ruptured ectopic gestation if they occur in a woman who has had a long period of sterility, or a history of pelvic disease with irregular menstruation, and especially if menstruation has ceased for six weeks or so, during which the early signs of pregnancy may be seen, and at the end of about six weeks there is a discharge of clots from the vagina, with colicky pain in the lower abdomen.

The treatment is immediate operation, and it is surprising how well these patients do afterwards, even though they may be, and usually are, terribly blanched at the time. I have occasionally given a transfusion of blood, and it seems to help. Blood transfusion may be of the greatest value in those cases where the bleeding is due to injury.

I have not gone into a detailed differential diagnosis of all the surgical lesions included under the heading of the acute abdomen; it would make the paper much too long. In the early stages there is usually little difficulty in deciding which organ is responsible. Again, I have refrained from discussing abscesses and peritonitis. These are complications, and the acute abdomen should be operated on before they appear. If a patient suddenly experiences acute abdominal pain, followed by nausea or vomiting, or both, and this is accompanied by tenderness and rigidity, possibly in some degree over the whole abdomen, but most pronounced over the most painful area (which is suggestive of the site of the lesion), with or without shock, or if with the pain and vomiting there is absolute constipation, there is an acute abdominal condition present which is almost certainly of a surgical nature. If the lesion can be accurately placed so much the better; but this does not matter so much to the surgeon as getting the case early, for he should not tackle the job at all if he is not able to deal with any surgical catastrophe which may be exposed on opening the abdomen.

TWO CASES OF CARDIAC INFARCTION:

ONE FOLLOWED BY CALCIFICATION OF THE HEART,
THE OTHER BY RUPTURE.

BY

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DURING the past two years there has been a considerable increase in the literature on coronary occlusion, cardiac infarction, and rupture of the heart. The two cases to be described have been put on record owing to certain unusual and interesting features.

CASE I.

A man, aged 55 (shoe hand), was admitted in June, 1901, in a state of acute excitement with hallucinations. He slowly became demented, but led an active and useful life as messroom worker, vanman, and hospital messenger. In August, 1925, at the annual physical examination, the heart was found to be enlarged to the left nipple line; there was a soft mitral systolic murmur and occasional extra-systoles. The lungs were clear. On November 13th, 1925, he was confined to bed for a few days with bronchitis and slight oedema of feet and legs; there was no albuminuria. He was soon up and about again, working as usual. On the evening of December 12th, 1925, he returned from afternoon leave and complained of not feeling well. He went to bed and slept for an hour; at 10 p.m. he went to the ward commode, collapsed, and died at 10.3. There was no record of the Wassermann reaction.

Post-mortem Examination.

The heart was greatly hypertrophied and weighed 809 grams (28½ oz.); length from aortic ring to apex, 14 cm.; mid-ventricular diameter, 12 cm.; left ventricular wall, 2.5 cm. thick; right ventricular wall, 0.9 cm. thick. The pericardium was much thickened, and was adherent around the apex over an area 8 cm. wide; a plentiful supply of blood vessels passed from the adherent pericardium to the apex. There was a small quantity of straw-coloured fluid in the pericardial sac. The lower half of the left ventricle was ballooned out sufficiently to contain a golf ball. The anterior wall of this dilatation was fibrous, with scattered patches of calcification and extensive areas of recent sub-endocardial haemorrhage. The apex and the left lateral, medial, and posterior walls were fibrous, with marked calcareous deposit which extended 7 cm. along the interventricular septum. There was formation of fibrin on the calcareous nodules that projected here and there through the endocardium, which otherwise was intact, and lined the irregular inner aspect of the dilatation. The myocardium was brownish and soft, and the papillary muscles much hypertrophied. All the chambers were dilated and hyper-

trophied. The aortic cusps were thickened, and the mitral thickened and incompetent. There were a few small patches of atheroma in the ascending aorta.

The left coronary artery was healthy at its origin, but just inside the mouth there was atheroma with early calcareous deposit; 3 cm. from the origin of the main vessel there was complete occlusion of the anterior descending branch for a distance of 2 cm., and beyond that the walls were greatly thickened, with narrowing of the lumen. The right coronary artery showed slight atheroma.

Microscopical Examination.—The left coronary artery was atheromatous and occluded by organized thrombus. There was great proliferation of the intima, and fibrous tissue was arranged in whorls around small vessels formed during the canalization of the thrombus. Yellow granules of haemosiderin lay amongst the fibrous tissue. The muscle fibres of the myocardium were irregular

FIG. 1.—Case I. Section through calcified cup in left ventricle: (a) adherent pericardium; (b) calcareous nodules; (c) recent haemorrhage.

in size, stained indifferently, and many were dotted with black granules; fibrous tissue was increased between the muscle fibres. At the apex the muscle fibres were very scanty, and there was abundant white fibrous tissue overgrowth. The lungs showed signs of chronic bronchitis and old pleural adhesions. The kidneys were in a state of chronic interstitial nephritis.

Close inquiry revealed no history of any illness nor complaint on the patient's part to point to occlusion of the coronary artery, yet the anterior descending branch had been blocked and extensive infarction of the inferior half of the left ventricle, including the septum, had occurred. The infarct had undergone softening and been replaced by fibrous tissue which had stretched, producing aneurysm of the heart. Extensive calcareous deposit had occurred in the dead heart muscle, and judging from this the patient must have carried on with a damaged heart for a considerable time, possibly years, until he presented signs of heart failure in November, 1925, from which he apparently recovered, but which was followed by sudden death, possibly whilst straining at stool. Goodall and Weir¹ report three cases of coronary disease and rupture of the heart in patients who died suddenly in or near lavatories. Crawshaw² reports another case of ruptured ventricle in similar circumstances.

Hamman³ considers that occlusion may occur so slowly that no symptoms mark the final occlusion. Thayer⁴ quotes cases of recovery for periods of three years and thirteen years after infarction, and states that ultimately death may be sudden, or from gradual heart failure. The case described certainly bears out these points.

Calcification of the heart is rare. In Gibson's⁵ collection of forty-three cases of infarction from English, American, and Continental literature there is no example of calcification of the infarct. Scholz⁶ describes a case of calcification of an infarct following coronary occlusion in a male, aged 74, demonstrated radiographically *intra vitam* and confirmed *post mortem*. He remarks that where calcium salts are deposited in a cardiac infarct "the calcifying process gradually takes on a massive solid form involving a large area and all the elements of the heart wall. It may be worth noting that this apparent elimination of so large a portion of the heart muscle causes very little distress to the patient unless the conducting system of the heart happens to be interfered with by the calcifying process." Scholz also quotes a case of coronary occlusion, followed by calcification of the infarct, described by Burns in 1809, and a case described by Askanazy in 1901.

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.

- ¹ Goodall and Weir: *BRITISH MEDICAL JOURNAL*, 1927, i, p. 834.
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.*

BY

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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

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