CASE II.

A woman, aged 65, was admitted in November, 1925, in a state of epileptic confusion and restlessness. There was a history of epilepsy for the previous thirty years. On admission the cardiac dullness was normal, but the heart sounds were soft and faintly heard; the action was regular in rate and rhythm. On May 14th, 1926, she complained of pain in the chest, vomited, and breathed with difficulty; the next day the temperature rose to 101° F., pulse 120, respirations 38, the pain in the chest was acute, dyspnoea marked, and the face extremely congested. Fever continued for six days, and the pain and dyspnoea slowly passed off. The patient apparently recovered and was up and about from May 25th until June 6th, when she again complained of pain over the heart. From June 24th there was fever ranging from 99° to 100.4° F., pulse 80 to 100, respirations 20 to 40; dyspnoea, cardiac pain, and cyanosis until June 27th, when there was sudden syncope and death.

Unfortunately, there is no record of the character of the pulse, cardiac dullness, sounds and action, or of the presence or absence of pericardial friction, etc., and the case was not diagnosed during life. The Wassermann reaction was negative.

Post-mortem Examination.

The heart was hypertrophied and weighed 481 grams (17 oz.); length from aortic ring to apex, 10 cm.; mid-ventricular diameter, 8 cm.; left ventricular wall, 2 cm.; right ventricular wall, 0.75 cm. thick. The pericardial sac was distended and contained over two pints of fluid blood; clot was wrapped around the heart like a cast. The pericardium was adherent around the apex and to the anterior surface of the right and left ventricles over an area 6.5 cm. wide. Blood oozed from the left ventricle along the edges of the pericardial adhesions. There was infarction of the apex of the left ventricle involving the myocardium for a distance of 4 cm. along the interventricular septum. A laminated thrombus 3 cm. thick filled the apex. The myocardium appeared healthy; the anterior cusp of the mitral valve and the aortic cusps were thick and fibrous; the valves were competent. The aorta was atheromatous at the arch.

The left coronary artery was healthy at its origin, but 1 cm. from its mouth the vessel was thrombosed for a distance of 2.5 cm. The right coronary artery was healthy.

Microscopical Examination.—The left coronary showed irregular "signet ring" proliferation of the intima, with final occlusion of the lumen by thrombus which was being organized. The infarct showed necrotic muscle fibres. There was active phagocytosis by large multinuclear cells and leucocytes; bands of white fibrous tissue replaced the dead muscle fibres, and the

FIG. 2.—Case II. Section through left ventricle; (a) adherent pericardium; (b) infarct; (c) laminated thrombus.

pericardium adhered by a delicate fibrous tissue meshwork, in which there were small haemorrhages. The lungs were healthy. The kidneys were pale and fatty, and the vessels sclerotic.

There were no marked signs of cardiac disease until coronary occlusion occurred; then there was sudden acute cardiac pain, dyspnoea, vomiting, and fever lasting ten days. The patient apparently recovered and was able to be up and about for a period of four weeks; meantime ischaemic necrosis had occurred. Pericardial adhesions formed over the infarct and were apparently efficient for four weeks, when, presumably, stretching of the infarct took place with bleeding into and through the softened tissue to the pericardial sac along the line of the adhesions, resulting in haemopericardium. Hamman³ considers that it is exceptional for a softened infarct to rupture and cause death soon after occurrence of the occlusion.

I am indebted to Dr. J. Francis Dixon, medical superintendent, for permission to publish particulars of the above cases.

REFERENCES.

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² Crawshaw: *Ibid.*, 1927, ii, p. 99. ³ Hamman: *Bull. Johns Hopkins Hosp.*, April, 1926. ⁴ Thayer: *Internat. Clin.*, Philad., ser. 33, No. 1, pp. 1-26, 1923. ⁵ Gibson: *Lancet*, 1925, ii, p. 1270. ⁶ Scholz: *Arch. Int. Med.*, vol. 34, 1924.

AURICULAR FLUTTER.*

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AURICULAR flutter is an abnormal heart rhythm the diagnosis of which is difficult in the absence of instrumental methods, but usually possible if certain points are considered.

The fundamental factor of the condition is that the auricles start a very rapid rate of contraction, somewhere in the neighbourhood of 300 times a minute. The diastolic period allowed at that rate is so short that the ventricles are not properly filled and their output is diminished. The ventricles are usually unable to follow the auricular

FIG. 1.—Shows auricular and ventricular rate at 264. 1-1 flutter.

rate owing to the conductivity of the bundle of His being unequal to such rapidity of function, and for that reason every other stimulus is held up by the bundle and a condition of 2-1 heart-block is set up; this is really a protective heart-block, which is useful in that it protects the ventricular muscle from exhaustion following on an extreme tachycardia. Exercise, which stimulates the sympathetic, is known to increase the conductivity of the bundle, and sometimes it is found that exercise may cause the ventricles to follow exactly the auricular rate for a short time (Fig. 1).

Usually a 2-1 block is present, so that the ventricular rate is about 150 a minute; and this rate is very constant—it remains the same whether lying, standing, or even moving about. Sometimes the ventricular rate is about 75—a 4-1 block—and this is where we are very apt to miss the condition on a cursory examination, but really it is the best chance for diagnosis. If exercise is given the rate, instead of rising slowly, jumps with a sudden bound to double the original rate—namely, about 150—or in some cases it may become quadrupled to about 300. This is characteristic of flutter, and there are only two conditions where this may occur: in a trained athlete on exercise the rate may rise with a jump from about 60 to 120, and on rest the same thing may occur in the opposite direction; auricular flutter is the other. It is commonly said that this may occur in paroxysmal tachycardia, but, although sudden jumps of rate do occur at the onset and offset of the attacks, the increase is not a multiple of the former rate.

In flutter an intermediate rate may occur, for the block may vary between 2-1, 3-1, and 4-1; in this case there is ventricular irregularity, and the clinical diagnosis then becomes still more difficult. The symptoms to be expected are some degree of heart failure if the rate be much above normal, owing to deficient ventricular output, yet, clinically, at rest the heart may appear to function properly. The size of the heart may be little if at all increased; the rate may, as we have seen, be about normal. Occasionally a constant ripple may be seen in the jugular vein in the neck, corresponding to the auricular contractions, but this is not often present. The chief points for diagnosis are an unvarying rate over long periods, unless by doubling or quadrupling, associated with breathlessness.

The condition may occur in apparently healthy or in obviously diseased hearts; it is essentially a similar condition to auricular fibrillation, the former being a regular tachycardia, whereas the latter is an entirely irregular

* Received for publication June 20th, 1927.

tachycardia. The one condition may very easily pass into the other, and, in fact, may be made to do so by means of drugs.

The treatment consists of the administration of digitalis or quinidine, or both at the same time; its results will be demonstrated in the cases recorded below.

CASE I.

A strong, healthy-looking man, aged 28, was invalided out of the army with disorderly action of the heart. He received a pension for five years, and then it ceased as he was considered well. Eighteen months later he developed extreme tachycardia on exertion and was sent to my out-patients' department. The size of the heart was doubtful, but a radiogram showed that it was very much enlarged. There was a soft systolic murmur at the

FIG. 2.—Shows 2-1 block in auricular flutter. P, auricular waves; R, S, and T, ventricular waves.

apex. The rate was 132 at rest, but this rose to 264 on exertion. The condition was manifest. An electro-cardiogram showed 2-1 flutter at rest (Fig. 2), but 1-1 flutter on exertion (Fig. 1). He was admitted and placed on tincture of digitalis, 20 minims thrice daily, and in a few days the ventricular rate was slowed owing to a mixture of 2-1, 3-1, and 4-1 block (Fig. 3). Digitalis was continued (15 minims thrice daily) for three weeks, with no effect but that the block became 4-1. Quinidine was then given.* The condition changed from flutter to normal rhythm after the third capsule, so no more was given; flutter, however, recurred the next day. A further course of quinidine was ordered, and again he became normal after the third dose, but this time he was given two capsules the next day, and then one capsule a day for fourteen days. He remained at normal rhythm and was discharged. He continued well for eight months, but was then taken ill again after walking up a steep hill. Flutter was again present. He was once more put on digitalis, and this time on the third day the condition changed to auricular fibrillation (Fig. 4). The digitalis was at once stopped—for if one can force flutter into fibrillation the heart often lapses into normal rhythm as the effect of the digitalis wears off; however, he relapsed into flutter. Digitalis was again administered, and quinidine was given, but

FIG. 3.—Shows a mixture of 2-1, 3-1, and 4-1 block. P, auricular waves; R and S, ventricular waves.

the full five doses had no effect. Digitalis was continued and the condition again changed to fibrillation. On stopping the drug this time he regained normal rhythm; this has remained so till the present time, a matter of four months (Fig. 5).

The next two cases came on after an attack of influenza.

CASE II.

A woman, aged 36, was sent to hospital by her doctor, who described her as a case of heart failure which was obscure, as the heart did not appear clinically very bad. She had influenza in 1922, and six months later her feet began to swell. A year later she had ascites, oedema of the legs, and palpitation, which gradually got better, but she remained very breathless. She was admitted in June, 1924. The heart was enlarged to a slight degree, there were no murmurs, and the pulse rate was 144 per minute. A tracing showed 2-1 flutter. Digitalis changed the block to 4-1, but nothing else happened; the rate was then 72 a minute. She was given quinidine, but the second capsule made her feel very ill and it was stopped. She therefore remained

* The method is as follows: a preliminary dose is given of 0.4 gram in a gelatine capsule, to see that there is not an idiosyncrasy against the drug, and the next day the patient is given 0.4 gram every two hours for five doses.

on digitalis, and I went on holiday. On my return I was informed that vomiting had occurred, and therefore she had been taken off digitalis. Examination showed that the rhythm was normal, and it has continued normal till the present time.

CASE III.

A boy, aged 17, had an attack of influenza during which he was taken with vomiting and extreme tachycardia associated with much dyspnoea, but no signs of heart failure. The tachycardia continued for eight months. I saw him in August, 1921. The heart was not enlarged and there were no murmurs. The pulse rate at rest was 144, but on exercise this went up to 288 with pulsus alternans, as shown by the sphygmomanometer. Auricular flutter was diagnosed, but no electro-cardiogram was available at that time. He was given digitalis, which brought the rate down to 72, and he was kept at this rate for a long time. Holiday time came on and I went away. On my return I found the

FIG. 4.—Shows auricular fibrillation. The ventricular rhythm is quite irregular. f, fibrillation waves in diastole.

rate at 60, and, judging that the flutter had ceased, the patient was allowed up, and it was found that the tachycardia had passed off. He was sent up to London to see Dr. Parkinson, who reported that the electro-cardiogram was normal and that the heart also appeared normal. Since then the boy has never looked back; he has played Rugby football and gone through his course as a medical student. He appears to have a normal heart. It was unfortunate that I missed the turn over of both these cases, but probably the flutter was followed by fibrillation and then reverted to normal rhythm.

CASE IV.

A man, aged 50, had suffered with emphysema of the lungs with cyanosis for some years, but for the last four months had oedema of the legs with a sensation of fluttering in the chest. His heart was enlarged, the sounds were faint, but there were no murmurs. The pulse was irregular, and a tracing showed auricular flutter with a varying block—3-1 and 4-1. Digitalis kept him at a regular 4-1 block, but did nothing further. He was given quinidine, five doses on two consecutive days, with no effect, so digitalis was pushed till fibrillation ensued. On stopping the digitalis the flutter returned, but on repeating the process the

FIG. 5.—Shows normal rhythm. The ventricular waves are the same, R and S. There is only one P wave occurring just before the R wave.

rhythm returned to normal and he was discharged. Six weeks later he relapsed and flutter reappeared.

CASE V.

A man, aged 37, had had rheumatic fever at an early age, with three recurrences since, the last being in August, 1926. He was admitted on September 1st, 1926, suffering from gross heart failure with a very rapid regular pulse of 182 a minute. An electro-cardiogram showed flutter with 2-1 block. This is a very high rate for a flutter, for the auricular rate was 364. The next day the heart was found to be grossly irregular, and the condition had changed to fibrillation. As he had advanced mitral stenosis it was thought he would do better if the fibrillation was made permanent, so he was given digitalis and the rate slowed to about 75 a minute, and was kept at that rate by a dose of 15 minims of tincture of digitalis thrice daily. He did very well, and has since gone back to work, which is luckily not of an arduous nature.

The number of cases here reported is too few upon which to base any remarks, but it is interesting to note that the two influenzal cases are the only ones which have not recurred—one over a period of three and the other of six years. The probability is that they will not now recur.