A Clinical Lecture ON THREE CASES OF ARTERIAL DISEASE.*

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

REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF CAMBRIDGE.

GENTLEMEN,—The young woman whose case will occupy the first part of our lecture this morning has been repeatedly a patient in the wards of this hospital, and also, I think, more than once in St. Thomas's Hospital, under the care of Dr. Hawkins. I believe I may venture to say that in the interpretation of this case I have the advantage of Dr. Hawkins's agreement. X. Y., now aged about 28, came first into the Addenbrooke's Hospital four or five years ago, and the account of her illness which she then gave has been repeatedly verified. Before her admission she had been in service as housemaid in one of the houses of a large public school. On a certain morning she helped to lift a bedstead. She is, as you see, an ill-developed girl of no great physical strength, and on making this unusual effort she felt a sudden pain or sense of distress in the chest under the sternum. She was carried to bed faint and ill, and from that time until recently she has been unfit not only for her proper duties but for any kind of bodily exertion whatever; the slightest effort being followed by a renewal of the substernal pain and of the distress. Up to the date of this accident, except for some anaemia, her health had been fairly good. She was admitted to this hospital soon after the accident, and on the first occasion she remained with us for many weeks; afterwards, as her distressing symptoms continued for a much longer period of time, she returned to our care more than once, and, as I have said, was meanwhile received once or twice in the wards of St. Thomas's Hospital for the same malady. Thence it was that Dr. Hawkins, being much interested in the case, was so good as to write to me about her. At a later date she came once more into this hospital, and finally, as her health became gradually re-established, she attended here, regularly or occasionally, as an out-patient. She is now restored to something like health—that is to say, she has no complaint to make to you; and she has been kind enough to attend here to-day only for the purpose of our clinical demonstration.

To return to her symptoms on admission: she was then suffering from paroxysms of intense pain having its origin, as I have said, under the upper and mid sternum—a pain attended with a peculiar crushing agony, as if the breast were in a vice, and with a sense of impending death, due, I believe, to a powerful afferent impression upon the cardiac centre. The pain radiated from its substernal origin down the left arm to the elbow, or, when more severe, to the ulnar side of the left hand. When severest it would extend in like manner to the right arm. During the first stage of her illness—a stage which lasted for many weeks, or I think I may even say for some months—these paroxysms recurred almost continually, constituting what some of you may have heard me call in similar cases the *status anginosus*. Very slight bodily movements, or a little mental excitement, sufficed to arouse these attacks of pain; and it was only by means of specific remedies—by the nitrites especially—that her sufferings could be mitigated.

As this incessant distress told upon her powers of endurance, the difficulties of the case were increased more and more; her self-control was weakened, the pain of the attacks, or even the mere dread of them, threw her into states of anxiety and apprehension which brought her into a condition of fretfulness so persistent that it was difficult at times to say how much of her minor distresses were due to the primary causes, and how much to the unbalancing of her mind and nervous system. But of the interpretation of the main features of her malady—namely, that she

was suffering from angina pectoris in its most typical form and extreme degree—there was no doubt whatever.

On physical examination, the only sign which I need now recall for you was a loud systolic murmur at the base of the heart, carried into the arteries of the neck. There has been no regurgitant murmur at any time. With the heart, beyond some functional disorder which under ordinary circumstances would have been of little moment, no definite fault could be found during this first period of her illness. The accessible arteries of the body seemed to the touch to be free from disease, and the arterial blood pressure was not increased.

From the time of her first admission to the present date the story may be briefly summed up as one of a very slow recovery, the attacks of pain becoming less frequent and less severe. For many months,* she now tells us, she has been entirely free from anginal attacks, and her health is fairly satisfactory. I may add that, as the specific pains subsided, her mind and her powers of self-control became re-established; and, as you see, she is now natural and self-possessed in bearing.

But to-day physical examination of the heart will tell us another and somewhat different story. When you proceed, as you shall now do, to examine the heart, you will find the left ventricle considerably hypertrophied; the systolic murmur still persists at the base of the heart, and I will add that it is louder and more prolonged than before. Moreover, on laying the hand upon the upper part of the sternum you will find a strong vibratory thrill with the cardiac systole, which thrill, I think, was scarcely perceptible in the first few weeks of the illness, but has since become more and more evident. There are no signs of aneurysm. The case is, then, one of aortic stenosis, with compensatory hypertrophy of the left ventricle. Apparently the compensation is sufficient, so that, for the present at any rate, the patient is free from discomfort; and, although avoiding much physical stress, she is occupied in the quieter duties of life. There is now no reason to fear that her life will be cut short prematurely.

In its main lines the interpretation of this case is not difficult. The strong bodily effort of lifting the bedstead, perhaps by causing a sudden rise of arterial pressure in a young woman of weak frame, and perhaps originally with some aortic hypoplasia, seems to have done some injury in the aortic area. As neither then nor since has any murmur of aortic regurgitation been heard, it would scarcely seem that this injury fell directly upon the aortic valve; it would seem rather to have injured the aorta near its origin, and there to have set up inflammatory changes which extended to the valve and orifice, so that, in the course of time, a stenosis of the orifice was established. With the healing of the lesion the pain, at first so agonizing, gradually abated and disappeared. On the precise form of the lesion it is not easy to give an opinion; I must content myself with reminding you that not infrequently in *post-mortem* examinations rents or splits of the inner coat of the aorta have been discovered; it may be that in this instance some such rent took place just above the aortic orifice and valve, which were gradually invaded by the consequent inflammatory process. Some lesion at any rate at the origin of the aorta made it abnormally sensitive, so that normal blood pressures produced a characteristic pain which in the majority of such cases requires some excess of arterial pressure to call it forth. I do not draw any parallel between this case and cases of aortic aneurysm, because in aneurysm there are nearly always morbid conditions precedent—such as syphilis—which in this case, to the best of our knowledge, were absent. Nor can I tell you why an aneurysm did not form in this case. Although the parts, so far as we can tell, are now thoroughly healed, and the pains have all ceased, yet of course the heart is permanently, if not as yet very gravely, damaged.

The diagnosis, then, is acute angina pectoris in its most typical form.† You may wonder how it happened that a

* Now perhaps a year or two (December, 1905).

* These three cases were brought forward on three several occasions, but for brevity's sake the three lectures are condensed into one. My thanks are, now and always, due to my colleagues, Dr. Bradbury and Dr. Humphry, for the generous way in which they allow me to select cases for my lectures from their wards.

† It seems necessary to remind some readers that by a "disease" we mean a group of symptoms which recurs with approximate constancy, and to which therefore we find it convenient to attach a label or name; moreover that, as similar events must result from similar causes, no approximately constant group of symptoms can result from different causes. Angina pectoris is a remarkably constant group of symptoms, and accordingly its cause is so far constant—namely, a morbid state of the first portion of aorta under a blood pressure absolutely or relatively excessive. "Tobacco angina" and "pseudo-

patient suffering from this disease in so intense and prolonged a form survived, and has even made, so far as the angina pectoris is concerned, a complete recovery. The reason is this, that throughout the illness the cardiac muscle was healthy. The complete and satisfactory compensation which has taken place is a sufficient evidence of normal cardiac conditions. Intense peripheral pain, such, for example, as the passing of a gall stone in a person otherwise healthy, or, experimentally, such a shock in an animal as irritation of the central end of a cut sciatic nerve, or even a sudden slap on the epigastrium, may, it is true, by reflex inhibition arrest even a healthy heart, but of course still more readily a heart which is something less than healthy; a heart, let us suppose, which, by disease of the coronary arteries, is deteriorated in muscular value. But fortunately in man arrest in death of a sound heart by peripheral pain is a very unusual event. In two cases at least I have seen it, one of them being a healthy young matron who died suddenly during a violent attack of gall stone; after death the stone was found in the duct, and all other organs were normal. The heart of the patient before us, then, being a sound one, weathered the storms. When the heart is healthy angina pectoris may not be, and usually is not, a fatal disease. One of the worst acute cases I have seen, a case as typical and as severe as of this young woman, though not so prolonged, was in a Selwyn undergraduate under the care of Mr. Arthur Cooke for acute rheumatism with aortic valvulitis and aortitis, due, as we surmised, to basic pericarditis. So far as the angina pectoris was concerned, he likewise recovered completely. Such also was the happy issue of a case in a middle-aged man of very severe recurrent angina pectoris, due probably to influenzal aortitis, whom I saw with Dr. Humphry. I have collected indeed a large number of cases in my own sphere of experience in which angina pectoris, even continuous and severe, ended in complete recovery; and this, moreover, in not a few elderly persons; though of course as life goes on the chances are that by disease of its vessels the heart becomes less and less able to withstand powerful inhibitory influences.

Now by cases such as these we learn that angina pectoris is not a disease of the heart; it is a disease of the first part of the aorta: it is the mortality of it which depends upon cardiac disease, and this is a contingent feature. I will take the opportunity of reinforcing the truth that the pain of angina pectoris is not a pain of the cardiac area, as in the discussion of cases—and, indeed, in the description of textbooks—is so often alleged, for such is the effect of a prepossession in loading our mental dice. On the other hand the not uncommon pain of heart disease, and especially of hearts under dilating stresses, is in the cardiac area; but this is not an intense and paroxysmal pain: it is a weary and more or less continuous aching. The pain of angina pectoris, which ranges from a sense of tightness and oppression ("stenocardia") scarcely amounting to pain, but imperiously arresting the patient in his walk, up to the violent degrees of it in chronic cases, and in acute cases such as the patient before you, is always seated, not in the cardiac area, but in the area of the upper sternum, and is, in its extremest degrees, as if the sternum were pressed inwards by an iron bar. Again, I must impress upon you that dyspnoea is no feature of angina pectoris; on the contrary, in the attack, respiration is arrested by reflex inhibition. A few weeks ago a gentleman, aged 73, was sent to me by his medical attendant with the diagnosis of angina pectoris. The story was that, being so far as he knew in health, on ascending an incline he was suddenly pulled up, not with substernal pain, but with dyspnoea and a not intolerable epicardial uneasiness. The physical signs were those of an inconstant mitral regurgitation and of dilatation of the left ventricle. We were able to assure him therefore, to his consolation, that the attack was not one of angina pectoris—a diagnosis which is now verified by the results of treatment with digitalis and exercises.

angina" are not angina pectoris in the clinical sense; as toothache is not, nor colic. In my Lane Lectures of 1896 I described a somewhat definite group of symptoms which seemed to me then to be an aberrant form of angina pectoris. But the pain is across the epigastrium; and although it is attended with apprehension, and is of ill omen, it is not extreme in degree, it does not radiate, the patient can cry out, there is some dyspnoea, and it is not dispelled by nitrites. I now believe these attacks to be primarily cardiac.

There has been no recurrence of the "attack," only the usual variations of a failing heart.

The second case to which I will draw your attention is that of an agricultural labourer, 55 years of age, who is sent up to us, before any complete examination has been made of him, simply with the record of "mitral regurgitation." We will proceed to examine him. For a weather-beaten man he looks somewhat anaemic, his lips are pale and bluish. On glancing almost instinctively at his ears, we are surprised to find them not dusky in colour but pallid. Again, on looking at the neck, where we expected to find some distension of the veins, we see no such distension; the veins of the neck are not remarkable. We observe, however, another feature of his neck which is of great importance for the diagnosis, namely, that the sterno-mastoid muscles on both sides are very tense and hypertrophied. Evidently he has been suffering from a chronic difficulty of breathing, and for a considerable time. On examining his chest we find that, during quiescence, thoracic breathing is scarcely perceptible, while abdominal breathing is exaggerated. On telling him to take a deep breath the thorax is rather lifted than expanded. The form of the chest is not much altered. On listening to his heart there is, it is true, a murmur of mitral regurgitation; but there is no oedema of the feet, no enlargement of the liver nor tenderness on palpation under the ribs, nor other signs of rise of pressure on the venous side. Instead, then, of the dusky ears, swollen and pulsating jugular veins, puffy ankles and tumid liver of mitral insufficiency we find a condition of arterial anaemia and an emphysema which is rather of the atrophic than of the voluminous form. That some mitral regurgitation exists is indubitable, but probably slight in degree—the mere loudness of a murmur is no guide to the degree of regurgitation—and not sufficient to bring about any evident clinical symptoms.

Now let us go a little farther. On examining his brachial and radial arteries and the dorsalis pedis, we find the arterial pressure low and the arteries not very well filled; but the vessels themselves to the touch are thick, tortuous, and uneven, as though the walls were the seat of some irregular deposits. This man presents a good example of what I have distinguished as the involutionary form of "arterio-sclerosis." With it emphysema is commonly associated, and, as I have often said to you, this association is something more intimate than a mere coincidence of two kinds of senile change. Now, if this patient had been submitted to a candidate for examination he might have given us a diagnosis of mitral regurgitation only; and, in answer to our questions on treatment, have proposed to us the administration of digitalis. I think you will have inferred, from the manner in which this case has been brought before you, that, although this patient has mitral regurgitation, he is not suffering from mitral regurgitation; indeed, no specific cardiac treatment is required. Clinically, his disease is not mitral regurgitation but primary senile arterio-sclerosis and emphysema.

This involutionary form of arterio-sclerosis I find—and I am reinforced in this opinion since I came to live in this rural district—to be very prevalent among the agricultural labourers; it seems in them to come on earlier than in the middle and upper classes of life, and, speaking generally, to advance more frequently to its extremest degrees. Why this should be so I cannot offer you even a surmise; it may be due to hard conditions of life, but the agricultural labourer is better fed than he was a generation ago. It may be due to some accumulation of hereditary tendencies by intermarriage, or to continuous exposure to cold without a sufficiently generous diet. Syphilis is not very prevalent among the agricultural people; and alcohol, even if, taken alone, it be one of the causes of arterio-sclerosis—which I regard as a very doubtful proposition—probably plays a much smaller part in agricultural than in urban populations. It is when other and more specific causes of arterio-sclerosis are at work that alcohol becomes their formidable ally. This, the primary, form of arterial degeneration is, as I have often told you, compatible with long, even with very long, if very unavailing, senescence. During this period the arteries may become almost grotesquely tortuous and deformed, the organs of the body undergoing a coincident atrophy; but, as the blood pressure is continuously moderate, the heart does not

dilate, and the blood vessels of the brain are liable rather to obliteration than to rupture.

When we turn to this third patient, however, we find both the present conditions and the story of his past to be essentially very different from those of the second. This man's age is 52, he is a fat, high-coloured man, and herein presents a strong contrast to the previous patient. He does not work on the land, but in some commercial occupation. He has lived well and taken alcohol freely. During the last few months he has complained of shortness of breath, and you will observe that he begins to pant on the comparatively slight effort of taking off his coat. You will note this in cases of chest disease, as you may note manual disabilities in diseases of the nervous system. His ears, you see, are dusky; the veins of his neck are rather large and oscillating, his liver is tumid, and you will perceive on palpation a slight oedema about the shins and ankles. The heart is very large and labouring, and you may presently hear an inconstant murmur of mitral regurgitation. Over the bases of his lungs behind the resonance to percussion is imperfect, and the respiratory murmur, because of intercellular oedema, is deficient; some little intracellular oedema gives rise also to a few crepitations there, on deep inspiration. The urine is scanty, of high specific gravity and lateritious; it contains a "trace" of albumen.

On turning our attention to the upper sternum, we find the arch of the aorta too perceptible above it, and on percussion the area of it is extended 3 or 4 cm. to the right in the first space. The subclavian arteries beat heavily and are somewhat displaced, and in this area there is a persistent systolic murmur. That the thoracic aorta is lengthened we infer from the arc of the cardiac swing, which is almost doubled in extent. On following the arteries into the arms, we find these vessels much thickened throughout the whole of their course, and the radial arteries also are leathery and tortuous. The pulse is large, and although you might hesitate to estimate even roughly the degree of blood pressure—because the larger an artery the more is the sense of increased pressure, whether increased or not—yet you will not fail to observe that the wave does not normally subside between the beats, but gives to your finger the impression which is known as that of the "sustained pulse." I inform you, however, that by means of the sphygmometer we have learnt that, in spite of the yielding heart, his systolic arterial pressure is still fully 200 mm. It is remarkable in cases of this kind that, even in the latter stages of the malady, the pressure, if lower than in the earlier stages, usually maintains a high figure.

In this case we may safely assume that the arterial pressure has been considerably higher than its present record—probably 230 to 250 mm. Here, then, is another case of arterio-sclerosis indeed, but a case of a disease very different from that of the previous patient. In this patient we have to deal with a *secondary arterio-sclerosis*, which I have named that of hyperpiesis; with an injury done to the vessels by the strain of an excessive arterial blood pressure acting upon these blood vessels for a number of years—five or six at least, probably more. The blood-pressure is now beginning to fall, but with no advantage to the patient; on the contrary, this fall is ominous of dissolution, for it depends not upon any diminution of the causes in which this high pressure originally took its origin—causes, namely, of peripheral resistance—but upon the failure of the heart which, after years of excessive effort, finds itself at last unable to continue an unequal strife. When dyspnoea appears, such a patient has entered the last stage of his pilgrimage.

Now in both these forms, in the arterio-sclerosis of involuntary degeneration, as in that of hyperpiesis, the aorta is practically always the first seat of atheromatous change; and, speaking generally, the arteries of the mesenteric area come next to the aorta in liability to this alteration. But, although the subjects of involuntary arterio-sclerosis, like other people, are not immune from periods of hyperpiesis, the arterio-sclerosis itself is not the cause of the elevation. In patients of the involuntary class the arterial pressure, as I have demonstrated to you again and again, and as you may see for yourselves abundantly in any workhouse, is moderate, or indeed even low—absolutely so, or low for the time of life in the

particular case. You are aware that in the advancing years of normal persons the arterial pressures range rather higher than in the third and fourth decades.

In normal elderly persons, however, and in elderly persons the subjects of arterio-sclerosis without initial hyperpiesis, the arterial pressure may rise occasionally, on the intervention of those obscure causes which produce it at times in any, perhaps in all, of us. Many and many are the cases in old folks, whether with thick or normal arteries, in whom, when certain discomforts, such as confusion or aching of head, sluggishness, depression of spirits especially of a morning, "biliousness," sleeplessness, call attention to the circulation, hyperpiesis is discovered. As yet the condition is so little realized and anticipated, that it is overlooked again and again; I had almost said more often than not. When detected, a course of calomel and other appropriate remedies dispels a temporary resistance of some kind, the arterial pressure falls to the normal, the fear of an apoplexy is averted, and the patient is set on the way again towards the fulfilment of his days.* But with those elders who suffer from hyperpiesis, who have so suffered persistently for a long time, the tale is a different one. During the first years of its establishment it is not inconsistent with apparently good health. When detected, by vigilant treatment the pressure may be reduced again and again, but not in all cases; in not a few of them, when of no long standing, it may be got rid of completely, more especially in persons not beyond middle life; but if of some standing it recurs again and again, and the patient dies, more or less prematurely, of apoplexy, of cardiac failure, or, as it sometimes happens, of an intercurrent pneumonia. These patients do not, as do the subjects of primary or involutionary arterio-sclerosis with moderate blood pressures, survive uncrippled into the seventh decade—unless, I repeat, the malady has been detected and combated in good time.

In children periods of hyperpiesis, with headache, coated tongue, foul breath, dyspepsia, loss of appetite, rings under the eyes, languor, low spirits, are by no means uncommon, and are curable by gentle mercurials and the like. In some of these children the stomach is large and squelchy. Whether such children are those individuals who become the subjects of hyperpiesis in later life is more than I can tell you.

In conclusion, I may remind you of the cases of my third, or extrinsically toxic, class of arterio-sclerosis. These I show you from time to time in the wards or in this lecture room, cases of arterio-sclerosis consequent upon such infections as diphtheria, syphilis or typhoid fever, or, again, in such chronic diseases as diabetes and plumbism. Recently I showed you such a case of general arterio-sclerosis in a diabetic girl, aged 14. In her, as in the majority of such cases, excepting the plumbic, the pressure was normal, or at any rate not elevated; strictly speaking, it was rather subnormal for her age. Thus to you, as to my classes for many years in Leeds and in Cambridge, I have been demonstrating almost daily the error of the doctrine that rise of arterial pressure is the consequence of arterio-sclerosis; how far it may co-operate in this direction is another question, but as it is one of no great importance I regret the less the postponement of it to another day.

*For some brief notes as to treatment I may refer, among other of my papers on this subject, to the *Trans. Hunt. Soc.*, 1885, and to the *Trans. Med. Chir. Soc.*, vol. 86, 1903.

CONSOLIDATION OF NEW YORK MEDICAL SOCIETIES.—At a special meeting of the Supreme Court, held at Rochester on December 9th, Judge Davy signed an order consolidating the Medical Society of the State of New York and the New York State Medical Association. By this act the medical profession of New York is united in one body. The members of the county societies become members of the district branches and of the State society; thus the new body has a membership of over 6,000. Negotiations towards the accomplishment of this end have been in progress for the last four years.

THE Governors of the Bolingbroke Hospital are making an active endeavour to obtain the £30,000 which are required to convert the institution into a general hospital. Towards this amount nearly £5,000 has been collected, of which £2,000 has been contributed by King Edward's Hospital Fund for London.