affections such as angina pectoris, and even in such cases nitroglycerine, morphine, and atropine are better and more easily regulated remedies. Alcohol is a protoplasmic poison, and under no circumstances can it strengthen the cardiac muscle. It lowers the arterial and raises the venous pressure, and increases the diastolic tension within the heart. In this way it acts as a cardiac stimulant, but as it has a paralysing effect on the cardiac muscle there is a loss of tone with tendency to dilatation of the heart; hence the large, flabby, fatty hearts of beer-drinkers, and the great retention of fluid in their vessels and tissues. Tobacco is a nervine sedative and cardiac depressant. Cases of heart disease do better without it.

CONCLUSION.

This address has already assumed such lengthy proportions that I must defer the treatment of that large class of cases of chronic degenerative lesions of the vessels, heart, and its valves until some future occasion. I have told you that a modern authority says that "cardio-sclerosis is progressive, and we cannot stay it," so I presume the other branch of my subject can afford to wait. On the other hand, I am inclined to think that if you follow in the lines which I have already laid down, you may not merely stay the progress of these degenerative lesions, but, what is infinitely more important, you may often prevent their occurrence.

REFERENCES.

¹ *Diseases of the Heart and Aorta*, 1854, p. 357. ² *Therapeutics of Circulatory Derangements*, by Professor M. J. Oertel, M.D. Translated by Edward J. Edwardes, M.D.

A CASE OF HEART-BLOCK, WITH FIBROUS DEGENERATION AND PARTIAL OBLITERATION OF THE BUNDLE OF HIS.

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THE following are the notes of an interesting case of heart-block in which the auriculo-ventricular bundle of His was almost completely degenerated. The case is unique, inasmuch as a complete heart-block has not, so far as I know, been previously observed as the result of rheumatic fever; cases of slight heart-block after rheumatism have been described (see, for instance, Cases XVIII and XXIII, pp. 324 and 339, Mackenzie's *Diseases of the Heart*).

Dr. James Mackenzie has kindly read over the case, and given me his opinion with regard to the tracings. Unfortunately the tracings which were taken of the radial pulse and some of the tracings from the neck, showing the venous and carotid pulses, have been lost.

A Case of Heart-block: very slow pulse: fibrous degeneration of the auriculo-ventricular bundle of His; calcareous nodule situated at the right end of the attachment of the anterior mitral flap extending into the septum, invading and almost completely obliterating the bundle towards its auricular extremity.

Clinical History.

J. F., aged 29, married, formerly a worker on the railway, was admitted to the Edinburgh Royal Infirmary on April 14th, 1907, complaining of pain in the joints, chest, and region of the heart.

Previous History.

The patient has had six attacks of rheumatic fever, at the ages of 15, 17, 20, 22, 23, and 26 respectively; since the first attack of rheumatic fever, when the heart is said to have been affected, he has been short of breath on exertion.

Three years ago, when shunting on the Caledonian Railway, he fell in front of a train; his left hand was crushed, and the left arm had to be amputated. He has not worked since this date.

The present attack of rheumatic fever commenced fourteen days ago; up to that time he was feeling quite well.

Family History.

Unimportant.

Social Conditions and Surroundings.

Fairly good. Formerly a heavy smoker, has smoked much less lately; a teetotaler.

Condition on Admission.

Almost all the joints are swollen and painful. Temperature on the morning of admission 101.8°, on the evening of admission 103°; pulse 106; respirations 34.

Cardiac pulsation seen over an extensive area; apex beat in the sixth left interspace, $2\frac{1}{2}$ in. below and $\frac{1}{2}$ in. outside the mammary line (4 in. from the sternum); a long, loud, harsh systolic murmur and a soft diastolic murmur heard in the aortic area; a long systolic murmur in the mitral area. Radial pulse 106; tension medium; arterial coats not thickened. Lungs normal. Tongue furred; slight constipation; liver not enlarged. Complaints of some headache but sleeps fairly well. Urine: specific gravity 1014; no albumen; a heavy deposit of urates. The red blood corpuscles number 3,920,000, the haemoglobin equals 70 per cent., and the white corpuscles number 7,400.

Treatment.

Sodium salicylate 20 grains, three times daily; milk diet.

Progress.

Rapid improvement; disappearance of joint pains.

On April 7th the temperature was normal and the patient was feeling very well.

On April 10th the patient woke at 2 a.m. with severe pain over the region of the heart; at 6 a.m., while washing himself, he suddenly became weak and breathless. On examination the pulse was found to be beating at the rate of 30 per minute. The patient vomited after breakfast. When seen by me at 10.30 a.m. the radial pulse numbered 26 per minute; the temperature was 99.2°. The cardiac ventricles were contracting at the rate of 26 per minute. The right side of the neck looked full (apparently due to distension of the right internal jugular vein); the right external jugular vein was distended and distinctly pulsating; the venous pulse on the right side of the neck numbered from 80 to 90 beats per minute; the pulsation of the right common carotid artery was forcible and numbered 26 per minute. The right radial pulse was small and feeble. The apex beat of the heart was in the sixth interspace, $2\frac{1}{2}$ in. below and $\frac{1}{2}$ in. external to the nipple line. Pulsation could also be seen and felt in the fourth and fifth left interspaces; the pulsation in the fourth left interspace seemed to occur a little later than the apex beat. A very long and loud systolic murmur was audible in all the cardiac areas; it was perhaps a little louder in the tricuspid area than elsewhere. This systolic murmur was immediately followed by a short murmur and then (that is, after the short murmur) by a sound which seemed to be the second sound. I was not able at the time to interpret the meaning of the second (? diastolic) murmur and of the sound (? second sound) which followed it. I simply state the facts as I thought I heard them—the description was dictated to my house-physician at the bedside.

The pupils were equal and measured $2\frac{1}{2}$ mm. in diameter. With each pulsation of the cardiac ventricle and slightly after it, a quick jerking movement of the head, sometimes double in time, occurred as a result of contraction of the neck muscles. This jerking of the head was a curious phenomenon which, so far as I know, has not been noted in any case of heart-block before, possibly it was due to reflex stimulation of the neck muscles through the vagus.

Some of the tracings, taken on April 10th and 11th, of the pulsation in the neck and heart's impulse are reproduced in Figs. 1 to 7.

Fig. 1, which is a tracing from the neck, shows both the venous pulse due to contraction of the right auricle and the carotid pulse. The small waves (a) in the tracing are chiefly

Fig. 1.—Tracing of the movements in the neck due to the carotid and jugular pulses. The small waves (a) are due to the auricle, and the large waves (a + c) are due to the carotid—an auricular wave in the jugular occurring at the same time as the carotid pulse (see diagram, Fig. 2).

auricular and are superimposed on the carotid pulse when it appears. The big waves are a combination of the auricular and ventricular (carotid) contractions (c + a). The other neck tracings also show the auricular waves. Fig. 2 is a diagram showing more clearly the independent rhythm of the auricle

Fig. 2.—Diagram representing the movement shown in Fig. 1. The perpendicular lines in the upper space represent the systoles of the auricle (As) as shown by the waves a; those in the lower space represent the systoles of the ventricle (Vs) as shown by the carotid beats. The relation of the Vs to the As is constantly varying, showing therefore that the systole of the ventricle occurs independently of the stimulus from the auricle.

and ventricle. Fig. 3 is a tracing from the left ventricle; it shows the rise characteristic of the ventricular systole. The small waves in this tracing are due to contraction of the left auricle. Fig. 4 is a tracing partly from the left ventricle (the

upstroke) and partly from the right ventricle (the fall). This tracing shows that both ventricles shared in the slow action.

In these four tracings (Figs. 1, 2, 3, and 4) there seems to be graphic evidence of the contractions of all four chambers of the heart. The small waves (a) in Fig. 1 are jugular waves, due to contraction of the right auricle; the small waves in Fig. 3 appear to be due to contraction of the left auricle; the contractions of the left ventricle are shown in Fig. 3, taken from the (left) apex beat; and the contractions of the right ventricle are shown in Fig. 4.

Fig. 3.—Tracing from the apex, showing the infrequent contractions of the left ventricle. The small waves between the large beats are due to the systole of the left auricle.

Fig. 5 appears to be wholly right ventricle and shows the inverted cardiogram in contrast with that represented in Fig. 3 from the left ventricle. Fig. 6 is, no doubt, left ventricle and shows the long pauses with auricular waves very distinct. Fig. 7 is a simultaneous tracing of the apex beat and of the pulsation in the neck; it was made on April 11th.

Fig. 4.—Taken over the right ventricle, showing a great fall during systole (the inverted cardiogram characteristic of the systole of the right ventricle); it also shows that the right ventricle participated in the infrequent action with the left ventricle.

April 11th. The patient appears to be slightly better this morning. The ventricular contractions and the radial pulse number 30 per minute. No change in the strength and character of the heart beats; a long, loud systolic and a short diastolic murmur still present; the radial pulse is rather weaker. During this day the patient had several attacks of very severe angina

Fig. 5.—Tracing from the fifth left interspace, made on April 11th, 1907. This tracing appears to be wholly due to the right ventricle, and shows the inverted cardiogram in contrast to Fig. 3.

pain; he stated that he felt as if he were being crushed between two buffers. The radial pulse during the attacks of pain was almost imperceptible and somewhat irregular. The jerking movements of the head with the apex beats of the heart still continue. There has been a great deal of vomiting. Amyl nitrite inhalations, strychnine subcutaneously, and a menthol mixture were prescribed.

Fig. 6.—Tracing from the apex beat of the heart made on April 10th, 1907. This tracing appears to be due to contraction of the left ventricle, and shows the long pauses between the ventricular contractions with small auricular waves interposed.

April 12th. Much worse; very restless all day. Pulse down to 24 per minute. The pain in the heart has been very severe and distressing. Morphine has been given at frequent intervals. Towards midnight the pulse went down to 22. The vomiting has not been so bad to-day. The pupils now measure from 5 to 6 mm. in diameter. The patient is perfectly sensible.

Fig. 7.—Combined tracing from the apex beat and neck made on April 11th, 1907.

April 13th, 2 a.m. The cardiac pain was so severe that morphine had to be given on several occasions. Pulse 19; respirations 17. An interval of 8 seconds was counted between two of the cardiac beats on one occasion. The radial pulse is very irregular. The patient died at 4.30 a.m.

Post-mortem Examination.

This was made by Dr. Shennan on April 13th, 1907. Both lungs were in places attached to the chest walls by old adhesions. The pericardial sac was obliterated as the result of old

pericarditis. The exterior of the pericardium was adherent to the thoracic wall opposite the apex of the heart.

The heart was injected with formalin in order that the auriculo-ventricular bundle might be carefully dissected out.

On passing the fingers through the auricles a large calcareous mass could be felt, on the upper border of the inter-ventricular septum posteriorly, apparently measuring about 1½ in. antero-posteriorly by about ½ in. vertically and about 1½ in. transversely. On further examination, after fixation, this corresponded to the anterior cusp of the mitral valve, which was extremely calcareous.

There was some bronchitis and congestion of the lungs, but no consolidation. The liver was enlarged, congested, and fatty. The kidneys were large and congested, with some swelling and fatty change. The spleen was firm, large, and congested.

Subsequent Examination of the Heart.

General adherent pericarditis; dilatation of all the cardiac cavities; hypertrophy of the ventricles; old endocarditis with calcareous deposits in the aortic and mitral valves; the anterior flap of the mitral valve markedly calcareous; some narrowing of the mitral orifice; thickening of the tricuspid with some dilatation of the orifice; pulmonary cusps slightly but uniformly thickened.

Dr. E. B. Jamieson of the Anatomical Department kindly dissected out the auriculo-ventricular bundle. His report on its condition is as follows:

The auriculo-ventricular bundle of His has apparently undergone fibrous degeneration, and the few muscular fibres remaining on the surface are very pale. A calcareous nodule situated at the right end of the attachment of the anterior mitral flap extends into the septum, invades and well nigh obliterates the bundle towards its auricular extremity.

NOTE ON THE PULSUS BIGEMINUS.

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My object in publishing this note is to draw attention to a curious, and I believe hitherto undescribed, form of jugular pulse associated with the pulsus bigeminus, which in itself is not a very uncommon phenomenon.

The patient, a man aged 40, was first seen on June 27th, 1906. He had been working at his trade as a painter up to a week before. He had had heart disease to his knowledge for seven years but had never been laid up on this account. At one time he weighed 16 st., but for some months he had been losing flesh. He had been temperate as regards both alcohol and tobacco.

He suffered greatly from dyspnoea, which necessitated his being propped up in bed. He was pale and flabby, and there was considerable oedema of the legs and trunk but no ascites. The pulse was 100, regular, with a large, markedly collapsing beat, and the marked throbbing of the arteries, characteristic of aortic disease, was very noticeable, and perceived by the patient himself. The heart was enlarged to the right, and to the left to 1 in. outside the nipple line. There were double murmurs at all areas of the heart but no thrill could be felt. It was obviously a case of failure of compensation in aortic disease. Tracings taken at this time showed that the pulse was quite regular, while there was a very large auricular wave in the jugular pulse, which was otherwise normal. He was much troubled at night with cardiac asthma, and the usual drugs were tried, without any but temporary relief to this symptom. In spite of treatment by rest in bed and cardiac drugs the oedema and other symptoms of distress persisted, and he died on July 31st. For the last fortnight he was taking 5-minim doses of tincture of strophanthus with citrate of caffeine.

A week before his death his pulse took on a bigeminal character, which was continuous, and persisted until the day before he died, when it became too feeble to detect its true rhythm. The tracing of the jugular and radial pulse when this condition was present is shown in the accompanying figure, from which it will be seen that at the extra beats which are the cause of the bigeminal rhythm, the auricles contracted during the ventricular systole, and there is one large wave synchronous with the extra beat in the radial artery. The increased size of this wave is due, as Mackenzie has pointed out, to the fact that the right auricle is unable to empty itself into the contracted ventricle, and therefore a larger quantity of blood than usual is sent into the jugular veins. The unusual feature in this tracing lies in the fact that the combined wave commences distinctly beyond the middle line—that is, nearer to the post-extra-systolic auricular wave than to the pre-extra-systolic. This puzzled me so much that I sent this and another tracing of two days' earlier date to Dr. James Mackenzie, who most kindly examined them