| *From* 1888 *to* 1891. | | | |
| Typhoid | 136 | 26 | 19 |
| Pneumonia | 419 | 94 | 22 |
| Acute rheumatism | 181 | 5 | 2·7 |
| Sub-acute rheumatism | 355 | 1 | 0·28 |
| Total of acute and sub-acute rheumatism | 436 | 6 | 1·3 |

Thus the diagnosis is uncertain excepting as regards the fatal cases, where it rests generally on post-mortem evidence, with the warrant that death was due to either typhoid or typhus. Typhoid was the usual form; typhus exceptional. In the latter period, typhus did not occur at all. We cannot safely draw any conclusion as to the results of treatment, but must be content with the knowledge that not only was there typhus in the hospital forty years ago, whereas now there is none, but that of typhoid there was a larger number of cases and a larger number of deaths in the earlier than in the later period. What was the proportion of deaths to cases in the earlier period we have no means of ascertaining.

With regard to pneumonia and acute rheumatism, the first impression is that the difference between the past time and the present is not so great as might have been expected, though both were undoubtedly more fatal then than now. The difference is most marked with regard to acute rheumatism. To appreciate this it is necessary that the inquirer should be made aware that what is now classed as sub-acute rheumatism would formerly have been called acute. In old days the class *sub-acute* was limited to local and trifling cases which could not be called chronic and certainly were not acute. All cases of general or multiple rheumatism of recent date and with any degree of febrile action were then classed as acute. Of late, since the introduction of the clinical thermometer, this term has been limited to cases where the temperature has gone above 102. Thus, general rheumatism, such as would formerly have been called acute, is now classed as sub-acute, whence it is necessary, in order to obtain a series of cases comparable with the old 'acute' class, to fuse together the 'acute' and the 'sub-acute' of modern times. On this showing the death-rate now is not half what it was. The total deaths in the hospital from rheumatism, notwithstanding the increased number of beds and the increased transmission of patients which we owe to Wimbledon, is now little more than half what it was. But

even this leaves the story half told, for the mischief of acute rheumatism is not to be measured by its immediate mortality, but must be judged of by its course and complications. The abbreviation of the disease and the lessened proportion of heart disease when such measures as I have described as belonging to the old period were exchanged for alkalies, are among the greatest improvements of modern medicine. As touching pneumonia—much more common of late, owing to the influenza—it has shown a smaller percentage of mortality. In the old days, five cases recovered for two that died; in the recent period, seven recovered for two that died. In the severe old days one would have thought that fewer would have survived; in the later times of indulgence, that fewer would have died. But yet the difference is decided, and may be expressed in this way: in the old days, five died where four die now. If we assume that the disease and the subjects of it were the same then as now, and attribute the difference only to treatment, we may say that our predecessors killed every fifth man—a sort of double decimation. This assumes our death-rate as the normal standard, as if our deaths were all due to nature and none to art. If, as may hereafter be found, our methods are responsible for some of our mortality, it will follow that the mortality due to the methods of our predecessors must be correspondingly increased. After all, the nature of a disease determines its mortality more than the treatment, besides which, if there be reason in what I have already advanced, the old treatment may have had certain advantages. It may have been of use to the lung *per se*, though not sufficiently encouraging to the lung's owner.

In presenting these particulars of the practice of our predecessors, let it be at once acknowledged that whatever may be the superiority of the practice of to-day, no such superiority can be claimed by the practitioner. Without using the term 'degenerate,' I may at least confess that we are the less heroic products of a later age. Wilson and Bence Jones, in their separate ways, were of exceptional

ability and of exceptional knowledge of what was then known. Thomas Young was of supreme ability. It is to be observed that Dr. Young and Dr. Wilson, though both frequently employed moderate blood-letting, were less indiscriminate and less liberal with regard to mercury and antimony than their contemporaries, of whom it may be said without disrespect that their intellectual eminence was less. Neither Young nor Wilson had in any great measure the confidence of the public. A physician is judged by his methods, not by his results. His method must be obvious and applied with vigour. If the patient die, there is the satisfaction that all was done that was possible; if he survive, the credit is given to the doctor, who did so much not perhaps *for* him, but *to* him.

Looking back upon the practice I have recorded, there can be little doubt that it was for the most part injurious—not, perhaps, always wrong in principle, but generally overdone. Patients would probably have been better off had they had no medicine at all. The leading motive was the belief that mercury, antimony, and starvation were antagonistic to fever and inflammation, which, on the other hand, were increased by food and stimulant. The next article in the creed was that many diseases were due to the liver which are now not so attributed, and that mercury was a hepatic panacea. With regard to the treatment of specific fevers, such as typhoid, we now find no reason to suppose that grey powder was beneficial, excepting that it may have done something to prevent constipation, perhaps not now always sufficiently guarded against. On the other hand, the lowering measures must have tended not 'to obviate the tendency to death,' but to increase it, when, as must often have been the case then as now, the tendency was to death by exhaustion.

As touching inflammatory conditions—pneumonia, for example—it behoves us, as I have already shown, to speak with circumspection, and even with some degree of reverence, at least, for the intentions of our predecessors. If we should resume their practice in a modified way, the extrava-

gance of their measures will ever be a warning against the like. I certainly do not see the use of the opium which they so constantly associated with the mercury. We no longer aim at salivation; while a slight purgative action would probably be beneficial where, as with pneumonia, there is so much to be got rid of.

The treatment of disease has advanced in the last forty years, not so much in the direction of what to do, as what not to do. We trust Nature more, and are less presumptuous in interfering with her. We employ more often than did our predecessors the policy of 'masterly inaction.' A few relics of superstition still remain—fewer, I will venture to say, at this hospital than elsewhere—but the three great scourges scourge no more. The lancet is in its sheath; mercury and antimony, employed with a parsimony which would call forth the scorn of our predecessors, do little harm and possibly some good; we have learned the use of iodide of potassium. The patient has no longer to struggle against the doctor as manfully as against the disease; and we may at least say this much for these latter days, that if he die, he dies of the disease, not of the physician.

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## *ON THE PRESYSTOLIC MURMUR FALSELY SO CALLED.*[1]

Some years ago I contributed two papers to the 'Lancet,'[2] which had for their purpose the correcting of what I then held, and still hold, to be an error—popular though it be—in regard to a murmur to which the term 'presystolic' has been applied. These essays gave rise to much discussion. They were directed against a favourite, I may even say a fashionable, hypothesis; and though they found some strenuous (it does not become me to say discerning) supporters, their opponents were, as might have been expected,

[1] The *Lancet*, October 1, 1887, p. 650.

[2] *Ibid.*, October 19, 1889, p. 779.

the more numerous. It was my design to have reproduced these papers at some opportune time; but now that the time appears to have arrived, I have judged it better to re-write than to re-print. I have introduced, however, the original diagrams without alteration. I thought that the papers might here be amplified and there condensed; some of the minor points stood in need of modification, and I could not but fear, since I held the contention to be sound, that its failure of general acceptance might have been due to insufficient explanation or unskilful advocacy.

Without attempting to follow the presystolic controversy at length, I will take brief note of one or two prominent points and prominent persons connected particularly with its early history.

Hope[1] recognised a diastolic mitral murmur, produced in the passage of the blood from the auricle to the ventricle, which, however, he did not attribute to auricular contraction. He says of 'the diastolic murmur of the mitral valve' that any lesion of this valve capable of sufficiently contracting its aperture may, under certain limitations, give rise to this murmur, which is extremely rare, and was formerly confounded with that of aortic regurgitation.

As to the origin of a murmur in auricular contraction, which is the later theory of Fauvel and Gairdner, and of general acceptation, Hope's remarks are as much to the point as if he had foreseen the future, and argued by anticipation. I will quote his words. Hope says: 'Will it be said that the auricular contraction, previous to the ventricular, should create a murmur? I have looked for it carefully, and have only once been able to suspect it, without being able to assure myself of its existence. Theoretical reasoning seems to countenance this result of observation, for, as the auricular systole is slight, the quantity of blood injected by it is not considerable; and, as the ventricle is already *full*, it cannot admit that extra quantity necessary to bring it to the state of distension without offering a

[1] A treatise on *Diseases of the Heart and Blood-vessels*, edit. 3, 1839, p. 78.

resistance to its ingress, which must greatly retard the force and velocity of the current—a force, indeed, which can never be great, because the auricles are not only weak vessels, but are unsupported by valves behind.' [1]

We now come to the presystolic theory, which was invented by Fauvel, to whom it owes its name. Shall I call Fauvel the apostle or the precursor of the new creed? If the precursor, Gairdner is the apostle, for it is to his teaching chiefly that it owes its spread.

M. Fauvel, in the year 1843,[2] drew attention to an apex murmur which commenced in the silence which follows the second sound, and in his view immediately preceded the first sound with which it terminated. This murmur he described as intense, rasping, rough, and energetic. He held it to correspond in time with the contraction of the auricle, which, as is known, immediately precedes that of the ventricle; and he showed by three cases, concluded by post-mortem examination, that it was associated with contraction of the mitral orifice. He considered the murmur to arise in the passage of the blood from the auricle to the ventricle, to be dependent on the contraction of the auricle, and to precede that of the ventricle. Here we have, at least, a distinct clinical gain; we have a definite murmur as the sign of a definite lesion. This is the result of simple observation, and admits of no cavil. The rule should have served for all time, but of late it has been so widely expanded and so loosely applied as to have lost much of its diagnostic significance. Whether the internal workings are as Fauvel inferred; whether the murmuring rush is driven by auricular contraction or ventricular; whether, in other words, it is presystolic or systolic, is a question which it is the purpose of this paper to discuss.

In the year 1862 Dr. Gairdner became the advocate of the presystolic theory as propounded by Fauvel; whether

[1] *Loc. cit.*, p. 79.

[2] 'Mémoire sur les Signes Stéthoscopiques de Rétrécissement de l'Orifice Auriculo-ventriculaire gauche du Cœur,' *Archives Générales de Médecine*, iv^e^ série, tome i. 1843.

he had acquired it from the French physician or arrived at it independently is not clear; but he explained this cardiac innovation with amplitude and detail, illustrated it with diagrams, and made it known not only within the Edinburgh Infirmary, but *in partibus infidelium*. Dr. Gairdner's view is in great part that of Fauvel. He describes the 'auricular-systolic' murmur as preceding and running up to the first sound, ending at the moment of this sound and of the apex beat, and as being the special murmur of mitral contraction. Dr. Gairdner gives diagrams of this murmur, representing it as beginning at the end of the diastole, or long silence, and 'running up to' the apex beat. This is the 'presystolic' murmur in question. Dr. Gairdner also gives illustrations of a murmur which begins immediately after the second sound, continues through the whole diastole, and terminates as does the other. This is a comparatively rare murmur, or it may be a combination of murmurs; so far as it is obviously in the diastole it admits of no difference of interpretation; it is diastolic, not presystolic, and from the auricle to the ventricle. My contention applies only to the murmur which is supposed immediately to precede the systole, and is named accordingly. This murmur is correctly described by Dr. Gairdner as rough, harsh, and of frequent occurrence. If it be, as is supposed, from auricle to ventricle, and is in fact a 'direct mitral murmur,' then the direct mitral murmur, instead of being, as used to be thought, obscure, infrequent, and a 'clinical curiosity,' becomes one of the most common and the most obtrusive.[1]

The presystolic theory was found worthy of all acceptation; it was thought to be supported by the most enlightened physiology; inferences founded upon it certainly came right in the dead-house; the presystolic cult became the fashion, and was adopted by all who would not be thought to be behind the forefront of medical science.

[1] The cases are so rare in which either the diastolic murmur alone, or the systolic and the diastolic murmurs together, can be fairly imputed to the mitral valve, that they are a sort of clinical curiosity. Latham *On the Diseases of the Heart*, vol. i. p. 38. 1845.

Every murmur supposed to belong to a contracted mitral, often erroneously so supposed, was called by this name. Every mitral murmur with the least sharpness in its termination was apt to be so spoken of; and so indiscriminately was the term applied that it came to have little use, excepting to give gratification in the employment of it.

Let me observe in passing that even in health under cardiac excitement, and under any condition of mitral disease in which it is possible for the valve to shut, a sharpness may be imparted to the end of the systole, which the eager disciple may think to justify the term 'presystolic.' Indeed, presystolic murmurs threaten to become so common that the old-fashioned mitral will be as much out of date as the rolling-pin stethoscope through which our predecessors used to listen to it.

To return to what I may call the legitimate misuse of the term 'presystolic,' a few persons, to some of whom especial consideration was due, either failed to understand or were unable to accept the new doctrine, and time added to the number of the dissentients. The late Dr. Ormerod and the late Dr. Barclay raised their voices against the new theory, and much discussion followed *pro* and *con*. Dr. Ormerod[1] disputed the auricular systolic character of the murmur on the ground that the auricular contraction was too weak and brief to cause so loud a sound, which he held to be due rather to the contraction of the ventricle, and to be regurgitant. Dr. Barclay[2] followed on the same side, with more elaboration, in the contention that the murmur in question was systolic, not presystolic; not direct, but regurgitant. He believed the murmur to occur at the beginning of the systolic contraction of the ventricle, to be due to regurgitation through a contracted and thickened mitral valve, and to owe its abrupt termination to the closure of that valve towards the end of the systole. He drew attention to the absence of valves in connection with the pulmonary veins, pointed out that with no means of

[1] *Medical Times and Gazette*, 1864, vol. ii. p. 154.
[2] The *Lancet*, 1872, vol. i. pp. 283, 353, 394.

closing the outlet backwards the auricle could exert but little power in driving the blood forwards, and maintained that it was 'scarcely possible that one of the loudest and roughest murmurs ever heard in cardiac disease should be produced by contraction of the auricle.'

Many years after Dr. Barclay had published these opinions my attention became directed to the matter to which they related. Putting together clinical and post-mortem evidence, I was led to the conclusion which my predecessor had already enunciated. I did not derive my views from his, for, to my shame be it said, it was not until I thought of writing down my own that I ascertained what his were; but I need not say that I was much confirmed in my interpretation of this murmur when I found it was also that of so excellent an auscultator, and so close a reasoner.

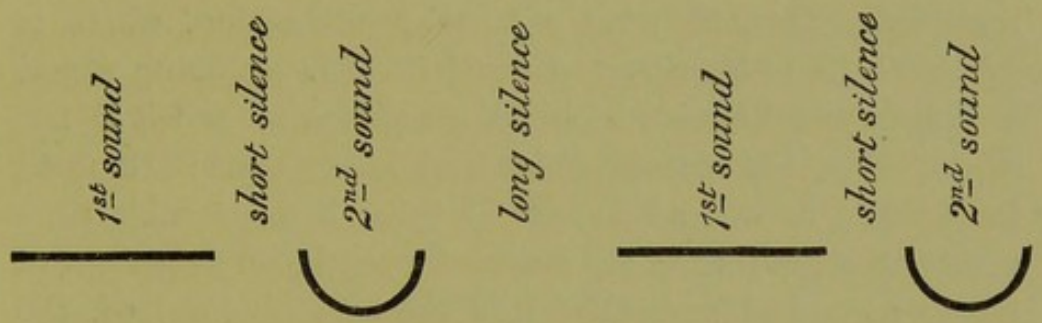
Without following in further detail the history of the controversy, but leaving much of value and interest relating to it to be found in the journals and elsewhere, I will now attempt a brief exposition of the subject in my proper person, and as it appears at the present time. In the first place it is necessary to define the murmur in question, as it offers itself to our senses. And it must be defined in terms which do not prejudge any difference of opinion. In the first place *presystolic* is one thing and *diastolic* is another. As the term 'presystolic' asserts a special relation to the systole, it may be presumed that the diastolic interval must be all but over before we can properly describe a murmur by this name. That only is presystolic which is in contact or apposition with the systole. If the murmur be obviously in the diastole, as it must be if it follows immediately upon or very soon after second sound, then it is diastolic, not presystolic.

The only murmur to which this term can be applied is one which begins at or near the end of the diastolic pause, sings small at first, rapidly gains in volume as it proceeds, and at its maximum suddenly stops with a snap and concussion. Then comes a short silence, and then the second

sound. This murmur, as evident to the ear, is often accompanied with a thrill evident to touch, with which it is synchronous.

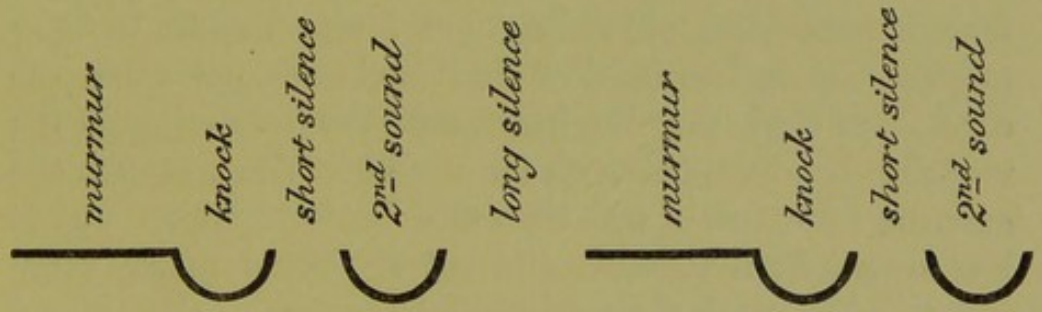
The change in the heart's tune may be readily represented on paper. The normal sounds may be represented as in diagram 1 : two sounds only, a long and a short with intervals. With the superaddition of the murmur in question there are, distinguishing between sounds and murmurs, two sounds and one murmur, three in all. In

Diagram 1.




Sounds of the heart in health.

Diagram 2.




Sounds of the heart with the so-called presystolic murmur.

health we have a long and a short ; under this form of disease one long and two short. The *trochee* becomes a *dactyl*. The change consists in the transformation of the first sound into a murmur, with a knock at the end ; and in the murmur and the knock lies the mystery or the doubt.

In judging *what* the murmur is the first thing to determine is, *when* is it ? Now I cannot doubt, unless I should distrust the evidence of my own senses, that the murmur

is coincident with the contraction of the ventricle, and belongs to the systole. Anyone who will bring two senses to bear at the same time, and employ his fingers together with his ears (a combination easier with the double stethoscope than the single), can assure himself that he hears the murmur at the same time that he feels the impulse. The impulse in these cases is often heaving and abnormally distinct, and serves exceptionally well to denote the phase of cardiac action. If there be a thrill it is felt with the impulse; the chest-wall vibrates as it heaves. The murmur does not occupy the whole of the systole, but terminates abruptly with a snap or concussion, which is evident both to hearing and touch. This snapping sound is exactly synchronous with the carotid pulse as felt in the neck. Now the carotid pulse does not present itself until the systole is well advanced; it takes a measurable time after the beginning of the systole for the blood to get out of the heart and make itself felt in the carotid. Indeed, the carotid pulse is generally represented as occurring when the systole is two-thirds over. I should apologise to those to whom this paper is mainly addressed for recurring to the facts of elementary physiology, but I may venture to do so briefly. It is known that the blood does not reach the carotid, or even leave the heart, until the contraction of the ventricle has been going on for a certain time, though the extreme estimates of this time differ widely. The ventricle has to pull itself together and get the blood under weigh before it can reach the vessels, and during this time mitral regurgitation may be taking place if there is anything to prevent the exact closure of this valve at the beginning of the systole.

As the carotid pulse does not mark the beginning of the systole, but a later stage of it, it follows that any transaction which *immediately* precedes this pulse must correspond in point of time with the early part of the systole. The murmur which 'runs up' to the snap, and to the carotid beat which is synchronous with it, is therefore, in whole or part, in the systole. The systolic time of the

murmur has already been inferred from its relation to the cardiac impulse.

The prevailing misunderstanding, as I hold it to be, rests on the assumption that the short sharp sound, the snap or knock, up to which the murmur runs, represents the whole systole instead of its termination. How far the knock represents the first sound may be taken into question; it is at least all we hear of it when the murmur goes before. Physiologists are not agreed as to the causation of the first sound; some think it is largely muscular, others that it is the result of tension established on the closure of the auriculo-ventricular valves. For my own part, I think it is chiefly or entirely due to causes which act within the cavities, of which sudden tension may be the chief. If the closure of the mitral valve be postponed, so may be the sound which depends upon it. The knock is a late and altered first sound, so far as it depends on intracardiac tension. But let us not deem that this sound covers the whole systole, instead of merely denoting its conclusion. Herein lies the 'presystolic' fallacy. The phrase 'runs up to the first sound' may be correct, but is misleading if the sound is taken as the equivalent of the act. If we say of the murmur that it runs with the systole up to the sound with which it concludes, we shall lose a cherished phrase and rid ourselves of a cherished error.

I have ventured to assert that the murmur called 'presystolic,' which I have sufficiently defined, is to be heard while the cardiac impulse is evident to touch. I must revert for a moment to this association. I have long been in the habit of testing the concurrence with stethoscope and finger, and must be forgiven if I speak with confidence. If the finger be pressed between the ribs, particularly in a thin person and when the heart is not too rapid, and if at the same time the double stethoscope be adjusted to the murmur, the beginning of the murmur will be found synchronous with the beginning of the impulse. Supposing the murmur to begin with the very beginning of ventricular contraction, then the murmur should be audible a

small fraction of time before the impulse is tangible; for it is obvious that a fraction of time must elapse between the very beginning of ventricular contraction and the movement of the parietes which is the result of it. I have often tried to satisfy myself that this sound, as theoretically it should do, preceded the impulse by never so little before joining company with it, but I never could assure myself further than that the two began together, and continued together until the characteristic knock or sharp sound put a simultaneous end to both. In this matter the cardiograph gives little help, since the instrument records movement but not sound—we want the two together. The stethoscope and the finger can be worked together satisfactorily in a suitable case; or, if it be desired to make the impulse evident to sight instead of to touch, a simple device may be employed: a wedge-shaped piece of cork may be fixed into the intercostal space with diachylon plaster and a light shawl-pin stuck into the cork.[1] The movement of the pin will indicate that of the apex; this confirms what the finger declares, but has little advantage over it.

If my observations are correct, which I cannot doubt, it must be concluded that the special murmur in question is in the systole, and the short sound which immediately follows it—up to which it runs—marks the conclusion of that act.

I must here dispose of a misconception which is not uncommon. The systolic knock resembles in shortness and tone the second sound, and is sometimes mistaken for it. The true second sound following, the two are apt to be classed together under the term 're-duplication of the second sound.' This error only requires to be mentioned to be guarded against.

Turning now to the heart after death, we find, as a rule, when the murmur has been definitely expressed, a narrowed mitral orifice with thickened valvular structures. Certain exceptions have been recorded, to which I shall advert presently. Having regard to the rule and not to the excep-

[1] See *Lancet*, October 1887, p. 651.

tions, the orifice presents many degrees of constriction, from perhaps half the normal calibre to what will admit only a cedar pencil or even the blade of a penknife, though it may be mentioned in passing that when the extreme narrowing is reached, such in shape and dimension as to suggest a small knife as its gauge, the murmur often ceases to be audible. The valvular structures are generally much thickened with fibroid contractile tissue, apparently the result of ancient endocarditis, though atheroma may be in some cases largely concerned. The dense fibroid thickening, whether simple or complicated, leads to obvious impairment of valvular action, the delicate fringe at the edge of the flaps is obliterated, and the nice adjusting organism replaced by one of which the chief characteristics are constriction, stiffness, and want of adaptability. The opening from auricle to ventricle is narrowed by a general contractile process which often brings together the mitral flaps laterally, so that a sort of funnel is presented, of which a small hole at the bottom represents the only communication between the two chambers. This hole may be like a buttonhole, a comparison which has been often applied.

Two defects of action are inevitable in these cases: there must have been obstruction and there must have been regurgitation. The auricle is usually somewhat thickened, but not much—not enough to give muscular power comparable to that of the ventricle, or to explain the intensity of the murmur and sense of force which it imparts on the theory that it is of auricular origin.

The murmur has, indeed, been known to occur when 'there has been practically no efficient muscle in the auricle capable of contraction,'[1] in which cases the murmur has been attributed to the elasticity of the auricular walls or to the suction of the ventricle, neither of which hypotheses commends itself to the mind; the murmur is too intense and too long to be the result of elasticity apart from muscular force; besides which the effect of elasticity must diminish

[1] Sansom on *Diseases of the Heart*, p. 346.

as tension is relieved, whereas the murmur increases as it goes on. As to ventricular suction, this can scarcely account for a murmur which is supposed not to present itself until this act is approaching its conclusion.

Regurgitation through the mitral valve is a physical necessity under the pathological circumstances; the process has all the force of the ventricle behind it, and it is contrary to what we know of the heart in disease to suppose the regurgitation to be without sound. Where there are two murmurs belonging to the mitral orifice (not so rare an event as our ancestors supposed), one may be, even must be, from the auricle and in the diastole; but where there is but one, which is the condition I am now contemplating, and that one of the kind under discussion, the probability is so greatly in favour of its being from the ventricle and systolic that it might be assumed to be so unless there were some clinical reason to the contrary. But I have shown that all clinical reasoning is in favour of, not opposed to, its ventricular origin.

The snap or knock which stops the murmur and concludes the systole must be looked at, like the murmur, in the light of pathology. The snap or knock conveys to the hand a jar or sense of concussion, which ensuing after the peculiar lifting impulse, is so characteristic that mitral stenosis can generally be detected by touch before the ear is called in. The valvular change, attended as it is with rigidity and contraction, necessitates delay in the closure of the valve, but allows the closure to be accomplished at last, and this with a sudden contact of hard edges, or at least with the sudden establishment of tension due to closure. The delayed closure is no doubt brought about at last by the contraction of the ventricle and its grasp upon the valve and orifice. This closure necessarily puts an end to both regurgitation and murmur and marks the end of the systole. Whether the knock or snap is produced by the percussion of closure or by the tension thus suddenly established may be held in question, but at any rate it is due to closure. The knock which stands for the first sound, or for the valvular

part of it, all we hear, does not take place until closure is accomplished, which owing to the rigidity of the valve is abnormally delayed. In health the delicate edges of the mitral float together early in the systole; in the state under consideration the thin fringes no longer exist, the flaps are thickened and stiffened, and the closure and the sound which goes with it is postponed, during the whole of which delay the regurgitant murmur is going on.

While maintaining that the so-called presystolic murmur is really systolic, far be it from me to deny the mitral diastolic, or to question that it is made from auricle to ventricle.

A mitral murmur which begins immediately after the second sound, and continues through the diastole, can have no other interpretation. It arises as the blood enters the ventricle through the mitral valve, whether in virtue of ventricular suction or auricular contraction. Suction probably has the more to do with it; the contraction of the auricle, at best a feeble act, begins at the end of the diastole when the murmur in question has been long in progress. After all, the ventricle is the chief agent in moving the blood, whether in maintaining the circulation or making murmurs. The diastolic or direct mitral murmur, though now well known, is rare, whereas the *pseudo-presystolic* is common. When the former presents itself, the latter is apt to follow, as a contracted orifice is generally one which permits of regurgitation. The diastolic ends before the 'presystolic' begins, but there is no abrupt separation between the two. The diastolic sometimes has an undulating character, and, like the 'presystolic,' may be accompanied with a thrill. It has been often looked upon as part of the 'presystolic,' though, if my contention be correct, it is essentially different, as belonging to a different cardiac act. It has often occurred to me as a difficulty that these two murmurs, up and down, in and out, should not be abruptly separated. One slopes into silence, from which the other softly emerges without any obvious note of reversal of the current. I suppose there is always an actual pause, however

brief, between the two, so that the contrary murmurs are not in actual apposition.

While upon the concurrence of murmurs, I may advert to what I hold to be a common misinterpretation in this respect. The 'presystolic' is held to 'run into' a systolic. It occasionally happens that a so-called presystolic runs on through the knock, and then ends softly after the manner of the ordinary systolic. The knock is, as it were, imbedded in the murmur. This I presume to be an early systolic continued into a late systolic. The valve closes enough to give rise to the concussion, but not so as totally to stop the regurgitation and abruptly cut off the murmur. The common phrase which represents the 'presystolic' murmur as 'running into' a systolic is not satisfying. In this view the first murmur, with the blood going in one direction, is inseparably blended with the second, in which it is going in another. The full volume and gush is continued without hesitation or abatement. The knock is heard within it, but the murmur is absolutely continuous; and yet we are asked to believe that in the midst of it the course of the blood is reversed. When the so-called presystolic follows the diastolic—in which, in my contention, there is actually a change of direction—there is, as I have said, usually a suspension, however brief, between the soft ending of one murmur and the soft beginning of the other.

Some approach to the presystolic tune may sometimes be heard, even though mitral stenosis be slight or absent, so long as the mitral valve retains its power of ultimate closure. If we have regurgitation early in the systole, and closure of the valve at last, we shall have a murmur with an abrupt ending, and an excuse for the more extensive misuse of the term than obtained in the early days of the presystolic era. There is quite a body of speculative literature recording and endeavouring to explain the occurrence of the 'presystolic' murmur without mitral stenosis. If the murmur be, as is popularly held, a direct mitral, it is certainly difficult to account for its presence when the mitral orifice is uncontracted. But if the murmur be regurgitant,

the mystery disappears. Looking at such reports as I have alluded to, we generally find some condition of the mitral valve which would not only account for, but necessarily give rise to regurgitation. And if that regurgitation is succeeded by mitral closure, then we have an approach to the murmur to which the term 'presystolic' is applied. We are indebted to Dr. Phear for a useful collection of published cases in which the so-called presystolic murmur was present without mitral stenosis. Dr. Phear is not of my way of thinking, but perhaps his evidence is not the less valuable on that account. If it were desired to prove the frequency of mitral regurgitation in the circumstances to which he has regard, it would be scarcely possible to find a mass of evidence more conclusive in this respect. Of forty-five cases in which the state of this valve is recorded, I find twenty-two in which the mitral valve was thickened, five in which it was the seat of vegetation or atheroma, twelve in which the orifice was dilated. In several, shortening or thickening of the chordæ tendineæ is mentioned. Knowing as we do how slight an interference with the mitral mechanism will suffice to give rise to regurgitation, we are justified in presuming that this was present in most, if not in all. I append a more detailed analysis of Dr. Phear's cases.[1] It will be seen that

[1] 'State of the Mitral Valve in Forty-five Cases of "Presystolic" Apex Murmur without Mitral Stenosis,' collected by Dr. A. G. Phear, published in the *Lancet*, September 21, 1895.

| | |
|---|---|
| Valve thickened | 2 |
| Thickened and incompetent | 1 |
| Thickened, but competent | 3 |
| Thickened and contracted | 2 |
| Thickened, with vegetations or endocarditis | 4 |
| Thickened, with orifice dilated | 5 |
| Thickened, with dilated orifice and vegetations | 1 |
| Valve thickened, chordæ tendineæ thickened or shortened | 4 |
| Patch of atheroma on valve. Dilatation and hypertrophy | 1 |
| Mitral orifice dilated | 7 |
| Orifice dilated, vegetations | 1 |
| Orifice measuring from $3\frac{3}{4}$ in. to $4\frac{1}{2}$ in. | 4 |
| Vegetations on mitral valve | 4 |
| Much hypertrophy, valve healthy | 3 |
| 'No stenosis | 1 |
| Healthy | 2 |
| | 45 |

of the forty-five one is insufficiently described. Of the rest, all but five present conditions in which regurgitation is either certain or probable. As to the five exceptions, it may at least be said that of all ways in which the heart can go wrong, none is more apt to be overlooked after death than mitral regurgitation; perhaps it was overlooked in some of those few cases where the lesions belonging to it are not recorded. In fine, Dr. Phear's facts point to a conclusion which he has not discerned—that mitral regurgitation, not mitral obstruction, is the essential lesion of the 'presystolic' murmur.

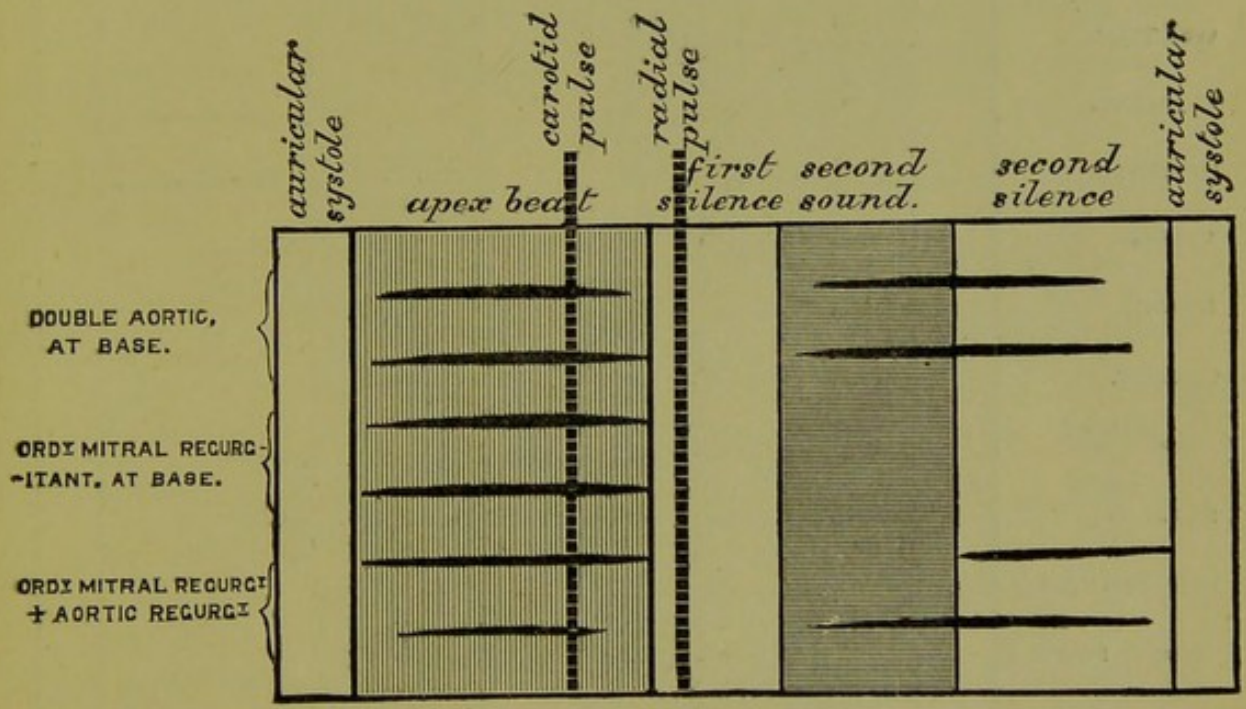
I here reproduce from a paper to which I have already referred some diagrammatic illustrations of cardiac murmurs.[1] I have arranged an ordinary physiological scheme of the heart's cycle in a horizontal instead of a circular form, and placed within it the murmurs and sounds in question. The systole is here represented as a space indicating a due proportion of cardiac time, instead of as a line, which is often done and is deceptive. The murmurs are represented by continuous marks; the 'knock' by detached vertical lines. The relation of the murmur to the systole, or in other words to the tangible impulse, is charted on the joint evidence of touch and hearing, both of which witnesses also give evidence of the position of the knock, which can be felt as well as heard. The position of the arterial pulses is duly marked; that of the carotid is, as has been shown, of especial interest in relation to points in dispute. It may be said that a diagram common to many cases will sometimes not exactly suit all: the diastole may be a little longer in one case or the systole a little shorter in another. I may have employed a little of the method of Procrustes, but if there be any misrepresentation it touches only the relative length of the acts, and not their character.

Diagram 3 shows some ordinary murmurs about which there is no question. Diagram 4 displays the incidence of the so-called presystolic in relation to the impulse and the

[1] *Lancet*, October 19, 1889, p. 780.

knock with which the impulse terminates. I have given a preference to cases in which diastolic mitral murmurs existed as well as the 'presystolic,' so that the comparatively rare diastolic murmur appears in the diagram to be less infrequent than in the wards. The interval between the diastolic murmur and the false presystolic is shown; the nearest approach to a continuous murmur is in the last case, where the diastolic murmur was occasionally much prolonged.

Diagram 3.




Ordinary mitral and aortic murmurs, for comparison with next diagram. The ordinary mitral systolic are shown as going through the whole systole without abrupt stop. The aortic regurgitant begin sometimes within, sometimes immediately after, the second sound.

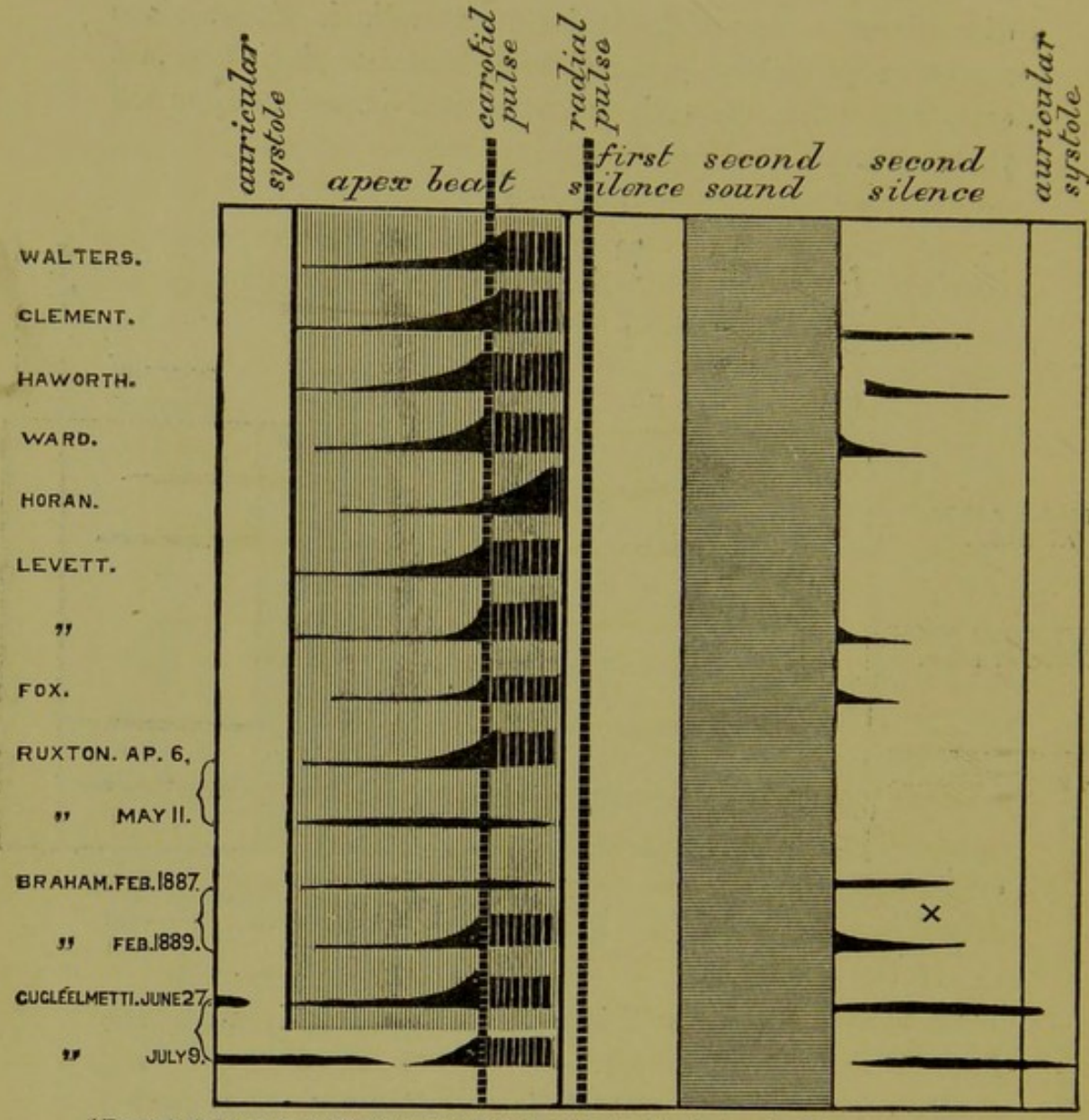
Diagram 4 shows this case (Guglielmetti) with the figure differently arranged.

To conclude—the presystolic question may be regarded as turning on the knock or snap which follows the murmur; if this brief and momentary sound represents the whole systole, the beginning as well as the end, then, since the carotid pulse is synchronous with this sound, arising exactly with it, there is no interval between the beginning of contraction in the ventricle and the completion of the act as indicated by the filling of the great vessels. But

this is contrary to the teaching of physiology; there is such an interval, and it has been measured.

Again, if the knock represent the whole systole, then

Diagram 4.




'Presystolic' and diastolic mitral murmurs at apex in mitral stenosis. Murmurs are represented by a continuous horizontal mark, the knock by vertical lines. In many cases the so-called presystolic murmur is accompanied by a short diastolic or direct mitral, from which the presystolic is distinct. The two cases before the last show a not uncommon occurrence—a systolic murmur at one time, and a 'presystolic' at another. In the latter phase of the last case the elongation of the diastolic murmur and the abbreviation of the 'presystolic' prevented its fitting the table; it is therefore reproduced in the next diagram.

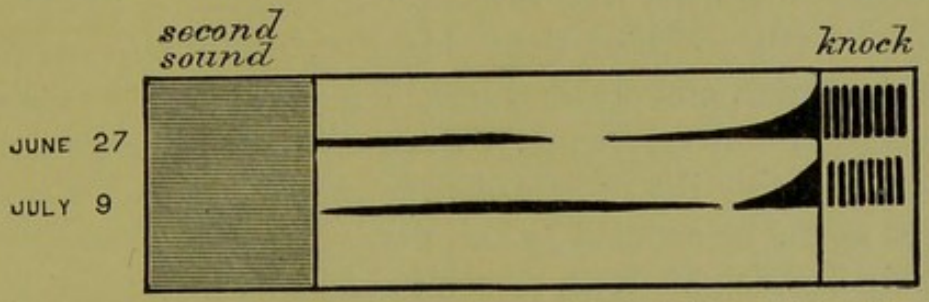
the impulse which precedes it, which may be conspicuous, powerful, and prolonged, and is at the apex, must be diastolic, merely what may be called an accidental move-

ment, imparted to the ventricle by the contraction of the auricle—an explanation which presents itself to my mind as improbable.

If these two deductions be accepted, then we may pin our faith upon the presystolic creed, and join with those who hold it in a sort of auricular confession.

But if it be as I have contended, if the impulse represent the systole of the ventricle and the knock the end of it, if the ventricular contraction begin, as physiologists suppose, an appreciable time before the carotid pulse, and therefore before the knock which is synchronous with it, then the murmur which immediately precedes the knock

Diagram 5.




The last case represented in Diagram 4 is perhaps better shown in this, which is differently arranged, giving the second sound and the knock as the only indications of the place of the murmurs. The variable position of the break between the murmurs and the apparently variable length of the systole made it impossible to fit this case into the preceding scheme.

concurs with the systole and must be called systolic, not presystolic. The presystolic faith must in this case no longer be ours, but be respectfully put away among the errors of the past.

If the contention of this paper be granted, I would propose that the term 'presystolic' be totally abandoned. Let us call all murmurs in the diastole *diastolic*, all murmurs in the systole *systolic*. If we choose to give a distinguising name to the special regurgitant murmur of mitral stenosis, let us call it, as I formerly proposed, 'early systolic,' or, better still, 'arrested systolic.' We might call the familiar blowing murmur which is continued into a soft ending, the 'prolonged systolic.'

## *ALKALIES IN RHEUMATISM.*

Many years ago, while holding a subordinate position at St. George's Hospital (that of Medical Registrar), which gave me an opportunity of viewing with an equal eye the practice of all the physicians, I directed my attention to the various modes of treatment employed for acute rheumatism, which were then sufficiently different to promise different results unless the course of the disease were incapable of being influenced bv medicine.[1] I collected and tabulated all the cases I could bring to hand between the years 1857 and 1861, having regard especially to the state of the heart. The measures resorted to comprised most that were in vogue at that time—bleeding, blistering, mercury, opium, nitre, and alkalies, besides reputed specifics such as guaiacum and colchicum.[2] Some of the reputed remedies were probably the reverse of remedial; of the rest, some at best might be counted as harmless, while others were probably beneficial. With regard to blood-letting, which gave the worst results in my series, the heart became affected after it with either endocarditis or pericarditis in exactly half the number—four of eight. The number is small, and it may be fairly presumed that all were severe. In most of them other measures, generally calomel and opium, were employed also, which somewhat complicates the question. Placing together all the cases treated without alkalies, twenty-eight in number, the heart became affected under treatment in eleven, or more than a third. Nitre gave better results than any other medicine excepting alkalies; of thirty-five cases treated with this salt, with or without salines, seven, or one in five, contracted heart disease under it.

The collection of cases to which I have referred gives no adequate information with regard to the effects of opium, formerly much commended in rheumatic fever. I supplied the deficiency from the practice of Dr. Sibson at

[1] See paper in the *Medico-chirurgical Transactions*, vol. xlv.

[2] See page 209.

St. Mary's Hospital. Of twenty-one cases of acute rheumatism admitted while the heart was as yet sound, and treated with opium, fourteen, or exactly two-thirds, acquired either endocarditis or pericarditis under it. The minute carefulness of Dr. Sibson probably never let a murmur escape him, and he has given his results without mitigation or remorse. With all allowance, the record of heart disease is at least a caution. Medicine appears to have done its worst in the hands of this benevolent and conscientious physician. I think it might reasonably have been inferred that opium would prove injurious in the disease under consideration. If we have to do with a poison which is capable of elimination, the locking-up of any of the doors by which it may make its exit seems scarcely the way to speed the parting guest.

Reverting now to St. George's Hospital, I come to the treatment by alkalies. This was practised chiefly by Dr. Fuller, and the results were abstracted, as in the other cases, from the books kept by the clinical clerks. Of the cases subjected to the 'full alkaline treatment' (such as to keep the urine constantly alkaline) these books recorded only one instance of cardiac inflammation coming on under treatment out of forty-eight cases admitted without heart affection. In this instance an endocardial murmur, together with pericardial friction, was noted on the second day of treatment, and neither was found after the tenth. Negative evidence is in its nature liable to error. Neither physicians nor clerks note everything, and possibly Dr. Fuller did not possess the stethoscopic fastidiousness of Dr. Sibson. Some murmurs must have been overlooked (unless the alkalies afforded a more complete protection then than now), and some probably regarded as hæmic which were valvular; but nevertheless the difference is so striking that it can scarcely fail to have a basis of fact. It is to be borne in mind, as tending to a fair comparison, that the evidence of the issues of every kind of treatment was drawn from corresponding records. The *prima facie* conclusion is that alkalies, given to alkalinity of the urine, do something which the other methods of treatment hitherto considered do not do towards prevent-

ing the cardiac complications of acute rheumatism. This conclusion, based on clinical evidence, is consistent with what is known of the chemical action of these agents in preventing the coagulation of fibrine. If the conclusion be well founded, and I do not see how it can fail to have something in it, we have the means, or at least *a* means, of preventing a certain amount of chronic disease, and depriving rheumatic fever of some of its remote mortality.

This conclusion was arrived at by observing and comparing the practice of others; since it was formed I have had considerable opportunity as physician to two hospitals of putting it to the test myself. While thus employed the problem was complicated by the introduction of salicin as a remedy for acute rheumatism—a remedy which was eagerly adopted and soon given almost universally either as salicylate of soda by itself, or this salt together with alkalising salts of potash. I myself never abandoned the alkalies, though I accepted salicylate of soda as an adjunct. Some others used salicin or salicylate of soda alone. The effect of these drugs in mitigating or cutting short the articular and the febrile effects of rheumatism does not admit of question; but it remains a question whether they extend any such protection to the heart as it appears to derive from the alkalies. The protection of the heart must be the chief aim in the treatment of rheumatic fever. If we look at the large mortality of valvular disease and at the large proportion of it which is to be traced, however remotely, to rheumatism, we see before us a problem the practical importance of which can scarcely be overrated.

It is the fashion to place a low value on statistics. Statistics may mislead when the items are erroneous, but so far as the individual statements are true, the more of them the better, and the sounder the conclusions to which they point. But besides statistics, or experience expressed numerically and in writing, there is something in experience the records of which are written in the brain—not always in detail, but in a condensed and portable form easy

to make use of. Since I have been a hospital physician I have invariably employed the alkaline treatment, unless, what rarely happened, there was some special reason for not doing so. Formerly I gave the alkalies alone, latterly in conjunction with salicylate of soda. My early observations had left me with so strong a conviction of the protective virtues of the alkalies that I should have thought it wrong to withhold them or to make experiments with mint water, or otherwise, which might have among their results chronic disease and premature death. I therefore have had little experience myself of the treatment of rheumatic fever without alkalies, but I have witnessed the results of such treatment in the hands of others with a feeling of relief that the responsibility was not mine.

The alkaline treatment, as I have been accustomed to employ it, and as it has been generally applied at St. George's Hospital by those who made use of it, consists in the rapid introduction of alkali to the alkalescence of the urine, and the repetition of the medicine at such intervals as to keep it so. I have been accustomed to give for each dose a drachm of bicarbonate of potash with fifteen grains of salicylate of soda, to administer one immediately on the patient's admission, to give three more doses at intervals of two hours, then two or three at intervals of three hours, and afterwards, the urine being now fully alkaline, to continue at intervals of four hours, insisting on its regular administration day and night, sleeping or waking. This must be long continued and not abruptly stopped—perhaps given for two or three weeks without diminution, and afterwards cautiously increasing the intervals or lessening the dose, taking care to keep the urine alkaline until the disease is at an end. It must be insisted on that insufficient doses, not enough to keep the urine uninterruptedly alkaline, are of little or no use. I have known cases in which heart complications have unexpectedly occurred, and been accounted for by the discovery that the medicine had been carelessly omitted and the urine allowed to recover its acidity.

Under this system, *carefully and exactly pursued*, I am satisfied that there is less endocarditis, less pericarditis, and fewer relapses than under any other with which I am acquainted. Another point of importance as bearing on this method is the frequency with which, under its influence, slight apex murmurs or slight abnormalities of sound, such as threaten further or more decided disease, disappear; and they often disappear so immediately on the establishment of the treatment that we cannot but assign the treatment as the cause of their disappearance.

As bearing on the question of treatment, I introduce some evidence which I owe to the Medical Registrars between the years 1888 and 1894. In this I have taken no personal part, excepting that many of the patients referred to as under full alkaline treatment were mine. Almost all the cases of late years have taken the salicylate—some with full alkalies, some with little, some with none. As my purpose was to ascertain the effects of the *full* alkaline treatment, all cases under inadequate doses were excluded. The statements relate only to patients who were admitted with the heart sound. The abstracts are placed in chronological order, and in the words in which they were supplied to me by their authors.

*Abstract by Dr. Lee Dickinson of Cases reported by Dr. Sisley and himself.*

'During the period in question, extending from January 1, 1888, to June 30, 1893, almost every case of acute and sub-acute rheumatism was treated with salicylate of soda, and the great majority with alkalies also. For the most part the alkalies were given intermittently, or in quantity not amounting to "full alkaline treatment," which was strictly understood as not less than one drachm every four hours. The object being to compare the frequency of cardiac complications under the combined salicylate and full alkaline treatment, and under salicylate without alkali, all the cases under partial alkaline treatment were rejected. Further, all those were rejected in which on admission the heart was abnormal,

or in which the complication appeared less than twenty-four hours after admission.

'The development of a valvular murmur was taken as evidence of endocarditis; friction as evidence of pericarditis.

'Owing to the effect of salicylate of soda in cutting short the rheumatic process, and to the fact that every case had been already under treatment for twenty-four hours, the proportion of cardiac complications is very small.'

*Dr. Sisley's Registrarship.*

'Twenty-eight cases under combined salicylate and full alkaline treatment. Two developed endocardial murmurs, but in both cases after the alkaline treatment had been relaxed.

'Seventeen cases under salicylate without alkali. Two developed endocardial murmurs:—

'(*a*) Temporary systolic apical murmur on fifth day.

'(*b*) Diastolic and mitral systolic murmurs on third day.

'One case admitted with a slight systolic apical murmur lost it under salicylate alone. Six cases admitted with the same murmur lost it under the combined treatment.'

*Dr. Lee Dickinson's Registrarship.*

'One hundred and twenty cases under combined salicylate and full alkaline treatment. Ten developed endocardial murmurs, three pericarditis. In the latter three, and in seven of the former ten, the complication appeared more than one, and generally several days after the alkaline treatment had been relaxed or discontinued.

'There remain three cases of endocardial murmur developing while full alkaline treatment was in force.

'(*a*) Soft systolic apical murmur on twelfth day.

'(*b*) ,, ,, ,, ,, third day.

'(*c*) Very faint systolic murmur just outside apex on fifth day, not persisting.

'Forty cases under salicylate without alkali. Two developed endocarditis, and one pericarditis also.

'(*a*) Diastolic murmur on fifth day, eventually disappearing.

'(*b*) Blowing mitral murmur three weeks after admission, followed by pericarditis.'

*Dr. Cyril Ogle's Registrarship, reported by himself*, 1893–94.

'Salicylate and full doses of alkali. Of twenty-six cases so treated, none developed signs of either pericarditis or endocarditis. Of these cases fourteen were on admission judged to have the heart normal, whilst the rest of them, twelve in number, had either an excited and irregular action, or muffled or murmurish first sound at the apical region, which was regarded as evidence of early heart implication; but under treatment the heart became normal in all cases.

'Salicylate only. Of five cases, one developed an apical systolic murmur in hospital.'

Putting together the results of the three Registrars they amount to this: 224 cases of acute rheumatism were admitted with the heart unaffected at the time of admission; 162 had full alkalies together with the salicylate. Of these, three developed endocardial murmurs while under this treatment. Several developed murmurs and pericarditis after the alkalies had been relaxed or discontinued. Sixty-two were treated with the salicylate alone, under which five developed endocarditis and one pericarditis also.

The conclusion is that under full alkalies, and while under them, one case in fifty-four displayed heart affection; under the salicylate by itself, one case in twelve.

The rule which I have followed of considering only cases admitted without heart affection limits the inquiry. Had I not been thus restricted, I might have drawn attention to many instances: such, for example, as those referred to in Dr. Ogle's report, in which the heart, though not natural on admission, became so under treatment.

I have written this short paper as an afterthought

while the others were going through the press. My opportunities of hospital observation have come to an end, and in looking at the impressions which remain with me, I find none which seem to have more practical importance than those which relate to the treatment of acute rheumatism. One thing is certain, however it is to be explained. Heart affections under rheumatic fever, perhaps especially pericarditis, are greatly less frequent than they were thirty or forty years ago. Modern treatment has probably done something, and the discontinuance of the ancient treatment may have done something too. It is difficult to look back upon the frequency and severity of cardiac complications in the days of depletion without suspecting that the old measures, or some of them, whether opium, bleeding, or mercury, actually invited the cardiac lesions (see p. 209). Be this as it may, the better results of modern times admit of no doubt; and as they became apparent before the salicylates were employed, I think they may fairly be ascribed to the somewhat general substitution of alkalies for remedies of more questionable advantage. So confident am I that a degree of protective power belongs to the full alkaline treatment, that it has seemed to me that I should be failing in a duty if I forbore to submit, and even to urge, my belief in this respect. It is apparent that, as regards salicylates, the heart is safer under salicylates and alkalies than under salicylates alone. The protective power of the salicylates is a matter of doubt; it can scarcely be that they have none, for they shorten the disease and with it the cardiac liability, but it is not to be supposed that they exert any special saving influence.

If my observations are erroneous, which they can scarcely wholly be, it may be said at least that if alkalies do no good they can do no harm; if there be a doubt, let the patient have the advantage of it. When I say 'no harm,' I mean practically none, or none that will last. A long course of alkalising treatment may be followed by anæmia and hæmic murmurs, but these are transitory and are by no means limited to this method. Let those who

do not recognise any protective power in alkalies (it is not claimed that they are absolutely protective) at least recognise that there may be room for doubt, and justification for an experiment which promises so much and costs so little. And let those who have not been in the habit of treating rheumatism with alkalies do it thoroughly. No drivelling doses and careless intervals, but let the treatment be prompt, sufficient, and persevering. Let them employ the system as if they believed in it, and I think they will probably end by doing so. Neither alkalies nor anything else we know of can entirely put a stop to the cardiac results of rheumatic fever, yet I think there is little doubt that by their means we may make these all-important consequences less frequent.

In conclusion, I have to record my obligation to Dr. Lee Dickinson and Dr. Cyril Ogle for having helped me in particulars which I have sufficiently indicated.

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