lieved" (*loc. cit.*, p. 42); and this, so far as it goes, may be accepted. But who can decide what and how much a "rational person" may believe? Some people are credulous and some are sceptical; some believe only in themselves, while some place implicit confidence in certain statements that come to them with a special kind of authority. There are "rational" people who believe many things that others consider absolutely irrational, and which they utterly discredit. The things, the propositions, the notions of right and wrong, that were received as true by masses of educated persons a hundred years ago, and in this country, are, many of them, rightly or wrongly, utterly repudiated now. There is change in regard of credible things; there is addition and elimination; and we believe that there is growth, and that "the thoughts of men are widened by the process of the suns". Some of us believe that we are wiser than our fathers were, and know more than our fathers did; but some think that we are now following only the cunningly devised fables of science, and that we should do well to swallow a few large camels in order that we might not waste energy in straining at insignificant gnats. But with regard to delusions, it is obvious that their range is wide; and that, with respect to some alleged delusions, it is simply impossible to bring them into contact with fact. For example, regarding all those morbid ideas which have to do with the feelings of misery that some lunatics entertain with regard to the course of their own thoughts—that their imaginations, wishes, and purposes, have been wicked to a degree that is beyond that of other human beings—who can say when and at what point this feeling of personal sinfulness is morbid, and when and where it is, according to the belief of a large number of good people, and in conformity with certain printed books, and certain texts of very high authority, but the most hopeful sign of the commencement of a religious life? Very often, however, some feelings of this kind have passed on into the condition of melancholia, or of mania, or of some other form of declared insanity. Again, a common class of delusion is that with regard to the affections—esteem and confidence of others; but who in every instance can measure the value of the ground upon which these alleged delusive notions rest? In the presence of any third person, A may treat B with all the kindness and consideration that B requires; but when alone, there may be an altered manner, a silence, a tone, a look, a gesture, that shall be quite enough to account for the notion that all is not as it used to be; enough sometimes to convert a so-called delusion into a very real and disagreeable fact. When a man says that he is Julius Caesar, or possessed of countless millions of money; when he affirms that his legs are made of glass or his head of butter; that he is the Devil impersonate, or the Holy Trinity, there can be no hesitation as to the mode of treatment (both social and individual) that he requires. If he really believe his statements, he has a delusion, and is undoubtedly insane; but when his, as it appears to others, unreasonable idea or delusion is, that his wife is unfaithful to him, or his son unkind, or that some friends have altered their feelings to him, since his condition, it may be, has changed from prosperity to adversity, who can tell how much truth there may be in all these assertions? or, if no real truth, how much there may have been which should justify, in one depressed by circumstances, the ideas and the feelings he had entertained?

I do not for a moment wish to underrate the occasional value of delusion as an indication of insanity; but I reaffirm that it is not always present, and that, when present, its value, as a sign, is of very variable amount, and may be nothing.

ON SOME OF THE SYMPTOMS PRODUCED BY URÆMIC POISONING IN CHRONIC BRIGHT'S DISEASE.*

By P. W. LATHAM, M.D., F.R.C.P.,

Physician to Addenbrooke's Hospital, Cambridge; Deputy to the Downing Professor of Medicine; etc.

[AFTER referring to the importance of being able clearly to understand how the various symptoms in chronic Bright's disease are produced, Dr. Latham proceeded as follows.] It is to Dr. George Johnson that the merit is due of having discovered the true cause of one of the complications of this disorder, viz., the production of hypertrophy of the heart—hypertrophy, that is, without valvular defect, and more especially of the left ventricle. He has pointed out that in chronic Bright's disease the morbidly altered blood is impeded in its passage through the minute arteries not only of the kidneys, but of the system; the impediment being caused by excessive contraction of the muscular walls of these vessels. This contraction in time develops hypertrophy of

the muscular coat, the existence of which can be demonstrated after death, when the minute arteries are examined under the microscope. As the tonic contraction of the small arteries opposes the passage of the blood, greater force is required to drive it onwards; and so the hypertrophy of the left ventricle is developed by the resistance offered to the circulation. The explanation is so simple and satisfactory as to carry conviction with it.

Now it appears to me that this excessive contraction of the small arteries in different organs will help us to understand several of the other complications which arise in Bright's disease, the endeavour to explain which has led to so much discussion. I will discuss first that paroxysmal dyspnoea or asthma which sometimes shows itself. I take this first, partly because, by means of auscultation and percussion, we can tell more readily what is actually going on in the lungs than in other organs; and partly because a very striking instance (which I shall presently relate) of this form of disorder came under my notice at the early part of the year, and led me to make the remarks with which I am now troubling you.

Let us consider what would be the result if excessive contraction of the minute branches of the pulmonary artery took place. There would be impeded circulation of blood to the pulmonary capillaries; and, though air would be freely admitted into the air-cells of the lungs, the patient would experience "want of breath": he would, in fact, be much in the same state as if he had embolism of the pulmonary artery, except that the obstruction, instead of being at the commencement of the artery, would be at its ultimate ramifications. At the same time, the heart tries with increased force to drive the blood onwards; there is increased pressure upon the minute branches of the pulmonary artery; and partly from this cause, and partly from the deficient amount of albumen in the blood, the serum filters through the minute vessels into the tissue and air-vesicles, oedema of the lung is developed, and thus in a secondary manner the dyspnoea is increased.

By what general and physical signs would such a state of things as I have here described be recognised? First of all, from the impediment to the aëration of the blood, the patient would have dyspnoea, or rather, as he requires free action of the diaphragm, orthopnoea. There would be a certain amount of blueness of the lips and skin; and, as the serum filters through the tissue into the air-cells and finer bronchi, coughing, with copious frothy and serous expectoration, would take place. With the expectoration of large quantities of serous sputa, there may be in some degree vicarious elimination of the abnormal constituent of the blood; or, during the paroxysm of dyspnoea, the amount of the abnormal constituent may be lessened by the action of the kidneys. The minute arteries would then be no longer so strongly excited to contract, and the dyspnoea for a time would subside.

On listening to the breathing of a patient under these circumstances, we should expect to hear the expiratory murmur generally over the lungs, but mixed up with moist râles; and to find the percussion-note normal, unless the serous fluid have driven all the air out of the air-cells, in which case we should find in the most dependent portions of the lung no respiratory murmur, and the percussion-note dull.

Now, at the time when I was considering these points, a particularly interesting case was recorded in the BRITISH MEDICAL JOURNAL for March 30th, by that very accurate observer and accomplished physician, Dr. Gairdner of Glasgow. It so well exemplifies the view which I have been led to adopt with regard to this paroxysmal asthma, that I am very glad to avail myself of the case as an illustration. The particulars are not recorded as having any bearing whatever on the condition of the minute arteries, but on account of the appearance and disappearance of certain cardiac murmurs. With that portion I shall not trouble you. I wish merely to direct your attention to the symptoms which the patient presented when he first came under observation, and which you will see, as I read the account, presented some difficulty as regards their explanation.

"J. D., aged 35, a hairdresser, was admitted to the Glasgow Royal Infirmary on January 8th, 1872. The description of his symptoms, which was recorded in presence of the clinical class on January 15th, was in many points that of a case of spasmodic asthma with nocturnal exacerbations, attended by wheezing and coughing, marked orthopnoea, and expectoration of frothy mucus, leading on to the end of the paroxysm. Notwithstanding a certain amount of dullness on percussion at both bases, the state of the respiratory murmur gave no sufficient indication of the cause of the distress; the urine was albuminous, of specific gravity 1010-1012, acid, pale yellow, with hyaline tube-casts and a few blood-corpuscles. There was a moderate amount of dropsy of the limbs, and the complexion bore strong traces of the anæmia of renal disease. The physical examination of the heart led to no very clear inference as to its condition—a rumbling prolongation of the first sound, which could not be definitely pronounced either exocardial or

endocardial, being the leading fact observed ; while the symptoms were, in Dr. Gairdner's opinion, open to the interpretation of being, at least in part, of cardiac origin, yet without the possibility of excluding other factors from an important, perhaps a primary, share in their production. It was particularly observed that there was no specially cardiac type of pain—indeed, little characteristic pain, properly so called, of any kind ; but, on the other hand, the very strikingly marked thoracic oppression and orthopnoea gave to the obviously imperfect descriptions by the patient somewhat of the tone of that peculiar and indescribable suffering which Dr. Gairdner is in the habit of calling *angina sine dolore*, from its association with the most terrible aspect of the true *angina pectoris* ; viz., the dread of instant death."

If now we compare these symptoms with those which theory (on the supposition of excessive contraction of the minute branches of the pulmonary artery) would lead us to expect, we see how closely they correspond. The thoracic oppression, the orthopnoea with exacerbations, cough, expectoration of frothy mucus, the character of the respiratory murmur, the dulness on percussion at both bases, all agree ; and therefore the symptoms are to be explained, I venture to think, by the stopcock action set up in the minute branches of the pulmonary artery.

I have so far made no reference to the bronchial artery. If its minute branches were simultaneously affected, of course there would be increased discharge from the lungs ; but the paroxysmal dyspnoea can only be explained by the affection of the pulmonary artery. There appears, moreover, to be a reason why the branches of the pulmonary artery should be affected more strongly than the bronchial. If we assume, with Frerichs, that in Bright's disease the toxic symptoms are caused, not by urea, but by carbonate of ammonia, into which it is converted—if, I say, we assume this, and that the minute arteries contract from direct contact with the morbid blood—then, as this fluid passes through the pulmonary capillaries, a certain quantity of the carbonate of ammonia is exhaled from the lungs, giving the breath a peculiar ammoniacal urinous odour ; and so, when the blood passes to the left side of the heart, it contains less of the noxious agent than before, is therefore less irritant, and would consequently, on reaching the minute branches of the bronchial artery, be less liable to excite their contraction.

The preceding remarks were, as previously stated, really suggested to my mind by a case to which I was called in consultation at the early part of the year, and of which I will now give you the particulars.

On Tuesday, February 20th, 1872, I was requested to see a brewer's foreman, J. H., aged 64. I found him sitting up in bed, gasping for breath, with a most anxious expression and great dread of impending suffocation. His complexion, lips, and skin were bluish—the last cool ; pulse 84, of moderate volume, and rather hard. The tongue was moist, slightly furred on the dorsum, with a bluish tinge. The bowels were regular, from medicine. The urine was reported to be scanty, high coloured, with a copious deposit of lithates on cooling. The respirations were hurried, 42. There were very decided orthopnoea with paroxysmal exacerbations, frequent cough, and abundant expectoration of white foaming serous sputa. He was otherwise free from pain. I was told that for three or four years he had suffered from cough and expectoration during the winter ; which ceased, however, to trouble him each year as the warm weather set in. During the last winter, the cough and expectoration had been more troublesome, and associated with shortness of breath of a spasmodic character when he exerted himself. Latterly there had been nocturnal attacks of dyspnoea, obliging him, from the feeling of suffocation, to jump out of bed ; and, after gasping some time, and "as soon as he got up some phlegm, he was easier." Although "the cold mornings were much against him," he had, until the Saturday previous to my visit, been able to attend to his duties.

On this Saturday the dyspnoea had become more urgent, and the paroxysmal attacks very frequent, recurring at times once or twice or oftener in the hour ; from that date he had been unable to lie down ; nor had he slept for more than a minute or two at a time, as directly he began to doze the feeling of suffocation startled him, and he jumped out of bed almost before he knew what he was about. During the paroxysms the face became very dark. On examining the chest, auscultation and percussion gave the usual signs of a somewhat hypertrophied heart with the apex depressed, but no sound of valvular mischief ; the valves were perfect. On listening to the lungs, the respiratory murmur could be heard distinctly all over the chest except quite at the posterior bases, where it was somewhat obscured, and moist *râles* were everywhere audible, having a finer character towards the lower portion of the lungs than elsewhere. The percussion-note, excepting the increased extent of præcordial dulness, was everywhere normal. There was, therefore, no emphysema, nor were either the symptoms or the auscultatory signs quite those of

spasmodic asthma. Considerable doubt was felt about the diagnosis. Obstruction in the pulmonary artery suggested itself, but seemed unintelligible, and, therefore, considering the spasmodic character of the disorder and the relief afforded by free expectoration, the patient was ordered bromide of potassium and ipecacuanha. There was no uneasiness in the region of the kidneys, nor had there been any swelling whatever of the face or ankles, but seven years before, the man had suffered from severe renal colic, and had voided by the urethra two small uric acid calculi. I saw him at the time, and then the urine was albuminous, but after the voidance of the calculi it became perfectly healthy and normal in character. On examining a sample, however, which was sent to my house the next day, I found it albuminous, acid, and containing hyaline and granular casts. On again visiting my patient, I found him worse ; in fact, during one attack of the suffocative dyspnoea his attendants thought he had succumbed. Taking now the view that his symptoms were due to uræmic poisoning, he was ordered a large dose of jalap at once and a mixture containing acid tartrate of potash and spirits of nitre. After he had had three or four copious watery evacuations the urgency of the symptoms abated, and on the following day I found that he had had several short intervals of sleep, and the paroxysms of dyspnoea had not been nearly so frequent ; but, just as my colleague and I had retired to consult together, we were recalled to witness a short but severe paroxysm. The patient's face was bluish and terror-stricken, the eyes prominent, and cold perspiration was on the forehead. The heart was beating forcibly, but a little irregularly. He was gasping for breath, and yet, on auscultating the back of the chest, I could hear air freely entering ; there was no spasm of the bronchial tubes, the spasm was confined to the blood-vessels ; this state continued for a few minutes, there was expectoration of frothy mucus, and the patient was relieved. The paroxysms of dyspnoea now subsided, and on Friday, the 23rd, he seemed decidedly better, and the secretion of urine was augmented. In the evening he complained of headache and became drowsy, the next day the drowsiness increased ; he could be roused when spoken to, but fell asleep even in the act of replying, and it was evident that the same process was going on in the brain which had existed in the lungs, contraction of the minute arteries, and consequent upon it œdema of the brain. The stupor continued to increase, and he finally succumbed some thirty-six hours later.

It seems to me, then, not only that Dr. Johnson's discovery assists us in explaining the hypertrophy of the heart, but that the condition of the minute arteries, taken together with the impoverished state of the blood, help us to understand why there may be disturbance of the respirations and œdema of the lungs. The same explanation may be given of the condition of the brain in some forms of uræmic coma. Traube has ascribed this uræmic coma to œdema of the brain, with consequent cerebral anæmia. This view accords somewhat with the one I have advanced, except that the order should be inverted ; first, contraction of the minute cerebral vessels, and then consequent œdema.

A question here arises : Why should there be in some cases uræmic convulsions, and in others gradually increasing stupor without convulsions ? The hypertrophy of the heart may, I think, often account for this. If the disease have continued sufficiently long to develop the hypertrophy of the heart, I should be less apprehensive of convulsions occurring, but more afraid that either apoplexy or stupor from œdema would carry off the patient, and for this reason. A small amount of contraction of the cerebral arteries is sufficient materially to diminish the supply of blood to the brain (since the amount of fluid discharged by small tubes varies, *ceteris paribus*, according to the fourth power of their diameters), and if the heart were not hypertrophied there would be no compensating pressure to drive the blood onwards ; there would, therefore, be cerebral anæmia, but not necessarily œdema. And according to Kussmaul and Tenner, Brown-Séquard, and others, arterial contraction producing cerebral anæmia is a common cause of epileptiform convulsions. On the other hand, if there be hypertrophy of the heart *plus* impoverishment of the blood, when the arteries contract, increased pressure is exerted on the vessels, and this may be sufficient to cause the watery serum to transude through the walls of the vessels and produce œdema with its accompanying stupor.

Again, with regard to apoplexy : if, associated with Bright's disease, there be atheromatous degeneration of the arteries rendering them brittle, and increased pressure is brought to bear on the vessels from the greater propelling power of the heart and simultaneously diminished outlet, we can then easily understand rupture taking place ; and this has already been pointed out by Dr. Johnson.

But I think it is quite possible that rupture may take place without previous atheromatous degeneration of the arteries ; and in this way. From the contraction of the minute arteries, the velocity of the stream will be materially diminished (the velocity of fluid through an elastic tube varying as the square of the diameter), and

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