A CASE OF SUBENDOCARDIAL INFARCTION

BY

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Disease of the coronary arteries, by commoner occurrence or by more frequent diagnosis, is assuming increasing importance in clinical medicine. The following case is reported because it shows some interesting features, and full clinical, cardiographic, and pathological investigation was possible.

A schoolmaster, aged 55, was admitted to hospital on November 16, 1941, with a three weeks' history of increasing angina of effort. Clinical examination showed nothing abnormal except a blood pressure, 155/100; W.R. negative. After admission to hospital he continued to have short attacks of pain relieved by amyl nitrite, after some of which T wave inversion in leads I and IV persisted for several days (Fig. 1). He was nervous and irritable, and attacks were usually precipitated by annoyance over some imagined grievance against other patients in the ward. Cardiograms taken during an attack on December 11 showed inversion of T IV, and 10 minutes after amyl nitrite, recovery of T IV to the upright position (Fig. 2). On December 20, the patient had a prolonged attack of pain not relieved by amyl nitrite, after which attack the cardiogram showed persistent T wave inversion in leads I and IV (Fig. 3).

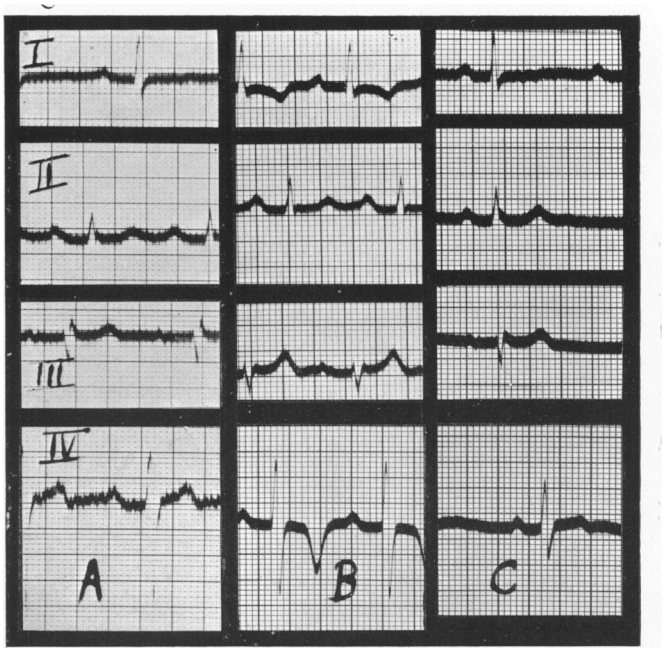

FIG. 1.—(A) Before attack (12/11/41). (B) Three days after attack of pain lasting 2 hours (17/11/41). (C) Return towards normal, 12 days later (29/11/41).

He made satisfactory progress and the blood pressure stabilized at about 150 mm. He started to get up on February 6, six weeks after his infarct.

Two days later, February 8, a severe attack of pain across the chest commenced at 6 a.m., and after failure to obtain relief from amyl nitrite, 1/4 grain of morphia was given and the dose repeated two hours later. At 11 a.m. he was seen by one of us (R. K. P.) and as he was still in great pain another quarter of a grain of morphia was given intravenously. This gave rapid relief and he was alseep within ten minutes. He woke again one and a half hours later and complained of pain and a further 1/4 grain of morphia was given subcutaneously. After this he remained comfortable.

An electrocardiogram taken the following day showed RS–T distortion with elevation

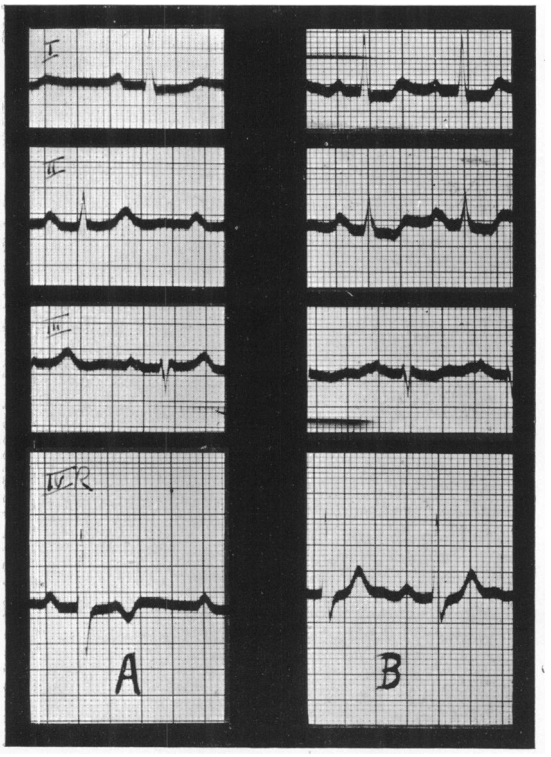

FIG. 2.—(A) During an attack of pain. (B) Ten minutes later, after amyl nitrite (11/12/41).

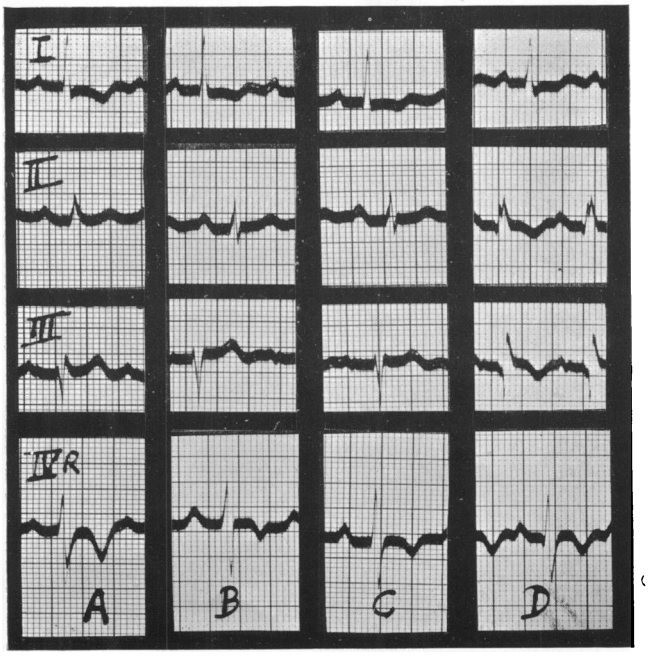

FIG. 3.—(A) Two days after prolonged attack of pain (22/12/41). (B) Before amyl nitrite (5/2/42). (C) After amyl nitrite (5/2/42). (D) One day after attack of pain lasting 5 hours (9/2/42).

in leads II and III, with inversion of T waves. His blood pressure remained low 90–100/70–80, and on February 14, six days later, he began to complain of shortness of breath. The quality of the heart sounds deteriorated and on February 16, gallop rhythm was heard at the apex and rales were detected at the bases of the lungs. The pulmonary œdema cleared temporarily after injections of neptal, but the patient's general condition became worse. On February 26, a blowing systolic murmur became audible at the apex and persisted. He developed acute pulmonary œdema on March 3, and died within a few minutes, three weeks after the second infarction.

SYNOPSIS OF AUTOPSY

The deceased was a heavily-built obese man. No œdema of the feet nor ascites was present. There was acute œdema of the lungs with 140 c.c. of non-fibrinous straw-coloured fluid in each pleural cavity. The liver showed early chronic venous congestion. The kidneys appeared normal. The pancreas was very fatty. A hydrocele of the left testis was present.

The heart was enlarged and weighed 567 g., due to left ventricular hypertrophy. Considerable pericardial fat covered the heart. There was no pericarditis or endocardial exudate. The aortic valve was competent but atheroma was present at the base of the mitral and aortic cusps. Considerable atheroma of the aortic arch and of much of abdominal aorta was found. Atheromatous fatty streaking of the pulmonary arteries and their main branches was present.

The main coronary arteries showed much atheroma but no actual occlusion. There was extreme atheromatous narrowing, one inch from the opening into the aorta, of the right coronary artery, and similar narrowing of the left coronary artery one-third of an inch from its origin.

Focal disseminated fibrosis was present in the left ventricular muscle in its left outer border throughout its length, and also in the anterior wall near the junction with the inter-ventricular septum in the proximal part of the left ventricle near the base of the heart.

There was a large "sheet-like" subendocardial infarct extending from the base of the left ventricle at the auriculo-ventricular ring to the apex, involving the posterior wall of the left ventricle including the posterior papillary muscle, and the interventricular septum throughout its length, and extending on to the anterior wall of the left

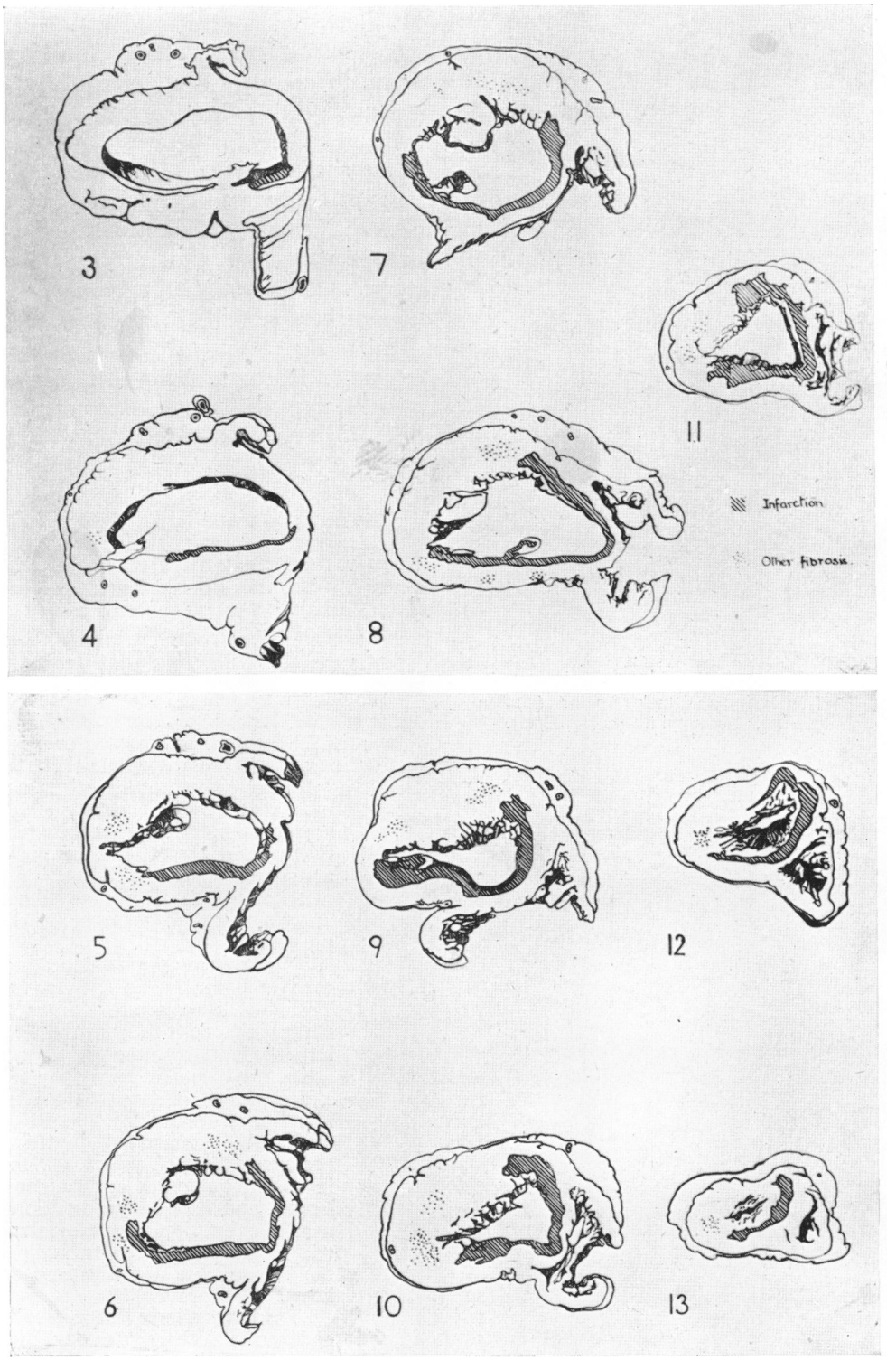

FIG. 4.—Photograph of a series of drawings of transverse sections through the ventricles, made at intervals of approximately 5 mm., extending from the atrio-ventricular ring (No. 3) to the apex (No. 13). The sheet-like recent infarct is shown cross-shaded ; the areas of focal fibrosis, as a series of dots.

ventricle in its middle and distal portions. Whereas the bulk of the infarct was subendocardial, this latter anterior portion moved away from the endocardium to lie intramurally.

This " sheet " infarct was of recent origin and was yellowish-red in colour. Its extent together with the areas of focal fibrosis are shown in Fig. 4. Comparison may be made with the distribution of the subendocardial fibres of the superficial bulbo-spiral muscle in Fig. 5.

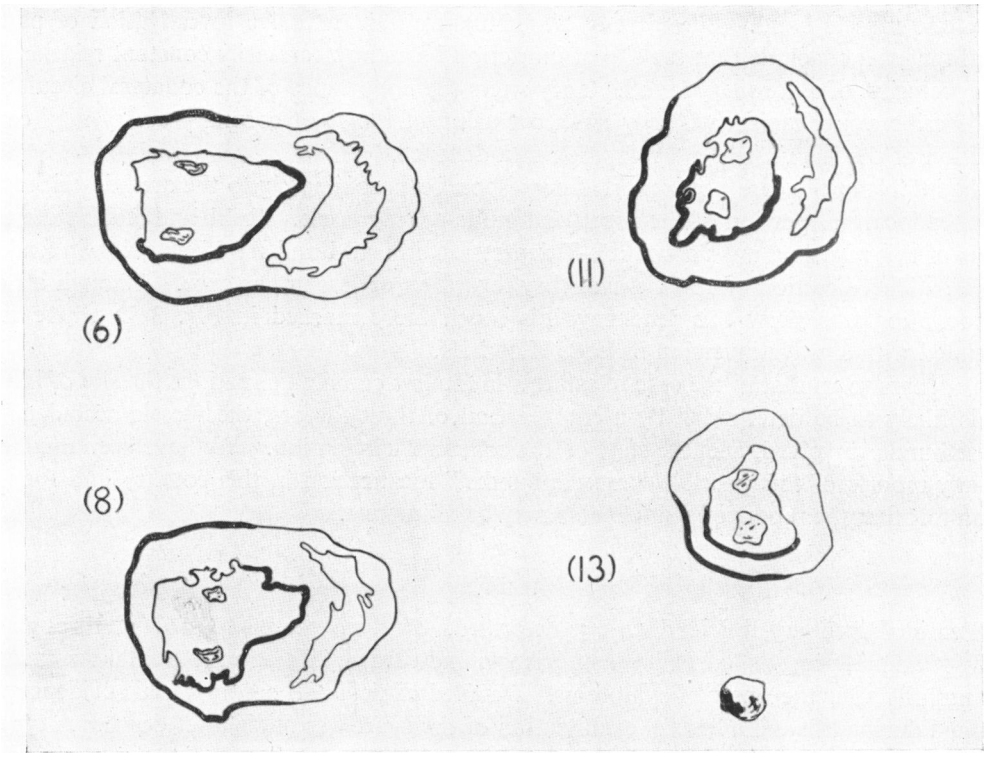

FIG. 5.—Photograph of a drawing to illustrate reconstruction of the superficial bulbo-spiral muscle. The numbers correspond approximately to the same level of transverse section as numbered in Fig. 4 [after T. E. Lowe (1939)].

DISCUSSION

A number of references to subendocardial infarction have been found. Parkinson and Bedford (1928) described occlusion of the circumflex branch giving rise to infarction of the lateral and posterior walls of the left ventricle usually involving the papillary muscles of the mitral valve. Whitten (1930) in an article on the relation of the distribution and structure of the coronary arteries to myocardial infarction mentioned one type of subendocardial infarct, which he suggested was due to occlusion of one or more of the large subendocardial branches that course often for a considerable distance in the subendocardial musculature, usually in relation to the columnæ carnæ or papillary muscles. Barnes and Ball (1932) investigated the incidence and situation of myocardial infarction in 1000 consecutive autopsies at the Mayo Clinic and found 49 cases, of which 28 were anterior apical, 24 posterior basal, 8 midventricular, and 3 diffusely beneath the endocardium; they state that in an occasional instance chronic infarction is diffuse, tends to extend in the form of subendocardial fibrosis and involves the entire left ventricle more or less completely. Lowe (1939) from a study of myocardial scars suggested a type of myocardial infarction that depends upon the interruption of the blood supply to a muscle bundle in the myocardium; this muscle bundle need not be supplied by a single main coronary vessel but may receive its blood supply from smaller branches of a number of vessels. In later articles, 1939 and 1941, he reported cases and attempted to correlate the clinical and cardiographic evidence with the post-mortem findings. Among the 5 new cases reported in 1941, Case 91 showed cardiographic changes consistent

with lesions of the superficial and deep bulbo-spiral muscles, and these findings were confirmed at autopsy. Robb and Robb (1939) have done experimental work on dogs and have also investigated a considerable number of human cases. They emphasize that the characteristic electrocardiogram appears, not in association with involvement of some one artery, not always in relation to the anterior or the posterior surface, but constantly in the presence of a lesion of the one muscle band. Blumgart *et al.* (1940) from an extensive investigation of pathological material conclude that the site of infarction bears no necessarily constant relationship to an occlusion of a coronary vessel. They stress the importance of the collateral circulation and describe a mechanism whereby fresh occlusion of the right coronary artery may cause infarction of the left ventricle, " infarction at a distance."

The infarct in the case now reported corresponds approximately to the area described for the subendocardial portion of the superficial bulbo-spiral muscle. Robb and Robb state that lesions of this muscle always cause RS–T depression in lead I and RS–T elevation in leads II and III. The direction of the T waves they regard as of less importance. Though all our electrocardiograms show this tendency, it is much more marked in Fig. 3 D where it is associated with T wave inversion in leads II and III.

It appears to us that in the case of infarcts in the less common situations the site of infarction is likely to be determined by a combination of three factors, the vessels occluded, the muscle bundle involved, and the state of the collateral circulation. We have been unable to find any reports of this possible " muscle bundle " localization in this country and for that reason feel that the report of a single case may be of interest.

Summary

A case of coronary arterial disease is described. It was observed to progress through the stages of angina pectoris, coronary insufficiency, and cardiac infarction to a fatal termination four months after the onset of symptoms. Detailed clinical and cardiographic evidence was obtained during life and a careful examination of the heart was made after death. A type of infarct is described that may correspond to a muscle grouping in the ventricle rather than to the distribution of a main coronary vessel.

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