which the heart obtained. But, on account of the greater length of this big contraction, it will be followed by a somewhat shorter pause, even if the heart returns again to its normal rhythm at once. The next systole will therefore be smaller; but as it lasts a shorter time, it is followed again by a longer pause, and the succeeding systole will again be larger."

Although the pulsus alternans has been demonstrated to be a symptom of impaired contractility, it does not follow that this function is intact in the absence of the pulsus alternans. Patients may die of depression of contractility, and this alternating action of the heart never appear. Its appearance may be very transient, due to some temporary cause. Fig. 1 was obtained from a patient immediately after exertion, and with the symptoms present of angina pectoris. As soon as I had taken the tracing the pulse became perfectly regular, though the other symptoms of cardiac exhaustion persisted. In the quotation from Wenckebach's book just given, it is stated that in experiments by changing once the frequency of the heart's action the alternating rhythm may be started. This is well seen in Figs. 4 and 5, where, after a long pause—due probably to a ventricular extra-systole—the alternating rhythm is started and continued for a brief period.

Fig. 4.—Tracing of radial pulse showing the appearance of the pulsus alternans after a long pause. From a patient suffering from angina pectoris.

Fig. 5.—Showing the same thing as Fig. 4. From a patient aged 72 suffering from great exhaustion after an attack of bronchitis.

It may be asserted that while the absence of the pulsus alternans must not be taken as evidence of the integrity of the function of contractility, its presence is, on the other hand, a distinct sign of the impairment of this function.

Symptoms Associated with the Pulsus Alternans.

All patients who exhibit this form of heart action invariably show a great limitation of the field of cardiac response; that is to say, while at rest the heart may seem to be doing its work efficiently, very slight exertion will at once produce symptoms of cardiac distress.

Conditions giving rise to the Pulsus Alternans.

When we inquire into the conditions in which this symptom appears, we find a striking resemblance to the conditions that provoke an attack of angina pectoris. Thus, I have found it appear for a short time in a patient with an enfeebled heart after walking hurriedly (Fig. 1). I have found it in several cases when the heart was exhausted after struggling against a very high arterial pressure (Figs. 2 and 4). I have found it in a patient after a long attack of paroxysmal tachycardia associated with profound exhaustion; and Dr. John Hay has shown me tracings of the pulsus alternans from a similar case. Wenckebach mentions that Huber had also found the pulsus alternans in the course of an attack of paroxysmal tachycardia. I have also found it in a youth with acute dilatation of the heart a few days before death, and in a female aged 72 after an attack of bronchitis (Fig. 5). In the record of two cases by Wenckebach and of a case by Volhardt the pulsus alternans was associated with other evidences of great exhaustion of the heart muscle.

Association of Angina Pectoris with the Pulsus Alternans.

I am confident that if tracings were systematically taken of the radial pulse in cases showing the symptoms of angina pectoris the pulsus alternans would be found fairly frequently.

It is not easy to detect it with the finger or by the heart sounds, though when the attention is specially directed to the matter it may be recognized. In this way further evidence of the association of angina pectoris with exhausted contractility will be found. In five of my cases of pulsus alternans the symptoms mentioned as characteristic of angina pectoris were present. In six other cases the pain was absent, but the symptoms of exhaustion, shortness of breath on exertion, and general distress, showed that practically the symptoms were identical with those of angina pectoris, minus the pain.

The Arterial Pressure is not a Guide to the Condition of the Contractility.

It might seem at first sight that the condition of the arterial pressure would be a guide to the state of the function of contractility. As a matter of fact it does not serve as a guide, as the pulsus alternans may be present in cases with an abnormally high pressure, as well as with a very low pressure. In the case of a man whose heart had been exhausted by lifting very heavy weights, and who suffered from attacks of angina pectoris with the alternating rhythm, the blood pressure was 120 mm. Hg, as measured by Martin's modification of the Riva-Rocci instrument. The pulse of the patient when Fig. 1 was taken was extremely soft and compressible. The pulse in the patient from whom Fig. 2 was taken was very hard and resistant, and the arterial pressure equalled 210 mm. Hg. When the pressure fell to 170 mm., the pulsus alternans and all anginal symptoms disappeared. In this case (and in that from whom Fig. 4 was taken) the association of the pulsus alternans and angina pectoris with the high arterial pressure, and the disappearance of both phenomena when the pressure was lowered, demonstrated the intimate connexion between the exhausted function of contractility and the symptom of angina pectoris.

Conclusion.

From the consideration of the fact that angina pectoris occurs in lesions of great diversity, some condition common to all must be the cause of the symptoms. The fact that angina pectoris appears only after the heart muscle has been long exposed to excessive strain points to the cause being situated in the muscle. All the functions of the muscle fibres save that of contractility can be shown to be intact in many cases that suffer from angina pectoris. The alternating action of the heart is a demonstrable sign of exhausted contractility, and its presence is always associated with symptoms that are included in the symptom complex of angina pectoris. The same exciting cause—extra strain on the heart—may provoke both the angina pectoris and the alternating action, and both may disappear with removal of the cause. The inference to be drawn from the consideration of these facts is that the symptoms that are included in the term "angina pectoris" are so closely associated with an impairment of the function of contractility of the muscle fibres of the heart that in all probability angina pectoris will be found to be an evidence of the impairment of the function of contractility.

A CASE OF SYNCOPAL BRADYCARDIA SHOWING THE INDEPENDENT ACTION OF THE TWO SIDES OF THE HEART.

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Cases of persistent bradycardia are still sufficiently rare to justify their being reported when an opportunity has occurred for their more or less continuous observation; in the following case, moreover, there were certain clinical signs which appear to invest it with additional interest:

History.

On November 24th, 1904, I was consulted by Mr. P., aged 57, whose occupation involved a good deal of standing, but without any violent exertion. He stated that two years before he had felt giddy, and fainted for a few seconds; he was seen by a medical man, who remarked on the slowness of his pulse, but said his heart was otherwise normal for his age; from that date till six weeks before he came to me he had no return of the symptoms, and there is no evidence as to the state of his pulse in the interval. For a month or six weeks he had been suffering from palpitation, shortness of breath and giddiness, and on the morning of

November 24th he had actually lost consciousness for a few seconds.

The pulse-rate was 34 a minute, increasing on exertion to 37; there was a systolic bruit, heard best in the aortic area. Three days later he consulted me again, saying he had had repeated attacks of giddiness. I found that his heart intermitted at intervals, had a rate of 32 per minute, and that during the periods of intermission he manifested symptoms of syncopal giddiness.

On November 30th I saw him in consultation with Dr. Alexander Morison. We found the pulse-rate had dropped

Fig. 1.—November 30th, 1903. Left radial recumbent; pulse 24; left ventricular systole, followed by two weak systolic sounds over right heart, the latter, of course, not registered by sphygmograph.

to 24 there was a well-marked systolic bruit in the aortic area and a full heaving systolic impulse, followed immediately by two impulses perceptible to the hand over the position of the right ventricle, and associated with two subsidiary cardiac sounds previous to the recurrence of the succeeding left ventricular systole; and, moreover, that these succeeding sounds were not registered by the sphygmograph, as the appended illustrations will show.

The patient, observed for some days later, evinced practically the same physical signs, although on the whole he felt more comfortable.

On December 6th Dr. Morison again saw the patient with me, and we found that the subsidiary heart sounds follow-

Fig. 2.—December 6th, 1903. The same, with slower pulse-rate, showing irregular length of diastolic interval.

ing the left ventricular systole at times numbered three or four, the more forcible of them being associated not only with sound but with impulse; the sounds could be heard both over the heart and over the large veins at the root of the neck; in detecting the sounds the straight stethoscope was found to be distinctly more useful than the binaural.

The prognosis was necessarily grave, and the patient was enjoined to avoid all stress calculated to affect him injuriously, as experience shows that the termination of such cases is frequently in fatal syncope.

The subsequent history of the case need not be given in detail, the physical signs remained practically the same;

Fig. 3.—December 12th, 1903. The same; pulse 32, and more regular.

at times the patient felt better, and had more regular pulsation with fewer syncopal attacks; at other times the intermissions were well marked, and the longest duration of such an intermission, timed by myself, was eight seconds, while the pulse rate varied from 18 to 40.

It was noticed that, on the whole, when the heart was slowest, which was usually when the patient was in the recumbent position, the subsidiary sounds were most numerous, but they were never registered by the sphygmograph, which only recorded the systole and diastole of the left ventricle.

On April 17th the patient, who had improved so much in health that I had not seen him for some weeks, went to his bath-room, and at some period of his ablutions evidently fainted and died suddenly, as his head and shoulders were found

under water, and the rest of his body out of the bath. No post-mortem examination was permitted.

Remarks.

The patient was treated with various drugs, including strychnine, with and without strophanthus, mercury perchloride, and trinitrin; strychnine, on the whole, appeared to be most beneficial, while trinitrin was abandoned, as it seemed to have somewhat an injurious effect. The accelerated influence on the heart's action of either stimulants, exertion, or trinitrin was very slight. This is in accordance with the usual experience in these cases.

The chief point of physiological and clinical interest appears to be the undoubted evidence the case affords of an independent action of the two sides of the heart; it will be noticed by reference to the sphygmographs that the descending or diastolic position of the curve shows no evidence of subsidiary pulsations communicated by the heart, and yet the ear and the hand detected secondary impulses which could not have failed to have been registered by the instrument had these originated in the left heart. The conclusion is that the sounds and impulses referred to were produced in the right side of the heart during the diastolic pause of the left ventricle.

THE ALBUMINURIA OF ADOLESCENTS.*

By CLEMENT DUKES, M.D. LOND., F.R.C.P. LOND.,

Physician to Rugby School and Senior Physician to Rugby Hospital.

DURING the brief time at my disposal I propose to submit for discussion a subject at which I have worked for a lifetime with ever-widening interest and instruction—the albuminuria of adolescents.

I do not mean the albuminuria which occurs during adolescence, for that would include scarlet fever and diphtheria nephritis. I intend that specific form to which an infinite variety of names has been assigned, such as "early," "latent," "functional," "cyclic," "intermittent," and "postural." Each of these terms connotes some feature of the ailment; but the most appropriate name I leave to you to decide. For myself, I merely describe the disease as I have found it among boys and girls at school, where it is most commonly to be observed.

The subject is obviously one which intimately concerns every member of the medical profession, whether he be consultant or general practitioner. The interpretation of its significance is still open to debate, and I feel that it ought to be discussed in all its bearings. For without precise knowledge great hardship is apt to be inflicted upon the young who are affected by this form of albuminuria.

In confirmation of the latter observation I mention the following facts:

1. Boys exhibiting the signs of the malady are sometimes sent from school on the ground that they are thus unsuited for the "rough and tumble" of school life. Only a year ago a lad, a patient in whom I was interested, was discharged from school, "because the doctor would not be responsible for him if he were allowed to remain." During his stay at school proteids were removed from his diet; and yet he was expected to do his work, and also to provide suitable material for his growth. I admit that at one moment his urine would boil like boiled white of egg, though in half an hour not a trace of albumen might be perceptible. The lad was in good health, and the condition of his kidneys indicated no permanent detriment to him—nothing beyond the present loss of albumen. I have no doubt that he will develop into a vigorous man, free from organic kidney disease.

What a blight upon a young life to be removed from school associations and games and from the comradeship of school-fellows so essential to growth in manliness, and doomed to the solitariness of a private education and to recreation suitable for an adult instead of a boy!

The only case of this description which I have ever had occasion to remove from school was an only child and fatherless. He was a feeble, delicate, overgrown boy, continuously ailing, and albuminuria was only one of his many weaknesses. I sent him, with a tutor, round the world, and he returned a strapping fellow, hearty and strong, and free from albuminuria. All his troubles apparently arose from excessive growth, entailing too severe a strain upon a somewhat feeble constitution.

* Read before the South Midland Branch of the British Medical Association at the Annual Meeting held at St. Albans on June 22nd.