may therefore become so slow in the particular part as to allow of the formation of a small coagulum of fibrin or a thrombosis; there would then be complete obstruction, and consequently the greatest possible pressure would be brought to bear on the arterial wall, and result very probably in rupture. In this way not only may cerebral apoplexy be accounted for, but pulmonary apoplexy and apoplexies in the kidneys and spleen, or hæmorrhagic infarctions, occurring in cases where no endocardiac or valvular mischief has existed. The same explanation may be offered with respect to oedema of the integument and dropsy, as is given for oedema of the lungs. A characteristic of the oedema in Bright's disease is that it changes its position, now attacking one organ, now another. How is this? And why does the morbidly altered blood cause contraction of the minute arteries; is it by direct contact with the vessel, or is this the result of the action of the blood on the ganglionic cells of the nervous system? These are interesting questions still requiring explanation.

THE PATHOLOGY OF BRIGHT'S DISEASE.

By GEORGE JOHNSON, M.D., F.R.S.,

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SIR WM. GULL and Dr. Sutton, in the short note which appears in the last number of the JOURNAL (p. 654), state that the vessels from the pia mater of the sheep in which they recognised the hyaline thickening of the tunica adventitia had been acted on by acetic acid. In this statement they have inadvertently erred. The vessels in question had been immersed only in a mixture of equal parts of glycerine and diluted naphtha (1 part naphtha to 9 of water). I have specimens in which glycerine alone, and others in which equal parts of water and glycerine, may be seen to have produced precisely the same hyaline thickening of the tunica adventitia in perfectly normal arteries. In each of these cases, the glycerine is, without doubt, the agent which effects the hyaline change in the fibrous tunic. It will be remembered that the specimens of Sir Wm. Gull and Dr. Sutton are mounted in a mixture of glycerine and camphor-water.

I have referred to the action of dilute acetic acid, only to show the extreme rapidity with which it produces a hyaline swelling of the adventitious tunic, not distinguishable from that which is more gradually effected by glycerine. At the *conferenza* of the Royal Medical and Chirurgical Society, I showed a specimen thus acted on by acetic acid with glycerine, and another by a mixture of glycerine and naphtha-water; the two specimens having precisely the same appearance. The fact that this appearance of the tunica adventitia can be produced by various fluids acting upon quite normal arteries, is scarcely consistent with the theory of "arterio-capillary fibrosis."

Without a detailed history of each case and of each specimen, I cannot undertake to say why it has happened that Sir Wm. Gull and Dr. Sutton have found that the vessels from certain subjects presented none of these changes, although prepared in the same way. The imbibition of fluid by the fibrous tunic, upon which the appearances in question depend, is doubtless influenced by various physical conditions not easily determined. It was stated in the paper that the changes which the authors describe are often very partial; the trunk of a vessel may be affected while the branches are free. So when the vessels from a healthy animal have been acted on by glycerine, some arteries may be seen quite unchanged in the midst of others conspicuously hyaline.

That Sir Wm. Gull and Dr. Sutton have met with these hyaline changes in healthy arteries, is a reasonable inference from their statement that they have found them unassociated with renal disease. Upon this observation they chiefly base their doctrine that a general arterio-capillary degeneration precedes the local disease in the kidney. But it is obvious that, if my interpretation of the "hyaline fibroid" appearance is correct, this affords no evidence of a pathological change in the blood-vessels preceding the renal degeneration.

Dr. Moxon reports, in the last number of the JOURNAL (p. 637), a case of extreme granular degeneration of the kidneys without hypertrophy of the heart; and he maintains that the occurrence of such a case is inconsistent with the view that the cardiac hypertrophy, which is so commonly associated with the contracted kidneys, is a result of the blood-changes and the consequent impediment to the circulation induced by the renal disease. I confess that I fail to see the force of this argument. By a similar process of reasoning, if a case of Bright's disease with a large white kidney terminate fatally, as such cases sometimes do, without the occurrence of dropsy at any period of the history, we might conclude that the dropsy which is frequently associated with this form of disease is not a result of the blood-changes induced by

degeneration of the kidney, but a consequence of some other unknown cause usually, but not constantly, associated with the renal disease.

It is an acknowledged fact that, while the hypertrophy of the left ventricle is found, as a rule, in all cases of contracted kidney, it is less constantly, though not seldom, associated with the large white kidney. Why is this? In the first place, it is a fact that the hypertrophy of the left ventricle is always associated with hypertrophy of all the systemic arterioles. With the contracted kidney, there is, as a rule, a free secretion of water with retention of urinary solids; and this uræmic condition is, in various ways, the cause of death. On the other hand, in cases of large white kidney, there is a scanty secretion of water by the kidneys, and, in consequence, a state of hydræmia and dropsy. In the first class of cases, the urinary solids accumulating in the blood probably excite the contraction of the systemic arterioles, and thus indirectly provoke the hypertrophy of the left ventricle. In the second class of cases, while there is some retention of urinary solids, there is a disproportionate accumulation of water, by which the contaminated blood is diluted, and thus rendered less irritating to the minute arteries. Hence it happens that the arterial resistance is less, and the cardiac hypertrophy less constant and pronounced in cases of large white kidney with consequent hydræmia, than in cases of contracted kidney with resulting uræmia.

THE ANATOMY OF BRIGHT'S DISEASE.

By PAUL W. SWAIN, F.R.C.S., Stoke, Devonport.

I WAS much interested in the paper and discussion on Sir W. Gull's views regarding the morbid changes produced by Bright's disease. I have had the good fortune to inspect carefully the specimens of hypertrophied capillaries shown by Dr. G. Johnson; and, as I have been in the habit of using one of Powell and Leland's best microscopes since the year 1844, I may lay claim to some experience in judging of the truth and value of microscopic demonstrations. I therefore trust that a few observations may not be unacceptable to many who may not have had the same opportunities of studying minute pathological preparations.

The action of the arterioles and capillaries causes the intermittent stream of blood, as delivered from the heart into the large pulsating arteries, to become a steady uniform current through the minute channels, as may be seen in the web of the frog's foot. The power thus possessed by the small vessels is derived from their muscular elastic coat. This power may be well illustrated by a reference to the India-rubber instrument used in producing the æther-spray. The action of the hand is analogous to the contractions and relaxations of the heart; and the action of the second elastic bulb resembles the effect of the capillaries in causing the current of the spray to be uniform and continuous. Of course, the elasticity of the air must be taken into account in the production of the spray; but the analogy is sufficiently complete for illustration.

Now, I do not mean for a moment to set up ingenuity of theory against absolute and undeniable facts; but I do say that, if we accept Sir William Gull's idea of a fibro-hyalin deposit, substituting an inert obstructive material for an active conservative force, we demolish what appears to me one of the most beautiful illustrations of design which, to my understanding, appears to rest on evidence as sound in logic as it is in fact. The morbid changes and their causes may be briefly summarised.

The kidney is no longer able to filter the blood and withdraw from it the nitrogenous and deleterious matters. When it reaches the capillaries thus deteriorated, it irritates the minute nerve-fibres spread over their internal lining; reflex action causes the muscular envelope to contract, thus reducing the calibre of the tube; the heart insists on forcing on the circulation, and thus at each end of the chain increased muscular action is induced, gradually but certainly causing muscular hypertrophy. To this capillary resistance Dr. George Johnson has very appropriately applied the term "stopcock" action; and there can be no doubt that the whole process is one of those wonderful efforts of Nature to make the best of a bad matter, and thus to prolong the life of the individual by setting up what is actually an abnormal proceeding. In the present subject of discussion, the question has been confined to instances selected from Bright's disease, in which it is probable that the starting cause of the muscular hypertrophy would be in the capillaries. But there are conditions of hypertrophy of the left ventricle which may be called endocardiac, and which are known to begin in the heart itself. Dr. Houghton's examinations of the enormous heart of the greyhound Master Magrath, and his reference to the size of the heart of Eclipse, led him to suggest that hypertrophy of this organ might not be always absolutely a morbid condition, but be produced, like the blacksmith's arm, by increased action, and actually having for its aim the adjusting the organ