felt sudden pain in the head, and noise in the ears. He was subject to flushings. He was not breathless on exertion. The pulse was abrupt and varying, in the sitting posture, from 84 to 110, these rates alternating within a few seconds. The sounds of the heart were sharp, and the first faintly double. He was advised to give up tea, to take active exercise and cold showers, and to have tincture of nuxvomica and iron in a bitter-infusion.

CASE VIII.—Mrs. M. B., aged 38, a travelling *religieuse*, had been in good health till she started on a long journey a month previously, since when, having been much worried in mind and body, and frequently deprived of her regular meals, she had taken very strong tea in large quantity, and often without much solid food. Latterly she had experienced fits of dyspnoea with palpitation, accompanied with a feeling as if she were "going to die"; these fits came on in bed. She had pain in the region of the heart; pulse small and rapid (120), but regular; heart's action excited; sounds sharp and clear. She had psoriasis guttata on the front of the chest; this had been always brought out by the free use of tea. She was ordered to have an opium plaster applied to the region of the heart, to take iron and nuxvomica, and to give up the use of tea.

CASE IX.—Mr. J. O. B., aged 35, a professional man of fine physique and active habits, temperate in regard to alcoholic stimulants, but fond of strong tea, which he had often taken three times, but invariably twice, in the day, and indulged in a cigarette occasionally; had had rheumatism, and was nervous in regard to his heart. Two years previously, he experienced for the first time palpitation and want of breath on exertion. Latterly, these attacks had become more frequent and severe. He was occasionally awakened by anomalous sensations in the region of the heart, accompanied by palpitation and difficulty of breathing. On going up-hill he was similarly affected, but there was no thoracic or brachial pain. The pulse was 84, full and regular, but occasionally accelerated while he was under examination. The position of the apex-pulsation and the impulse of the heart were normal. The second sound was sharp at the right base. He was ordered to take strychnia, quinine, and colchicum; to be exceedingly moderate in the use of tea and tobacco, or to give them up entirely; to continue active exercise, and live on an elevated site (he had been advised by a medical man to avoid exertion and change his residence), and to take showers and cold sea-bathing.

CASE X.—A general medical practitioner, aged about 50, accustomed to the use of strong tea, and an inveterate smoker up to eight months previously (his ordinary allowance being an ounce and a half of strong tobacco daily), when he ceased to smoke, owing to the occurrence of variation in his pulse, with occasional intermissions, and a sensation of "tightness" and oppression in the region of the heart and left side generally, was apprehensive of heart-disease. He looked healthy; his sleep and appetite were good. There was no dyspnoea on exertion; pulse 84, regular; the sounds of the heart were sharp. He was ordered to give up tea, to take shower-baths, and, thrice daily, tincture of nuxvomica, iron, and spirit of nitrous ether. I saw this gentleman a year later, when he came to consult me in reference to his brother. He was then free from all his symptoms, as I judge from his silence in reference to them.

CASE XI.—Mr. B., a solicitor, aged 30, naturally nervous and anxious, had been accustomed to active exercise, which two years ago he gave up entirely. He had excessive professional work and insufficient sleep. He was "certain" he had heart-disease and must die. There was sharp pain in the region of the heart, and palpitation, but no dyspnoea or cough. The heart's action was "rhythmically irregular", varying in the sitting posture from 84 to 120. The pulse was of good volume. The second sound of the heart was double. I assured him he had not disease of the heart, and prescribed quinia and strychnia, bromide of potassium and morphia at night to ensure sleep, and aperients. A week later, he reported himself as "not nearly so nervous", and sleeping well. His pulse then varied from 84 to 96.

CASE XII.—Thomas C., aged 16½, had been addicted to "self-abuse" (but not for the last six weeks), and accustomed to drink strong tea. He had an attack of palpitation five weeks previously; then thought he was dying, and insisted on sending for a clergyman. He had not had rheumatism nor hæmoptysis. There was no dyspnoea. The pulse varied from 72 to 96. The apex-beat was half an inch inside the nipple-line. The impulse was strong and attended with thrill. The sounds (especially the first) were sharp. There was varicose enlargement of the left spermatic veins. He was ordered to take iron and strychnia and aperient pills, to have an opium plaster over the heart and an elastic scrotal bag; to give up tea.

CASE XIII.—J. H., aged 19, a medical student; had been a great smoker since he was sixteen years old, using half an ounce of strong tobacco daily. He had taken tea freely, and had been addicted to

masturbation. He complained of palpitation, which was especially troublesome at night. He had visible pulsation of the arteries. Pulse abrupt, 96, but variable in rate. Whilst standing and making a forced inspiration, the pulse-rate descended to 48. A thrill was felt at the apex of the heart, which pulsated in the normal position. The impulse was strong and abrupt; the sounds sharp and clear. There was a faint systolic murmur at the base, louder at the left than at the right base, and not audible in the neck. He was ordered to have iron and strychnia, and an opiate plaster to the præcordia. A month later this gentleman returned, feeling much better. His pulse was then 84 and scarcely variable. There was a venous hum in the neck. He was ordered to have dialysed iron and digitalis.

ON AORTIC REGURGITATION AND THE CORONARY CIRCULATION.*

By T. CLIFFORD ALLBUTT, M.A., M.D., F.R.S.,

Senior Physician to the General Infirmary, Leeds.

IN the second edition of his work on *The Heart and its Diseases*, Dr. Milner Fothergill takes up and extends an opinion of Dr. Balthazar Foster concerning the relations of aortic regurgitation and the coronary circulation, which opinion seems to me to be erroneous, and to be based upon a not less erroneous conception of the theory of the circulation. Dr. Fothergill's works are so attractively written and give evidence of so much enthusiastic labour, Dr. Balthazar Foster's name again is a name which carries so much lawful weight, that I feel the more bound to protest against the opinion to which I refer, lest it should gain a credence wholly disproportionate to its truth. My two friends are so rich in achievement that they can well afford either to withdraw from an untenable position, if I prove it to be untenable; or, if I fail, to forgive my indiscretion. The subject is one on which for many years I have bestowed more or less unsuccessful pains, and the opinion I shall quote is one which, rightly or wrongly, I rejected long ago. It is expressed most distinctly on page 504 of Dr. Milner Fothergill's second edition. He says, "Aortic regurgitation is, usually, swiftly fatal, from the nutrition of the heart failing early, the blood escaping past the coronary orifices instead of eddying into the sinuses of Valsalva; indeed, Balthazar Foster has pointed, with terrible distinctness, to the effect exercised prognostically, by the fact of which aortic cusp is ruptured—if the one behind which there is no coronary artery, then the prognosis as to duration of life is better than if either of the other cusps are torn down."

Now, that aortic regurgitation is "usually swiftly fatal" is a proposition so much open to qualification as to unfit it for Dr. Fothergill's dashing and effective utterances, but it is when the author rises to the heights of melodrama that I think he is least successful. What if this terrific attitude of Dr. Foster and himself be taken up on a false footing, as I scarcely doubt that it is? Let us see exactly what Dr. Foster and his disciple mean. First, that in all cases of aortic regurgitation the blood "escapes past the coronary orifices, instead of eddying into the sinuses of Valsalva"; secondly, that this ineffective passage and its consequences are diminished if one of the cusps subtending a coronary artery persist. I think this is precisely what is meant; and it follows of necessity that these authors believe that in the normal state the coronary arteries are filled by a sort of squirting action which is incidental to the systemic circulation. Surely, to begin at the beginning, this latter proposition is based upon views of the circulation which all physicists have given up, or would give up, if they applied their present knowledge to the problem of the circulation. Nay, the opening paragraphs of Dr. Fothergill's own book led one to expect a larger view of the matter than this. I extract the following sentences, "Such a power of adaptation (to the needs of the organism) is secured by means of the beautiful nerve-adjustments through which and by which the regular rhythmic action of the heart is maintained"; again, "In order to maintain the balance of the circulation, and a steady equilibrium betwixt the bulk of blood in the arteries and in the veins, constant modifications in the heart are necessitated, including variations in the size of the ventricular chambers; and these are rendered possible by the exquisite nerve-balances amidst which the heart swings". Quite true; in the general allusive sense in which such introductory words are to be taken, nothing could be better. Then how comes Dr. Fothergill to fall from this real point of view to the clumsy notion, that in aortic regurgitation a prominent cusp acts as a shovel would act if held in a running stream? Surely the whole circulation since the time of Harvey has been regarded more and more as a "closed cell", and surely then the pressure upon

* Read in the Section of Medicine at the Annual Meeting of the British Medical Association in Cork, August 1879.

given square areas of surface above and below the semilunar valves is equal, whether those valves be present and efficient, or be absent. Granting this, how can Drs. Föster and Fothergill refine upon supposed sets given to the current this way, and that as they would reason of water in a closed culvert or in a rain water pipe? Nor again, can I admit the right of the author to find in diminished tension any such explanation as he assumes. Tension does not depend upon the force of the heart-beat, but upon those nervous adaptations to which Dr. Fothergill so eloquently appeals. Moreover, were it not so, he will scarcely deny that tension is equal at any and all points say of the inner surface of the ventricle and of the aorta. This being so, how can the blood thrust itself or be thrust harder into one coronary artery than into another? The most elementary knowledge of hydrodynamics would guard us from so crude a conception as this. I do not hesitate to advance that all elaborate prognoses based upon this conception are worse than wasted.

Once more, does Dr. Fothergill really believe that hypertrophy of a part bears an exclusive relation to the quantity of blood submitted to it? If blood be denied to the part, no doubt it must starve, but it would be too hardy an assertion to say that in aortic regurgitation the blood is shut off from access to the coronary arteries. Surely they have still the same supply that is given to any other artery in the body, and the cardiac muscle can grow still in obedience to the general laws which govern hypertrophy. We know that fatty degeneration of the heart may be detected during life, and the heart muscle nevertheless recover perfectly, if the coronary arteries be normal. Excessive arterial supply, on the other hand, cannot alone give rise to hypertrophy; so far as we know it is the *vis à fronte* not the *vis à tergo* which is herein concerned. We must look, therefore, to some causes of systolic failure in aortic regurgitation other than differential pressures at the mouths of the coronary arteries. The question is of such direct practical importance that for many years I have sought to solve it. Dr. Fothergill will, perhaps, remember that when he lived in Leeds, I showed him notes, preparations, and specimens illustrative of my views on causation of aortic arteritis and valvulitis by bodily effort acting either suddenly or very gradually. In these I frequently found the mouths of the coronary arteries lessened, and sometimes found them occluded by "atheroma".

It was thus possible in many cases to trace fatty change in the muscular tissue supplied by one coronary artery to atheromatous or embolic blocking and so to arrest of blood-supply. To prove that this cause is the only cause of asystole in aortic regurgitation would be difficult, and probably it is not the only cause: that it is a common cause of it I feel pretty confident. The compensation itself is but imperfect at best, and carries in itself no assurance of permanency. However, the purpose of my present criticisms is rather destructive than constructive. After I had committed these criticisms to paper, I consulted Dr. Stone, in order to secure myself against error. Dr. Stone wrote to me as follows on October 2nd, 1879. "Dear Dr. Allbutt,—My views would be entirely in agreement with yours. Indeed, it is quite unmechanical to suppose that the coronary arteries are filled by a 'squirting' action. Neither do I believe that hypertrophy originates in arterial action, though fatty degeneration may depend on coronary starvation. The regulating power must originate in the beautiful balance of motor and inhibitory centres which the arteries obey. Dr. Fothergill is really going back to the obsolete humorism. Dr. Ottomar Rosenbach's experiments, moreover, show conclusively that tension and propulsion, in the closed cell of the circulation, are perfectly separable factors, and that the pressure is maintained unaffected, even when the aortic or mitral valves are artificially broken down. This I worked out in my third Croonian lecture, which I am now preparing for publication."

AN INQUIRY INTO CERTAIN POINTS CONNECTED WITH ALBUMINURIA.

By ROBERT SAUNDY, M.D. Edin.,

Member of the Royal College of Physicians; Assistant-Physician to the General Hospital, Birmingham.

RECENT observations* have established the frequency with which albumen appears in the urine apart from structural disease of the kidneys or any marked deterioration in the general health. It has been long known†

that albumen is present in many grave diseases without structural renal disease or the other symptoms of Bright's disease accompanying it.

In an article published last year, on Albuminuria (*Birmingham Medical Review*, July 1879), I reviewed all these facts, and, having defined albuminuria as "the presence in the urine of a body coagulated by heat or precipitated by neutralisation"; I showed that there were only two albuminous substances which are likely to be met with giving these reactions—viz., 1. Serum-albumen, and 2. Paraglobulin. I am not aware that any researches have been undertaken to show whether paraglobulin ever occurs by itself in the urine; but Edlefsen, J. C. Lehmann, and Senator found it constantly present, together with serum-albumen, in the urine of Bright's disease. The first part of my inquiry was therefore directed to settle this point.

Chemists tell us that paraglobulin is wholly precipitated from its sodium-chloride solutions by saturation with the latter salt or by carbonic acid. On my applying for information on this point to Professor Crum-Brown of Edinburgh, he wrote to Professor Hoppe-Seyler, who referred me to a paper by Hammarsten (Pflüger's *Archiv*, Band xviii, s. 38), in which magnesium-sulphate is recommended; and this method Professor Hoppe-Seyler informed Professor Crum-Brown he had found trustworthy in his own investigations on milk. In the following experiments, all these methods were used, but in many they were combined.

The method of proceeding was to test an urine for albumen by boiling and adding acetic acid, then taking a fresh portion to precipitate it, filter, and test the filtrate as before; the two test-tubes were then compared. The cases were taken as they appeared in the out-patient room, without any kind of selection, and present most of the conditions under which albuminuria occurs apart from acute disease. In all, forty-two examinations were made; two of these being of the same person in one instance only. They comprised cases which may be thus classified: Chronic Bright's disease, 5; albuminuria of adolescents, 16; epilepsy, 7; morbus cordis, 3; amenorrhœa, 3; phthisis, 3; bronchitis, 3; gastric catarrh, 1; scabies, 1. The albuminous cloud diminished after precipitation as above in a large number of these cases, and entirely disappeared in two. One of these was a case of phthisis; the other presented symptoms of dyspepsia, and has been included among the cases of albuminuria of adolescents. On subsequently examining the urine of the same case, the albuminous cloud disappeared partially only. Hence the existence of paraglobulinuria as an independent condition is certainly rare, and cannot be held to explain the frequency with which the reaction of albumen is given by the urines of adolescents and others supposed to be suffering from merely functional derangements of the renal organs.

The next point alluded to in my article already quoted is the doctrine of "food-albuminuria", originated in modern times by Parkes (*Medical Times and Gazette*, 1852, p. 357, and 1854, p. 395). He found that the albumen was increased after food in two cases of chronic Bright's disease, although it was diminished after food in a case of heart-disease. He inclined to the view that the increase after food was due to the passage of imperfectly digested albumen, analogous to that which occurs when albumen is injected into the veins, or to the albumen undergoing some modification in the digestive process, such as its conversion into an acid albuminate, by which its diffusibility would be increased.

Parkes's facts do not seem to warrant this conclusion; for, while the albumen was increased after food in the two cases of Bright's disease, it was diminished under similar circumstances in the case of heart-disease, so that it is necessary to postulate a peculiar inability to digest albumen in the former cases which did not exist in the latter. On the other hand, if we regard albuminuria as a mechanical process, resulting from increase of the lateral pressure within the vessels to such a degree as to permit transudation, we can easily understand why, in the cases of chronic Bright's disease, the stimulus to the circulation increased the albuminuria in already inflamed organs, while it diminished it in the case of heart-disease, where the transudation was due to passive engorgement of the renal capillaries and veins. But Parkes's views have been adopted by many writers. Dr. Pavy (Gulstonian Lectures, *Lancet*, 1863, p. 573) supports Parkes by giving a table of the amounts of albumen excreted before and after breakfast in six experiments, all showing a marked increase. Dr. Pavy further adopted Parkes's view that this might be due to the increased diffusibility of the urine, and supported it by showing that the albumen in some urines is highly diffusible, but did not attempt to prove that the albumen passed after food was more diffusible than that passed before food, or to indicate to what circumstances this increased diffusibility might be due.

Most physiologists deny that unchanged albumen is absorbed into the blood, and the only piece of direct evidence I know to the contrary is the statement of Brücke, that he has found coagulable albumen in the

* Dr. Moxon, *Guy's Hospital Reports*, third series, vol. xliii.; Dr. Dukes, *BRITISH MEDICAL JOURNAL*, November 30th, 1878; Dr. Morley Rooke, *ibid.*, October 10th, 1878; Dr. Lauder Brunton, *The Practitioner*, June 1877; Professor Leube, *Virchow's Archiv*, Band lxxii, Heft 2; Dr. Saundby, *Birmingham Medical Review*, October 1879; Dr. Mahomed, *Guy's Hospital Reports*, vol. xxiv.; Dr. Geo. Johnson, *BRITISH MEDICAL JOURNAL*, December 13th, 1879.

† Dr. Parkes, *On the Composition of the Urine*, page 186; Dr. W. Roberts, *On Urinary and Renal Diseases*, p. 173; Dr. Quain, *The Lancet*, January 18th, 1879; Dr. Paul Fürbringer, *Virchow's Archiv*, Band lxxi, Heft 3.