arterioles which are blocked, whereby there occurs not necrosis of a large mass of nervous matter, but an impaired or disturbed nutrition of the part. In the one, as Dr. Hughlings Jackson would say, there is destruction of nerve-tissue and, as its result, paralysis; in the other, there is an unstable condition of grey matter and, as its result, spasm. "Now, significantly, the process of embolism was one which led to increased afflux of blood in those organs (brain, retina, etc.) which had what Cohnheim calls 'terminal' arteries. This is accepted doctrine. Every physician knows that, in their first stage, renal, splenic, and pulmonary infarcts are *red*, and embolism or thrombosis is the accepted explanation of ordinary 'red softening' of the brain. There was, indeed, from blocking of arteries, not simple hyperæmia, but also extravasation of blood."*

I am aware that the morbid anatomy of chorea is not considered to lend much support to what has been termed "the attractive hypothesis of embolism". But, acknowledging the fact, this only amounts to clinical observation and experimental research being in advance of pathology. There are many problems besides this which pathological-histology has yet to elucidate. Cases are recorded where embolisms have been found in the middle cerebral arteries in chorea, as in those recorded by Dr. Tuckwell of Oxford.† Dr. Tuckwell, in two most interesting papers, gives the *post mortem* appearances of two fatal cases of chorea. In these, vegetations were found on the auricular surfaces of the mitral valve, and patches of red softening in the brain, the result of embolism. Dr. Tuckwell's articles are worthy of careful perusal, for he enters very fully into the pathology of chorea, and contests Dr. Ogle's and Dr. Barnes's views. His first article appeared in the *British and Foreign Medico-Chirurgical Review*, page 506. Dr. Broadbent's‡ and other cases where capillary embolisms are found are few compared with the numerous observations in which no embolisms were discoverable. Drs. Wilks and Moxon only represent general experience when they say:§ "We have seen no microscopic emboli." Quite recently, Dr. Howship Dickinson|| has given, what has been thought by many, the death-blow to the embolic theory. I hope to show, on some future occasion, that Dr. Dickinson's observations have not that antagonism to the theory of embolism which he and others suppose. It seems scarcely fair to place theories in opposition to observation; but acknowledging, as all would who believe in the embolic origin of chorea, the value of Dr. Dickinson's researches; yet the changes found by, and the pathology of, Dr. Dickinson have not met with the acceptance of the acknowledged holders of the embolic theory, for the reason that they do not explain all the phenomena of chorea. For the present, at least, the pathological histology is too uncertain to decide the issue of the nature of chorea. In experimental research and in clinical observations, we may find the clue to the problem. It is this that has induced me to make these remarks.

I cannot regard the occurrence of my patient's chorea and her mother's hemiplegia as mere coincidence, for both had a cause in operation which would satisfactorily account for the nervous disorder. The cause was the same in each case; and the results, though not identical, were analogous and parallel. I hope it will not be thought that I have attempted to strain the circumstance, either with regard to the patients being mother and child, or in regard to a nervous affection happening in two persons the subjects of heart disease. Nothing could be further from my wish. That two persons having a valvular lesion of the heart, whether they were strangers or related, should fall down and break the same leg, would be a coincidence and nothing else; for, though the heart-disease might lead to faintness, causing the patient to fall, there would be no intelligible explanation of why they should break the same sided leg, or how the relationship conducted to the result. But the cases are quite distinct. The relationship in my cases shows that the mother may have transmitted to her daughter a temperament or diathesis, by which the latter became affected with endocarditis, causing a valvular lesion similar to that from which she herself suffered. The heart-disease in each case will account for the occurrence of embolism; the embolism will account alike for the hemiplegia and the hemichorea. The relationship between the two patients is more interesting than important to the argument. The fact is striking, and naturally leads a thoughtful person to inquire into the circumstances under which mother and daughter, affected with the same form of valvular disease, are attacked with what is generally regarded as a nervous disorder. And I have endeavoured to show that the most reasonable explanation of the

daughter's chorea is that which suffices for the mother's hemiplegia—namely, embolisms in the area of distribution of the middle cerebral artery. The relation between the heart-disease and the hemiplegia of the one, and the hemichorea of the other, must be regarded as causal, not casual. The range of the nervous affection—its unilateral distribution—points to the anatomical alliance of the two diseases.

In conclusion, I am aware that I have brought forward no arguments or facts which have not been adduced more ably before. The non-acceptance of these must be my excuse. Iteration has occasionally its uses.

OBSERVATIONS ON DR. LEARED'S REPLY TO DR. WILLIAMS ON THE MECHANISM OF THE SOUNDS OF THE HEART.

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1. IN contradiction to Dr. Leared's statement that "it is *absolutely impossible* to distinguish the sound caused by friction of the heart's surface against the end of the stethoscope from any allied sound—*i. e.*, in this case, muscular sound—I maintain, from large experience of auscultation of the naked heart, when assisting Dr. Sibson in his experiments, that it is both possible and easy; that the sound heard during the contraction of the empty heart is not frictional, but muscular; and that, consequently, whether there is sound produced by collision of blood or not, there is sound from sudden tension of the heart-walls. I do not understand Dr. Leared to deny that sudden tension of membranous valves and tendinous cords must be attended with the production of sound. There are thus two inevitable sounds in systole; and the introduction of a third cause of sound—collision of blood—were the conditions of its production present (which with Dr. Williams I do not admit to be the case), would not extinguish the other two.

2. I reply to Dr. Leared's question, to which he attaches capital importance—"Why is it that, in anæmia, the *first sound of the heart only*, and the *second never*, becomes replaced by a murmur?"—by disputing the fact. The first sound is *not replaced* under these circumstances, but accompanied by a murmur. I am supposing that Dr. Leared has in mind the basic aortic and pulmonic murmurs common in anæmia; but my statement applies to the cases in which the systolic murmur is apical and mitral.

This fact, if established, will be admitted by Dr. Leared to take much of the ground on which he relies from under him. I repeat, therefore, that a systolic murmur and a first sound may coexist. It is the rule for this to be the case when the murmurs are of anæmic origin; and it is very common in the course of acute rheumatism—not unknown even in chronic disease of the mitral valve. I have long been in the habit of noting the occasional presence of a first sound together with a systolic mitral murmur as a favourable element in prognosis.

It may, perhaps, save trouble to say that I am aware that there are two first sounds, and that I speak distinctly of the coexistence of a murmur and sound both of left-ventricular origin.

I may now put a series of questions. It is possible that some of these may have been answered in Dr. Leared's original paper, but this I have not at hand at the moment. I read it carefully, however, when it first appeared, and rejected his conclusions on account of difficulties which will be found embodied in the questions.

1. Any sound due to collision of the blood set in motion by the ventricles with the stationary blood in the great vessels must be produced at the aortic and pulmonic orifices. How is it, then, that the first sound is better heard over the ventricles, and even at the apex, than at the base and over the great vessels? The first and second sounds would, under Dr. Leared's hypothesis, be originated at the same point, and ought to be heard over the same area.

2. Among the signs of high tension in the systemic arterial system pointed out by my late colleague Dr. Sibson, one is, that the first sound is enfeebled or absent in the aortic area. No one will, I think, question Dr. Sibson's authority on a matter of simple observation such as this, and my personal testimony to the accuracy of his statement is unnecessary. We have, then, in the increased resistance to onward motion in the arterial system, which is the meaning of high arterial tension, and in increased propulsive power in the left ventricle, the concomitant of this—the conditions of an increase in the violence of the collision between the ventricular and aortic blood, and yet the first sound near the seat of this collision, its supposed cause, is enfeebled. Will Dr. Leared explain this?

3. There are two first sounds, usually heard together, but sometimes

* Dr. Hughlings Jackson: Debate following Dr. Dickinson's paper on the Pathology of Choreia (Royal Medical and Chirurgical Society, *Medical Times and Gazette*, 1875, p. 481).

† *St. Bartholomew's Hospital Reports*, 1869, p. 86.

‡ *Medical Times and Gazette*, 1875, p. 482.

§ *Op. cit.*, p. 609.

|| *Medico-Chirurgical Transactions*, vol. lix, 1876, p. 15.

distinct—one produced by the left, the other by the right ventricle. Well recognised differences in character between them are recognised. How does Dr. Leared explain these, if the sounds be the product of collision of the blood, and not of tension of the ventricular walls and valves?

4. The first sound undergoes remarkable modifications—(a) in dilatation of the ventricles; still more (b) in stenosis of the mitral orifice. It is not weaker, and is indeed often louder, than usual; but it is short and sharp, like the second sound. This is easily explained in the case of dilatation, if the ventricular walls and valves are supposed to be the origin of the sound. An explanation on the hypothesis of collision of moving ventricular and standing aortic blood is requested.

5. In dilatation of the ascending aorta, and in aneurism, the aortic second sound is not only louder than in the normal state, but greatly changed in tone. The increased and modified resonance is easily understood, and is, indeed, strikingly suggestive of the change in the walls of the vessel, if the sound result from the tension and vibration of the enlarged area of resonant membrane, but could scarcely be the effect of increase in the size of the column of blood.

NOTES OF A CASE OF CYSTINE CALCULUS DIAGNOSED BEFORE OPERATION.

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THE rare occurrence of cystine calculi, and the few recorded instances of any examination of urine containing these calculi, followed by operation for their removal, have induced me to publish the following notes of a case which came under the care of my father, the late Mr. George Southam.

T. M., a parish sexton, aged 57, married, consulted my father in May 1874, suffering from the usual symptoms of calculus vesicæ. On sounding the bladder, a stone was readily detected; and, on my father's recommendation, he entered the Manchester Royal Infirmary in the following month to undergo an operation for its removal, as he was anxious to obtain relief from the symptoms from which he was then suffering.

Previous History.—The patient was a robust healthy-looking man, and, though in his occupation as sexton he had been exposed to damp and all kinds of weather, he had enjoyed good health all his life, except that, on four different occasions (the first occurring when he was twenty years of age), he has been laid up for a few weeks at a time with attacks of inflammation of the kidneys. On the last occasion, about Christmas 1872, he was confined to bed for nearly six weeks. From this attack he completely recovered, and was quite well up to the following July, when he began to be troubled with a feeling of irritation about the bladder. He consulted a medical man, who examined his urine, but found nothing abnormal. He was told that he was suffering from "liver-complaint", and was treated for some derangement of that organ.

During the following winter, he suffered considerably from symptoms referable to the bladder, and, in the spring of 1874, these increased in severity, becoming so well marked, that he himself became convinced that a calculus was present. He, however, was able to follow his occupation as usual all through the winter, working up to the day of his admission into the hospital. He had never noticed the presence of gravel, or anything abnormal about his urine, except that, during the last few months, blood had been frequently present, especially after much exertion. During the last few winters, he had suffered slightly from bronchitis. He had had six children, three of whom are now alive and healthy, the eldest twenty-six, the youngest ten years of age. One daughter, since dead, when fourteen months old, suffered from an attack of inflammation of the bladder, and passed gravel in the urine for some time. He had one brother and several sisters here all alive and healthy. No other member of his family had ever suffered from calculus, or, as far as he was aware, from any affection of the urinary organs. He was accordingly admitted into the hospital as an in-patient, under my father, on June 15th; and, on examination a few days later, the calculus was again distinctly felt. On this occasion, a lithotrite was used. The diameter of the stone was made out to be a little over one inch. From the dull sound obtained on striking it, it was evident that it was of a soft nature externally; but no suspicion as to its real structure was entertained until a few days later, when, on examination of the urine, I found that it presented the following characteristics. It was of specific gravity 1020, of a pale amber colour, with peculiar aromatic odour; acid; it deposited a whitish sediment

after standing a short time. On heating and on addition of nitric acid, a small quantity of albumen was found to be present. Microscopical examination revealed the presence of numerous crystals mixed with pus-cells and a few red blood-cells; no casts. The crystals were in the form of colourless transparent hexagonal plates, varying in size and thickness; in some cases, forming perfect hexagons; in other cases, having their sides and angles unequal. Mixed with these were a very few prismatic crystals. The hexagonal form of these crystals was characterised by cystine; but, as uric acid is occasionally deposited in the shape of six-sided plates, the following chemical tests were employed to render the diagnosis certain. 1. On the addition of strong nitric or hydrochloric acid, the crystals all disappeared; 2. On the addition of acetic acid, no change took place, except that, after standing some time, the crystals appeared to be more numerous; 3. On the addition of a little nitric acid to some urine in a watch-glass, and allowing it to stand for twelve hours, the hexagonal crystals all disappeared, while numerous crystals of uric acid were present in the form of lozenges, and rhombic tablets of a brownish-yellow colour; 4. On the addition of liquor ammoniæ, the crystals all readily disappeared. Upon spontaneous evaporation of the ammoniacal solution, the cystine again appeared in its original form of six-sided plates. 5. On heating a little urine with liquor potassæ and lead oxide, a dark-coloured precipitate of lead sulphide was formed. This reaction was characteristic of cystine, as indicating the presence of sulphur; but it was here probably also in part due to the sulphur contained in the albumen, which was present in small quantity in the urine. 6. The transparency, absence of colour, and constant shape of the crystals, were also characteristic points as distinguishing from uric acid. 7. After standing forty-eight hours, the urine became quite neutral, with a strong disagreeable odour, and contained numerous crystals of triple phosphate and phosphate of lime. The cystine had almost all disappeared, a few perfect crystals, however, being still present, while others were seen to be gradually dissolving, becoming irregular in shape, with serrated margins. A few hours later, the crystals had all entirely disappeared, and addition of acetic acid was not followed by their reappearance. The examination of the urine, both chemical and microscopical, clearly indicated the presence of a cystine calculus; and, as the bladder was apparently healthy and capable of retaining a considerable quantity of urine, the stone of moderate dimensions, and, being cystine, consequently of soft consistence, the patient strong and of sound constitution, the case was considered one favourable for the performance of lithotripsy.

July 14th. Lithotripsy was performed, the patient having been put under the influence of chloroform. The stone was readily seized, and very little force was required to crush it, showing it to be of soft consistence. This having been repeated three times, the bladder was washed out with a Clover's bottle, several small pieces of calculus coming away at the same time and being caught in the glass receiver. These consisted of a soft yellow waxy-looking substance, having all the appearance of cystine. Microscopical examination showed the presence of the characteristic hexagonal crystals, and thus the diagnosis previously made as to the character of the calculus was at once confirmed. The operation was followed by slight irritability of the bladder, micturition being rendered more frequent and attended with some pain. The urine contained a quantity of detritus, pus, and mucus, but very little blood, and, on the following day, it had become alkaline, with an ammoniacal odour.

July 17th. The patient was a little feverish and perspiring a good deal; suffering from bronchitis, with a feeling of oppression about the chest; cough troublesome and accompanied with a considerable quantity of thick tenacious expectoration. Micturition was still painful, and he complained of pain over the region of the bladder, which was washed out daily.

July 21st. He was better as regarded the bronchitis and cough, expectoration being diminished. There was less irritability about the bladder.

July 24th. Lithotripsy was performed again to-day for the second time. Four pieces of calculus were successively seized and crushed.

July 26th. The second crushing had been followed by considerable relief, the irritation and pain about the bladder being much diminished. It was noticed that his breath had a sweetish odour, like that often present in diabetes; and, on examination of the urine, it was found to contain a slight trace of sugar; the specific gravity was 1012. It was strongly alkaline, with ammoniacal odour; it contained a small quantity of albumen, numerous crystals of triple phosphate, pus-cells, and a few blood-corpuscles. No cystine or casts were present.

July 29th. The bladder was washed out again to-day, a considerable quantity of detritus coming away. He felt much easier since the