as a sedative, but aids the radical cure of the disease, inasmuch as it causes the subsequently administered ferruginous preparations to be borne more easily. Both general and local blood-lettings may be used; but the blood must be taken in small quantities, a couple of ounces at a time being quite sufficient."

In such a case, you should diminish or omit the iron for a time and use morphia or bromide of potassium, with chloral, or valerian, or stimulants in small quantities. Instead of seeking to increase the quantity of the menstrual discharge, you will rather aim at diminishing it; particularly in cases where it keeps up in quantity whilst the patient is becoming more and more chlorotic. This diminution we may effect by keeping the patient in bed or on a couch for a couple of days before menstruation begins, and also whilst it is in progress; giving, at the same time, some bromide of potassium till the period is half over, and then using extract of ergot. Digitalis in some cases checks the flow, and this may be used instead of ergot when this drug seems to have no effect.

REMARKS ON A CASE OF BRIGHT'S DISEASE, ATTENDED WITH CARDIAC THROMBOSIS AND HÆMORRHAGIC INFARCTIONS OF THE SPLEEN, LEFT KIDNEY, AND LUNGS.*

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IF I were asked to enumerate those diseases which are of most interest to the pathologist and clinical physician, I should certainly include Bright's disease in that category; for every fresh case that comes under our notice offers us some new lesson—shall I say some new problem for solution?

The following case has proved a very instructive one to myself; and my object in bringing it before my professional brethren is to ascertain from them whether the explanation I propound of the causes and sequence of the complications which arose towards the termination of the case meets with their acquiescence.

I will read the notes which have been preserved of the case.

William Layton, aged 43, stonemason, living at Cambridge, was admitted into Addenbrooke's Hospital, under my care, April 16, 1872. He was a well-built man, his cheeks were a little dusky, and the capillaries on them were enlarged. He had been intemperate. On admission, he was unable to lie down in bed; the respiration was wheezing, rapid, and laborious. He at times perspired a great deal.

The following was elicited from him with regard to his previous history. Six years ago he had an attack of acute rheumatism; four years after that he had another; one year ago, he had a third attack; and last Christmas he was ill with the same disease for the fourth time. Usually his ankles, shoulders, arms, and back, were painful. He was thirsty during each attack, but says he never laid in bed more than half a day at once. At times his breath had been short for the last six years—*i.e.*, ever since he had the first attack of rheumatism. Since Christmas he had been an out-patient of the hospital. His feet were swelled, and his breath was short. He had pain in his back, shoulders, and arms. About two months ago he was much better, but five weeks ago his breathing and the swelling of the legs became as bad as ever, but the only pains he had were flying pains about his back.

On the night of his admission, he complained of pain on the right side over the lowest rib. His legs were swollen, pitting considerably on pressure. Temperature 98.2; respirations 52; pulse 96, in the minute.

Physical Signs.—On the left side in front there were prolonged expiration and medium-sized crepitation. Percussion was clear down to the upper border of the fourth rib. Behind at the left base there was largish crepitation; percussion was good. At the right base, posteriorly, there was rather finer crepitation, but no dulness on percussion. The heart's action was extremely irregular, but not very quick. No bruit could be heard, but a slight friction-sound was audible over the apex.

The urine was acid; its specific gravity 1033; and it was loaded with buff-coloured lithates. It was very scanty, and contained a large quantity of albumen, becoming almost solid with heat and nitric acid. It contained granular casts. He was ordered a slice of meat and bread for diet, with three ounces of gin; and the following draught to be taken four times a day.

℞ Potassæ nitratis gr. xv; spts. ætheris nitrosi ℥ss; tinct. scillæ mxx; decoct. scoparii ad ℥iss. M.

* Read before the Cambridgeshire and Huntingdonshire and East Anglian Branches, at Ely, on June 28th, 1872.

April 18.—The sputa for the first time were in parts uniformly dull red, like the sputa of pulmonary apoplexy. In other places the sputa were streaked with blood and tenacious. The patient passed very little urine.

April 19.—Sanguineous sputa still continued. The left eyelid drooped.

April 21.—Numerous granular casts were found in the urine—one with about ten fat-globules on it. The sputa still contained blood. The pulse was very feeble, that in the right wrist being the stronger of the two radial pulses. The hands were purple and cold. The pupils were equal. I ordered him to be cupped to the extent of five ounces; but the House-Physician could only obtain three-and-a-half ounces; the blood was dark and very coagulable, and would not flow freely.

April 23.—He passed a better night. His breathing was easier. No sanguineous sputa were expectorated to-day. He had wandered a little at times during the last four days. Two ounces of blood were removed by cupping over the chest behind. The percussion-note was equal on the two sides. There was crepitation on expiration, especially on the left side; the crepitation being larger at both bases. Respiration was shallow. The cardiac sounds were scarcely audible. There appeared to be no increase of cardiac dulness.

April 22.—He still wandered at times, and was very sleepy. There was no expectation.

April 23.—He was still very drowsy. He had wetted his bed. A few gangrenous spots were visible on the back of the right hand. On the evening of this day an attempt was made to examine the eyes with the ophthalmoscope, but the patient became violently delirious.

On the evening of the 25th his hands were warmer. His mind still wandered, but he was quiet in other respects. He died in a comatose state at 9 A.M. on April 26th.

POST MORTEM EXAMINATION, April 27th, twenty-five hours and a half after death.—There was a considerable amount of yellow serum in the right pleural cavity. The left pleura was adherent in front and at the base. The amount of fluid in the pericardium was normal. On the front of the heart, near the apex, there was a patch of shaggy lymph, about three-fourths of an inch in diameter. The heart was enlarged, and there was a superficial deposit of fat. The muscular tissue was softer than natural. The wall of the left ventricle was much thickened, and the cavity enlarged. The free edge of the mitral valve was a little thickened. The left auricular appendage contained an *ante mortem* clot, which was breaking down in the centre into a granular debris. The right ventricle contained a large *ante mortem* clot, which extended through the right auricular-ventricular opening into the right auricle. There was little, if any, hypertrophy of the right side of the heart. The right kidney was congested and slightly enlarged; the left was also in the same condition. In the left kidney, small hæmorrhagic infarctions were seen, both on the surface and on making a section. The capsule of the left kidney was adherent, and the surface rather finely granular. Granular casts were squeezed out of a cut portion. There were no hæmorrhagic infarctions in the right kidney. In the middle and lower lobes of the right lung there were patches of circumscribed pulmonary apoplexy. In the lower lobe of the left lung there was diffused apoplexy. In the spleen, there was a yellowish-white wedge-shaped fibrinous mass, the result of an infarction. It appeared to be of some standing. The liver was nutmeggy.

REMARKS.—There can be little doubt but that the starting point of the mischief in this case was rheumatism; and it is highly probable that the patient increased his tendency to this disease by his intemperate habits. Rheumatism and beer-drinking in excess are acknowledged to be two most important factors in the genesis of Bright's disease; and, in my opinion, they laid the foundation of kidney-disease in my patient. Of what duration the renal affection was, I am unable to say, as the patient was not under my care previously to his admission as in-patient.

You are all doubtless aware that it has been shown by Dr. George Johnson that the muscular coat of the small arteries throughout the body becomes hypertrophied in cases of chronic Bright's disease, and that this hypertrophy is due to these blood-vessels contracting so as to resist the flow of impure blood, or, in other words, of blood charged with urea, and deficient in its normal constituents, to the tissues, it being one of the functions of the small arteries to regulate the amount of blood supplied to an organ or tissue. In consequence of these blood-vessels impeding the onward flow of the blood, the left ventricle of the heart has to act with increased energy. We have then the small arteries opposing the onward flow of the blood, and the left ventricle of the heart endeavouring to overcome this obstacle, and, as a result of the increased work thrown upon the latter, it becomes hypertrophied.

What happens next? It is not difficult to see that, as a result of what I have just described, the blood-current must become slower;

and two conditions most favourable for the formation of clots in the heart are present; viz., a rough surface, furnished by the trabecular work of that organ, and retardation of the blood-current. Of all parts of the heart, the appendages of the auricles are the most frequent seats of thrombi, owing to the blood being with great difficulty completely expelled from them; and in the notes of the necropsy of the case, it is stated that the left auricular appendage contained an *ante mortem* clot, which must have been of some age, as it had undergone softening in the centre. In consequence of the thrombus in the left auricle breaking down into minute puriform particles, the latter became mixed with the blood which flowed into the left ventricle, and were sent during its systole into the systemic circulation. When these particles passed into vessels which were too minute to allow them to escape, they more or less completely plugged those vessels, and in this manner it is extremely probable that the splenic and renal infarctions were produced, as also the gangrenous spots on the back of the right hand. The peculiar wedge-shape of the splenic infarct, with the apex of the wedge towards the centre of the viscus, was due to the mode of distribution of the vessels, and its yellowish-white appearance to the colouring matter of the blood having been withdrawn from it, or to the decomposition of the hæmatine, and to the fibrine having undergone fatty degeneration.

The kidneys were larger than those generally met with in granular degeneration of these organs; but whether this condition, and the large quantity of albumen which the urine contained during life, were partly produced by the renal infarctions, I am unable to say.

I have now endeavoured to account for the hypertrophy of the left ventricle, the *ante mortem* coagula in the heart, and the hæmorrhagic infarctions of the spleen and kidney. I now proceed to consider the mode in which the pulmonary apoplexy was produced.

It has been shown by Virchow and others that, in all cases of pulmonary apoplexy, whether secondary to valvular disease of the heart or not, the first step in the genesis of the morbid change is embolism of a minute branch or branches of the pulmonary artery. Virchow has proved this experimentally, by introducing particles of fibrine, muscle, and elder-pith into the jugular veins of dogs, and examining the lungs after death, when he found that these foreign bodies had blocked up branches of the pulmonary artery, and produced, among other results, hæmorrhagic infarctions beyond the points obstructed. You will remember that, on *post mortem* examination, *ante mortem* coagula were stated to have been found both in the right auricle and ventricle; in fact, the thrombus from the auricle extended through the auriculo-ventricular opening into the ventricle. Well, it is my opinion that minute portions of these clots were washed into the pulmonary circulation, just as the granular matter found its way into the systemic circulation from the left side of the heart; and I would here remark that the circulation through the right side of the heart was sufficiently vigorous for the blood to be sent to the minutest ramifications of the pulmonary artery. When, therefore, minute portions of these clots arrived at pulmonary arterioles too minute to allow them an exit, they produced embolism and subsequent extravasation of blood.

The mode in which the hæmorrhage is produced subsequently to the plugging of the vessel is variously interpreted; but the theory of Ludwig, founded on the "influence which the contraction of an artery has upon its capillaries", appears to me to be by far the most feasible explanation. I will quote his words from the American translation of the seventh edition of Niemeyer's *Text Book of Practical Medicine*, vol. i, p. 156: "Tension within the artery below the point of constriction is diminished, since a liquid flowing through a narrow tube loses more of its impetus than in flowing through a wide one. But we must not infer from this that, when an artery is constricted, the contents of its capillaries are lessened, and that the parts which they traverse grow paler. The sluggishness of the stream thus produced in the capillaries rather has the effect of allowing the heavy blood-corpuscles to collect and become crowded together; now, as two or more blood-corpuscles, if brought into contact, are apt to become permanently adherent, the blood itself can form a plug capable of closing the capillaries. Such an occurrence, which converts the capillaries into blind appendices to the artery, must cause an increase in its internal pressure." "Let us add," says Niemeyer, "that, in consequence of the pressure, which, after the stoppage, is as severe in the capillaries with their delicate walls, as in their afferent vessels, a rupture of the distended walls and an escape of blood ensue..... The utmost that can be advanced against the accuracy of this explanation is, that the artery leading to an infarction not only is narrowed, but entirely closed. Such an objection, however, is untenable. The infarction of an embolus, which we find usually takes place at the bifurcation of an arteriole, very rarely produces absolute closure of it at first, but merely causes in it more or less obstruction. Afterwards when the infarction

has become established—an event requiring but little time—fibrine is deposited upon the embolus, and closure of the vessel becomes complete."

There is only one other point on which I will say a few words, and that is connected with the treatment of the case. A day or two after the patient was admitted, and when I was going round the wards with my pupils, I found the man's face of a purplish hue and bathed with perspiration; he was gasping for breath, and the pulse at the wrist could scarcely be felt. His extremities were cold. He was also expectorating bloody sputa. The heart's sounds were scarcely audible. What was I to do under these circumstances? One thing was quite certain; viz., that the man would die unless I could speedily relieve him. It occurred to me that the immediate cause of his distress was a surcharging of the right cavities of the heart, producing a state approaching to paralysis of that organ, and that it was just possible that cupping might relieve him. On the other hand, I had to contend with this difficulty; viz., that if I cupped him I should increase the coagulating power of the blood, and thereby the tendency to the formation of clots. I, however, asked the House-Physician to cup him, and the result was most satisfactory, for, after the operation, he was able to lie down in bed, and breathed more easily. The patient expressed himself as feeling decidedly relieved. He lived four days longer.

THE SPECIFICITY OF ACUTE DISEASES.*

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MORPHOLOGY and physiology are so intimately connected together in all organic beings, that it is doubtful (as Mr. H. Spencer has shown) whether any functional change can take place in an organism without a corresponding alteration of its structure; and modern science would lead us to assume that, whenever we find a change in the vital operations of an organ, there is, or has been, a corresponding change in its textures, whether this be apparent or not. Starting with such a doctrine, it is easy to perceive how the various phenomena accompanying the loss of health have been divided and subdivided according to the method of their serial development, and how the present system of classification of diseases has arisen. It appears to me, however, that there now reigns a large amount of confusion on the subject of the differentiation of diseases, from our knowledge being based rather upon hypotheses than on facts. What I mean by this statement, I now hope to explain by reference to the so-called specific diseases. Such diseases are supposed to originate in the system by the introduction of a "material principle, the product of a specific morbid process" (*Traité de la Contagion*, par Dr. Anglada, Paris, 1853, t. ii, p. 12), and many clever and able writers have upheld this doctrine. The latest investigations of Chauveau, Sanderson, Bastian, and others have failed to show that there exist any specific elements in the product of such lesions which differ in form and external characters from analogous elements met with in simple inflammatory lesions. Therefore, if there be a difference in the two (to quote M. Chauveau's words) "it must be in their intimate qualities and active properties." Our only means of investigating such "intimate qualities and active properties" is to observe the results of the introduction of such specific agents into the animal economy, and to see whether there be an agreement between the phenomena thus produced; and, if so, whether it be only general or differing with different agents. Such investigations can only be obtained by actual experiment; and, as we are mostly debarred from making such on the human subject, and experiments on the lower animals are open to many great objections, it will probably be a long time before this difficulty is solved. There is, however, one source of information open to us on this subject; and that is the process of vaccination. If it can be shown that on the introduction of vaccine into the human frame a progressive series of phenomena arises, definite and similar in all cases, and yet that this series invariably differs from all others induced by other agents, we shall have a strong argument in favour of the specificity of diseases in general. If, on the other hand, no changes take place after vaccination, which differ from such as may be induced by inoculating the system with another organic poison, then the hypothesis of specificity (as far as vaccination is concerned) is not yet proven. Before we consider the process of vaccination, however, it may be as well to review the subject of inflammatory skin-diseases generally, and to ascertain in what way, if in any, the typical vaccine vesicle differs from all other skin-eruptions. In tracing the course of hyperæmic or inflammatory skin-affections, we cannot

* Read before the Abernethian Society.