Mitral valve disease with atrial fibrillation is undoubtedly the commonest cause of arterial embolism (Wood, 1956). In any large series of living patients with mitral stenosis the incidence of systemic embolism may be 9 to 14 per cent (Sellors et al., 1953; and Wood, 1954). In many patients embolism is either multiple or recurrent, and in 1951 Daley et al. and Jordan et al. reported 245 patients with rheumatic heart disease in whom there had been 489 episodes of arterial embolism: in none of these were the coronary arteries involved. Study of the reported cases shows that only 3 examples of coronary artery embolism associated with rheumatic heart disease have been reported ( Rolleston, 1896; Cheng et al., 1953; and Segall and Harris, 1954). A further case is, therefore, described.

Case Report

A woman, aged 41, was first admitted to the Radcliffe Infirmary in 1947 as an emergency with a severe attack of breathlessness and palpitation. She had had rheumatic fever when 6 and chorea when 9, and complained of increasing breathlessness for three years. It was found that she had atrial fibrillation and clear signs of mitral stenosis. She was put on to strophanthus and discharged well. In November, 1949, she was re-admitted in heart failure. This responded well to digoxin, mersalyl, and rest in bed. She was re-admitted in 1955 for consideration of operation, but she refused this.

She was finally admitted in 1958 with an arterial embolism of the right leg which had occurred about three hours previously. Forty-eight hours before she had had some left loin pain which radiated to the hypogastrum. On examination she looked extremely ill. She had mitral stenosis, atrial fibrillation, mild congestive heart failure, and albuminuria. The right leg was cold and discoloured and no pulses could be felt below the femoral. A right femoral embolectomy was performed, an embolus being removed from the femoral artery 2-5 inches (6 cm.) below the circumflex branch. Following the operation the colour of the leg improved, though the foot remained cold and pulseless. As no further improvement occurred during two weeks, an above-knee amputation of the right leg was performed. She made a good recovery from this operation and the stump was healing well when three weeks later, she complained of sudden severe substernal pain with radiation into the left arm. An electrocardiogram taken about ten minutes later showed changes compatible with anterior infarction in the standard leads and aVR : aVL, however, showed ventricular fibrillation (Fig. 1) and she died before any treatment to correct this could be given.

At necropsy, the heart showed severe mitral stenosis with massive thrombus adherent to the endocardium of the left atrium and auricle. The foramen ovale was closed. The aortic valve cusps were slightly adherent, but neither the aortic nor the mitral valve showed evidence of vegetations or bacterial endocarditis. A greyish-red thrombus, 0-3 cm. in main diameter, was found loose in the proximal part of the main left coronary artery and could be seen at the coronary orifice (Fig. 2). No evidence of myocardial infarction was found and the coronary arteries were not atheromatous. Scars of old infarcts were present in the spleen and in both kidneys. A recent infarct was found in the left kidney.

Histological examination of the tissues confirmed the macroscopic findings. The thrombus in the coronary artery was a recently formed antemortem thrombus, having a similar structure to the superficial parts of the left atrial thrombus. A section of the proximal part of the left coronary artery showed no evidence of adherent thrombus.
FIG. 1.—Electrocardiogram taken ten minutes after the onset of sub-sternal pain, showing changes compatible with anterior infarction in standard leads and ventricular fibrillation in lead aVL.

FIG. 2.—Photograph showing embolus in the mouth of left coronary artery.

Discussion

Occlusion of a coronary artery by an embolus is a dramatic event and, probably because of this, arouses considerable interest when it occurs. In the first half of the 19th century, Marshall Hall suggested that sudden interruption of the coronary circulation would rapidly cause death. Taking this as his text, Erichson ligated the coronary arteries in dogs and rabbits and proved this speculation. This work was repeated
by other workers and possibly as a result, considerable confusion arose concerning the incidence of coronary embolism and thrombosis. Rolleston (1896) quoted Cohnheim as saying that cases are on record where the sole cause of death was embolism of the coronary artery. He pointed out, however, that these must be very rare since no example is recorded in the transactions of the Pathological Society. Marie (1896) stated that the only certain case of coronary embolism was that described by Virchow (1856). To Virchow must go the credit of describing the first case, but Marie’s statement is too sweeping, for Rolleston was well aware of the occurrence of embolism associated with endocarditis. He quoted a case of myomalacia cordis due to embolism of the left coronary artery, which had been described by Hebb in the transactions of the Pathological Society; he also added that embolism of terminal twigs occurs in pyæmia, giving rise to pyæmic abscesses.

The first detailed review of the condition was given by Saphir (1933). He described 3 cases of his own and considered that only 11 of the reported cases were proven in that the source of the embolus was found at autopsy. The condition was next extensively reviewed by Hamman (1941), who thought the condition was more common than generally realized and a conservative estimate of the incidence may account for 1–2 per cent of all cases of occlusion. He quoted as a basis for this statement the incidence of 14 cases of embolism occurring in 1542 cases of coronary occlusion described in three separate reviews. Further, he was able to find 10 cases from the records of John Hopkins Hospital between 1931 and 1939. Hamman’s claim is not as unlikely as it first seems, though many subsequent authors stress the rarity of the condition and Walker (1952) says coronary embolism in bacterial endocarditis is unusual.

However, Brunson (1953), reviewing coronary embolism in bacterial endocarditis, claims that a total of 155 cases have been reported and that 125 of these were due to bacterial endocarditis. He described 9 cases of bacterial endocarditis that he had studied and as 7 of these had emboli, he believed that coronary embolism was relatively common, if it was looked for. This is supported by Cates and Christie (1951): in their review of sub-acute bacterial endocarditis, coronary embolism occurred 8 times among 164 episodes of major arterial embolism diagnosed during life. Out of the 89 autopsies held, infarcts were found in 10 cases and in 2 more the coronary artery was the site of an infected aneurysm or abscess.

Hamman in his review gave six possible sources for emboli, viz.: (1) bacterial vegetations, (2) mural thrombus in the aorta (arteriosclerotic or syphilitic lesions), (3) intracardiac mural thrombi, (4) thrombotic material in a coronary artery, (5) thrombi in pulmonary veins, and (6) thrombi in peripheral veins (paradoxical embolism). The first has been discussed and the last three sources are all rare. The remaining sources are of some interest, and they account for at least 20 reported cases, the mural aortic thrombi being slightly the commoner. Many of the patients were young, i.e. under 35 years. Survival time varies: mostly death was sudden as in the case described, but some patients survived for up to 24 hours. The site of embolism was nearly always the left main artery or its anterior descending branch, a point that Rolleston mentions.

The diagnosis of embolism must always be difficult to make and naturally in view of the commonness of thrombosis it will rarely be considered. In the patient under 35 years of age it is, however, worth bearing the diagnosis in mind for, if the patient can be got over the initial shock and ventricular fibrillation prevented, the possibility of embolectomy arises. It would seem to be the treatment of choice, but it is unfortunate from the surgical point of view that nature should decree that embolism almost always occurs in the more inaccessible artery.

**Summary**

A case of coronary artery embolism associated with atrial fibrillation, rheumatic heart disease, and arterial embolism is described.

Reported cases are briefly reviewed and the rarity of the condition, except when associated with sub-acute bacterial endocarditis, stressed. The possibility of embolectomy is suggested.

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CORONARY ARTERY EMBOLISM

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