in the cabin. All these points might be ensured without any dangerous experiment, and there can be little doubt that the testimony of passengers would soon prove the efficacy of the plan.

CLINICAL LECTURE

ON

A CASE OF CHRONIC BRIGHT'S DISEASE, WITH RAPIDLY FATAL SANGUINEOUS APOPLEXY.

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Rapidly fatal Apoplexy in a Woman of Intemperate Habits.—Copious Hæmorrhage into the Brain.—Small Granular Kidney.—The Left Ventricle of the Heart hypertrophied.—Hypertrophy of the Muscular Walls of the Minute Arteries in the Kidneys, the Pia Mater, the Skin, and the Intestines.

GENTLEMEN,—A few days since there was admitted a typical case of rapidly fatal sanguineous apoplexy in an intemperate woman, who had also been the subject of chronic Bright's disease. I propose now to give you a short account of this case, and to explain to you its pathology.

Mary T., aged 46, was brought to the Hospital at 11.30 P.M. on February 10th. She was perfectly unconscious, and the people who brought her knew but little about her. They were with her in a public-house, where they saw her drink a glass of beer; and they believed that she had previously had some spirits. She was seen to stagger suddenly, and then to fall and to become unconscious. They lifted her up, and brought her to the hospital. She was said to be a single woman, about forty-six years of age. She is believed to have suffered from occasional attacks of giddiness, but no distinct history could be obtained. When seen by the house-physician, Mr. Warner, to whom I am indebted for the notes of the case, she was being supported in a chair, in a state of complete unconsciousness. There was no dropsical swelling. The heart's sounds were normal. There were no marks of external injury. The pupils were of natural and of equal size. When the right eyeball was touched, there was reflex action of the lids; none when the left was touched. The left arm and leg appeared to be perfectly relaxed and powerless, but there was slight resistance when the right arm and leg were flexed or extended. Two slight epileptic convulsions occurred, during which the whole body became rigid. The stomach was emptied by the pump, and a liquid smelling of brandy was withdrawn. She was then sent up to the ward, and placed in bed. She remained unconscious; the breathing gradually became more feeble; and she died quietly at 7 A.M., in rather less than eight hours after the first seizure in the public-house.

On examination after death, we found a very copious hæmorrhage into the brain. A large portion of the right hemisphere, on a level with the lateral ventricle, was excavated by effused blood; the outer margins of the corpus striatum and optic thalamus were partly broken down by the clot; and the blood had passed through the torn septum into the left ventricle. The arteries at the base of the brain had many atheromatous patches in their walls. The kidneys were small and granular; the right weighed $3\frac{1}{2}$ ounces; the left $2\frac{1}{2}$ ounces. The liver was rather pale and fat. The heart weighed $14\frac{1}{2}$ ounces. The walls of the left ventricle were hypertrophied. The valves were all healthy; and the coats of the aorta were not diseased. The lungs presented the engorged appearance which is always found when death has taken place by coma and consequent slow apnoea.

I have here under the microscope a section of the kidney. An examination of this specimen will show you the whole series of changes which occur during the progress of that form of chronic Bright's disease which results in the small red granular kidney. You will see that many of the tubes are opaque, in consequence of being filled with the granular debris of disintegrated epithelium. From other tubes, the

Fig. 1.—Normal artery from the kidney, showing the thickness of the walls in proportion to the canal. In the muscular wall there is an outer circular layer and a thin inner longitudinal layer.—X200.

epithelial particles have been removed; and these tubes, composed only of denuded basement-membrane, have an abnormally transparent appearance. Again, you will find that some of the denuded tubes are smaller than natural, having undergone various stages of atrophy. The minute renal arteries have their muscular walls much hypertro-

phied, presenting the appearance—so constantly found in the hypertrophied renal arteries—of an inner longitudinal layer of fibres equal in thickness to the outer circular layer. You must compare the appearance of a normal artery (Fig. 1) with that of an artery which has undergone this remarkable hypertrophy of its muscular walls (Fig. 2). You will

Fig. 2.—Artery with hypertrophied walls from the kidney. The inner longitudinal layer of fibres is equal in thickness to the outer circular layer. The canal of the vessel is filled with injection.—X200.

see in the section of the kidney no indication of a so-called proliferation of connective tissue between the tubes; but, where there has been much wasting of the gland, the atrophied remains of tubes and capillaries have a fibrous appearance, and the matrix around the wasted tubes seems to be thicker in proportion as the area of its meshes is diminished. The essential changes in this, as in all the forms of chronic Bright's disease, are found in the tubes and in the blood-vessels. The hyperplasia of connective tissue, as the essential anatomical condition of the small granular kidney, is a theoretical fiction, not an anatomical fact, as you may see from a careful inspection of this specimen. You see, too, the very striking changes which the tubes and the arteries have undergone—changes of which the theory in question takes no note, and which it is utterly inadequate to explain.

I have here also, for your inspection, specimens which have been kindly prepared for me by Dr. Kelly, of minute arteries from the pia mater, the skin, and the intestines, of the same subject. You will find that, in all the specimens, the muscular walls of the arteries are conspicuously hypertrophied (Fig. 3).

Fig. 3.—Artery with hypertrophied walls from the subcutaneous tissue. The canal of the artery is injected.—X200.

Now, having given you the morbid anatomy of this case, the pathology, or, as I prefer to say, the physiological interpretation of the facts, is sufficiently simple and intelligible. The patient was a woman of intemperate habits, and a large proportion of the renal gland-cells had been destroyed in the discharge of their functions as eliminators of alcohol. The minute renal arteries, as the regulators of the blood-supply to the gland, resist the entrance of blood in proportion to the destruction of the secreting cells; and this continued excessive action of their muscular fibres results in hypertrophy of their walls. The contraction of the minute renal arteries, in opposition to the blood-stream, when the secreting tissue of the kidney is partially destroyed, is exactly analogous to the arrest of the circulation by the contraction of the minute pulmonary arteries when, from any cause, the respiratory changes are suspended or greatly diminished. As the renal degeneration made progress, the blood became impure, and the physically altered blood excited unusual contraction of the minute arteries throughout the system. Again, this resistance in the minute systemic arteries was met and compensated by more forcible contraction of the left ventricle of the heart; and the result of this continual over-action was hypertrophy of the arterial walls and of the walls of the left ventricle. The amount of hypertrophy is an index of the degree and duration of the excess of muscular contraction during life. One result of this antagonism between the propelling and the regulating forces is to cause increased pressure upon the walls of all the arterial tubes be-

tween the heart and the resisting arterial stopcocks; and the effect of this strain upon the arteries was, in this case, a fatal accident.

We have seen that the larger cerebral arteries had atheromatous walls; they were thus rendered brittle. An arterial branch within the right hemisphere gave way; and then the strong ventricle drove the escaping blood into the brain-tissue with a rapidly fatal effect. We tried to find the seat of the arterial rupture by injecting water into the middle cerebral artery; but, before doing so, a number of vessels had been divided by slicing the brain, and the water escaped in all directions; the broken artery, therefore, could not be found.

The insensibility of the left eye, and the complete relaxation of the arm and leg on the same side, are explained by the large clot in the right hemisphere. The convulsions were probably caused by the irritation of the brain while the tissues were being torn by the escaping blood.

From the time when the cerebral artery gave way, this poor woman's condition was hopeless. When she first came to the hospital, the cause of the coma could not be so certainly known as it now is. Brought in, as she was, by her companions from a public-house, it was probable enough that to alcoholic intoxication her insensible condition might, in part at least, be attributed. It was, therefore, right to pump out of the stomach the alcohol which it still contained. That being done, she was placed in bed, and the symptoms took their inevitably fatal course. The case affords an example of what we often meet with in practice—namely, the coexistence of some degree of alcoholic intoxication with sanguineous apoplexy. When the police meet with cases of this kind in the street, they sometimes make an incomplete, and therefore an erroneous, diagnosis. When a man in a state of more or less complete unconsciousness is found to exhale the odour of strong drink, he is generally believed to be simply drunk, and in a large proportion of cases the diagnosis is correct; but it now and then happens that the drunkard is seized with apoplexy, and the result is an occasional death in a police-cell. In all cases of suspected apoplexy, look carefully for the signs of one-sided palsy or spasm in the eye, the face, and in the extremities; and, in all doubtful cases of this kind, it is better to err on the safe side. It is better to allow a drunken man to recover his senses in the ward of a hospital, than to send an apoplectic patient to die in the prisoner's cell of a police-station.

RUPTURE OF THE MEMBRANES BEFORE THE COMPLETION OF PREGNANCY.

MAURICEAU, in his observations, has recorded sixteen cases, in which a largish discharge of a watery fluid occurred in the later months of pregnancy, at periods varying from seven days to two or three months before the setting in of labour. In the earlier observations, he gives it as his opinion that the discharge comes from *quelque espèce d'hydrométrie de matrice*, for the reasons that when labour set in the patient again discharged a considerable quantity—*ces dernières eaux ayant percé comme à l'ordinaire*; and that if they had been the *véritables eaux de l'enfant*, labour would have set in a short time after their escape. In his later observations, however, he inclines to the opinion that the discharge was *les véritables eaux de l'enfant*, which had escaped from a small rupture in the membranes high up, in such a position that the rupture did not correspond to the internal orifice of the uterus, so that the waters did not entirely escape; and, a good part remaining, labour did not set in for a long time after the first escape of the waters, which renewed themselves in the interval. This change of opinion, for which he gives no reason, is all the more singular from his saying in one place that it is impossible for the ruptured parts ever to reunite. Dr. Carl R. Braun of Vienna, in his *Lehrbuch der Geburtshilfe*, pp. 134-5, amongst other kinds of dropsy of the womb, mentions two which occur during pregnancy, and are characterised by discharges of water before its completion. The first he calls *hydrometra ascitica*, and defines it as a collection of fluid between the chorion and uterine walls. He says further of this, that the discharge of this fluid does not interrupt the course of pregnancy, and that the state can be recognised after withdrawal of the after-birth. The second he calls *Hydrallante*—i.e., "*das falsche Fruchtwasser*", or a collection of fluid between the chorion and amnion. He says of this, that the discharge of the fluid under this condition often interrupts the course of pregnancy, and that it can duly be recognised with precision after the withdrawal of the after-birth, by the separation of the amnion from the chorion. My own experience is at present not very extensive, but I should incline to the opinion expressed by Dr. Radford, that the discharge is never real liquor amnii; and further, that it is always from some dropsical condition of the membranes external to the amnion.

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ABSTRACT OF A CLINICAL LECTURE ON A CASE OF TUMOUR OF THE HEAD OF THE HUMERUS.

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GENTLEMEN,—I wish to-day to direct your attention again to the case of Master C., aged 11, who has lately been under treatment in the wards. The patient's father—who brought me a note from my friend Dr. MacLagan of Berwick—told us, on inquiry, that a swelling was for the first time observed two months ago on his son's left shoulder. The swelling gave rise to no inconvenience, but has slightly increased in size since first noticed.

A careful examination of the swelling made before you on a former occasion taught us that it was a growth from the head of the humerus that it was of the size of a pigeon's egg, hard to the touch, firmly connected to the bone, and completely covered by the deltoid muscle and tissues superficial to this muscle, which with the other soft textures was freely moveable over the tumour. A further examination of the deep connection of the growth showed that the long axis of its neck ran in the direction of the outer margin of the bicipital groove, and passed upwards to a little below the anatomical neck of the bone. The tumour itself seemed slightly to overlap its neck, but owing to the covering of the thick muscle this point could not be very accurately determined. The movements of the joint and arm were perfect.

You will remember that after this examination, I remarked that, having concluded that the case was one of tumour growing from the bone, our next duty was to determine, if possible, the nature of the growth, in order that the proper treatment for it might be considered. The diagnosis to which we came was, that the growth was either an exostosis or a firm cartilaginous tumour; but that it was not easy to determine between these, owing to the thick covering of the deltoid muscle. On this occasion I further remarked, that if the case were one of exostosis, the proper treatment would be to expose the neck of the growth, taking care to avoid any injury to the capsular ligament of the shoulder-joint, and saw through it close to its connection with the bone. [The pathology of a cancellated exostosis was then described in connection with this treatment.] If, however, the tumour should prove to be cartilaginous, a more severe operation would be required, because the pathology of these latter growths teaches us that their structure is almost always infiltrated into the substance of the neighbouring bone, as illustrated in these specimens [preparations shown]. A cartilaginous tumour in this situation might be removed in one of three ways: 1. By enucleation of the tumour; 2. By excision of the head of the humerus, together with the tumour; 3. By amputation at the shoulder-joint. The first of these I did not approve of, as it was a most uncertain method of getting rid of the whole growth; and it could not in this case be carried out without injury to the joint, and therefore it had no advantage over the second plan, which from my experience of two similar cases in the practice of the late Mr. Syme was also somewhat uncertain, unless the shaft of the bone where divided was perfectly free from infiltration. The third plan was not at present to be thought of, as the disease was too limited and too little troublesome to necessitate so serious an operation. It might here be asked, Why interfere with the tumour if it be not causing inconvenience? The reply to this is, that it is a sound principle, with few exceptions, to remove all morbid growths at as early a period of their growth as possible, provided this can be done without danger to life. My proposal, therefore, is to expose the tumour and its connections by means of a free incision carried down through and parallel to the fibres of the deltoid muscle. Should it prove to be an exostosis, its neck will be divided and the growth thus removed; but if the tumour be cartilaginous and infiltrate the surrounding bone, I will excise it together with the head and neck of the bone, carefully examine the shaft of the bone where sawn through, and if it show any traces of disease, remove another slice, so as to take away, if possible, all the disease.

The operation, gentlemen, you saw; and as the growth was a well-marked example of the cancellated exostosis, it was removed alone, without injury to the joint, by dividing its neck, which was nearly two inches in length and an inch in width, and entirely osseous in its structure. The tumour had a thick layer of cartilage on its surface, and was surrounded by a distinct capsule formed of the expanded periosteum. After the operation, the whole surface of the wound was freely touched with Mr. De Morgan's solution of chloride of zinc, and Mr. Lister's anti-