Angiographic Evidence of Coronary Embolism and Resolution*

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SUMMARY

This report provides angiographic documentation of an embolus to the left coronary artery, followed by a second angiographic study which recorded disappearance of the embolus. The relationship between coronary embolism and ventricular dysfunction is discussed.

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Coronary artery embolism has been reported in cases of bacterial endocarditis, syphilitic heart disease, mitral valve disease, from prosthetic valves, intracardiac thrombus, Teflon patches, and paradoxically from the systemic veins in patients with right-to-left intracardiac shunts.[1-5] A diagnosis of embolic obstruction should be suspected if myocardial ischaemia occurs in a patient who has a disease which is known to be associated with emboli or in whom definite proof is obtained at postmortem examination.[2,6] Patients who survive the embolic episode may develop acute myocardial infarction, cardiac failure may increase in severity or a ventricular aneurysm may develop.[2]

There is also a group of patients in whom clinical episodes of myocardial ischaemia or systemic emboli have not been recorded but who present subsequently with abnormalities of ventricular function which are unrelated to, or in excess of, their underlying pathological condition. In such cases it is difficult to exclude antecedent coronary artery embolism.

We have not been able to find a published report which documents angiographically the presence of coronary artery embolism or its subsequent progress. We will report a patient in whom embolism to the left coronary artery was proved angiographically and in whom a second angiographic study showed that the embolus had disappeared. The importance of recording the natural history of the embolus and its relationship to abnormalities of ventricular function will be discussed.

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CASE REPORT

A 40-year-old Bantu female had had acute rheumatic fever in 1958. She was first seen in 1965 with grade II dyspnoea on exertion, orthopnoea and haemoptysis. She had atrial fibrillation, there was no evidence of heart failure and a right ventricular lift was palpable; auscultation revealed the classical findings of pure mitral stenosis with mobile valve cusps. The electrocardiogram (Fig. 1) showed atrial fibrillation, a mean frontal QRS axis of +70° and no changes of obvious ventricular hypertrophy. The chest X-ray (Fig. 2) showed a large heart with a cardio-thoracic ratio of 63%, an enlarged left atrium and pulmonary venous hypertension and congestion.

The patient was admitted to hospital and underwent transventricular mitral valvulotomy on 25 February 1965. The valve orifice was less than 1 cm² and a satisfactory valvulotomy was achieved using the Tubb's dilator. At surgery the left ventricle appeared normal. Three months later her symptoms had disappeared and the only residual auscultatory features were consistent with mild mitral stenosis.

In 1967 the cardiac failure recurred. Left ventricular enlargement was present on palpation with the signs of moderate mitral stenosis. The chest X-ray showed that the heart had become larger, the cardio-thoracic ratio was 70% and the left ventricle was prominent. Heart failure was treated with digitalis and diuretics.

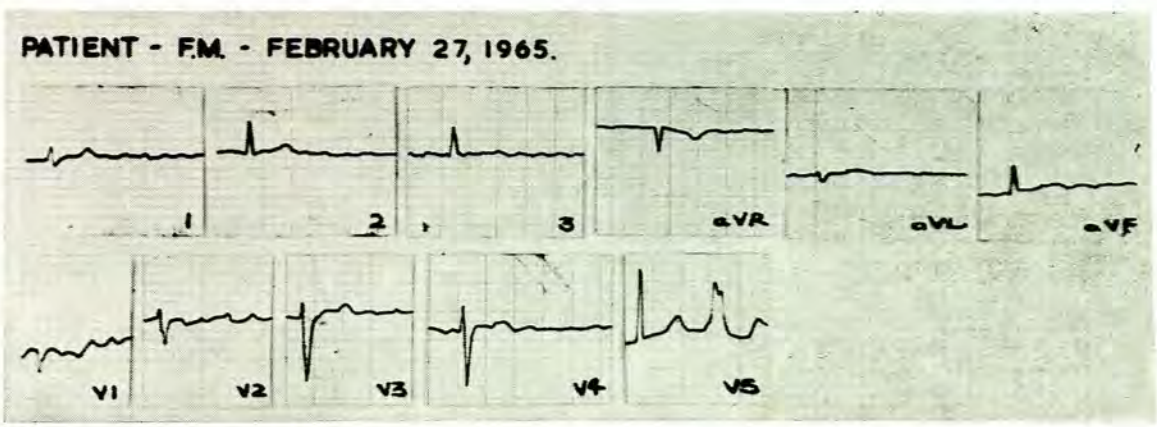

Fig. 1. Electrocardiography to show atrial fibrillation initially.

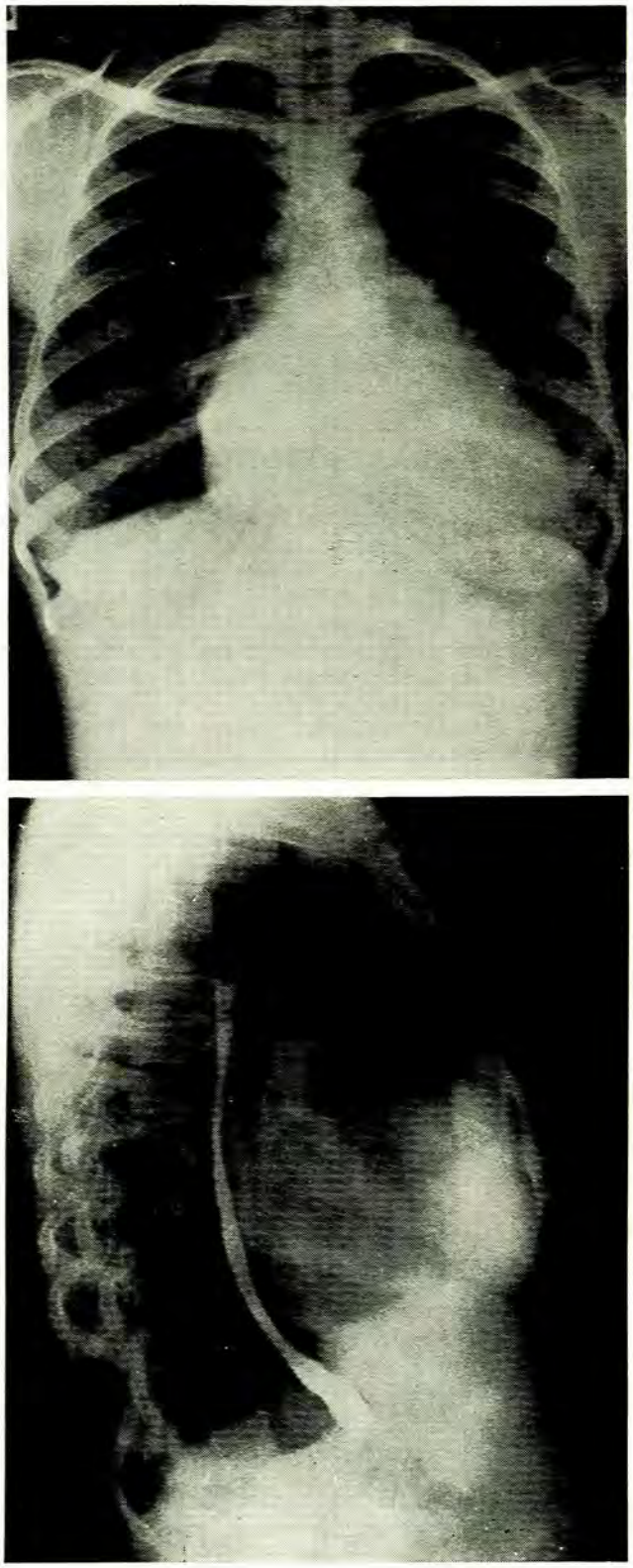

Fig. 2. Chest X-rays (postero-anterior (top) and lateral (bottom) before first valvulotomy in 1965. Slight left atrial and left ventricular enlargement and mild pulmonary venous hypertension.

In May 1969 she was admitted to hospital with a further exacerbation of heart failure. The left ventricle had increased in size and the apex beat was diffuse and heaving in character and could be palpated in the anterior axillary line.

A soft pansystolic murmur was heard in the apical area and radiated to the axilla. The electrocardiogram (Fig. 3) now showed voltage changes suggestive of left ventricular hypertrophy. Chest X-ray (Fig. 4) showed that the heart had increased in size, the cardio-thoracic ratio was 72% and the left ventricle was prominent.

Cardiac catheterization, undertaken on 5 June 1969 to identify the cause of the residual disability, showed mild pulmonary hypertension, mild mitral stenosis and a low normal cardiac output (Table I). Selective left ventriculography showed only very mild mitral insufficiency. However, the left ventricle was enlarged and a large area of dyskinesis, involving its apical and lateral walls, was present. The aortic root angiogram outlined the main right and left coronary arteries and these appeared to be normal. The mitral incompetence was very mild and insufficient to account for the degree of left ventricular enlargement. Moreover, the area of dyskinesis in the left ventricle was localized and for this reason a second study was performed on 16 June 1969 to determine its cause. Further left ventriculograms confirmed the abnormal pattern of left ventricular contraction. No complications were noted during angiography. Selective right and left coronary arteriograms were then made using the Judkin's technique.[7] The right coronary artery was normal. Selective left coronary angiography showed an obstruction at bifurcation of the main artery where it divided into its anterior descending and circumflex branches (Fig. 5). Contrast medium flowed past the obstruction and outlined normal distal coronary arteries. After the fifth injection of contrast medium the patient developed an episode of ventricular fibrillation. Sinus rhythm was restored by electrical reversion but the heart remained irritable and several further episodes of ventricular fibrillation occurred over the next 24 hours. She was treated with anticoagulants (Heparin 40 000 IU daily for 7 days and then Warfarin for 4 weeks), lignocaine and procainamide. She improved after 24 hours and made a good recovery; there were no neurological sequelae or other signs of systemic emboli. Electrocardiograms, recorded immediately after recovery from the episode of ventricular fibrillation, showed that an extensive anterolateral infarct had developed (Fig. 6). The chest X-ray had not altered (Fig. 7).

The coronary arteriogram was repeated after 4 weeks to see whether direct coronary arterial surgery might be feasible. The left coronary artery was now completely normal, the previous obstruction had disappeared and flow to the distal branches was normal (Fig. 8).

TABLE I. CARDIAC CATHETERIZATION DATA

Site	Pressures (mmHg) SD	Mean
Right atrium		9
Right ventricle	33/0 - 10	
Pulmonary artery	33/12	19
Pulmonary capillary wedge		14
Aorta	115/62	85
Left ventricle	115/0 - 10	
Cardiac index	2·9 litres/min/m^2	

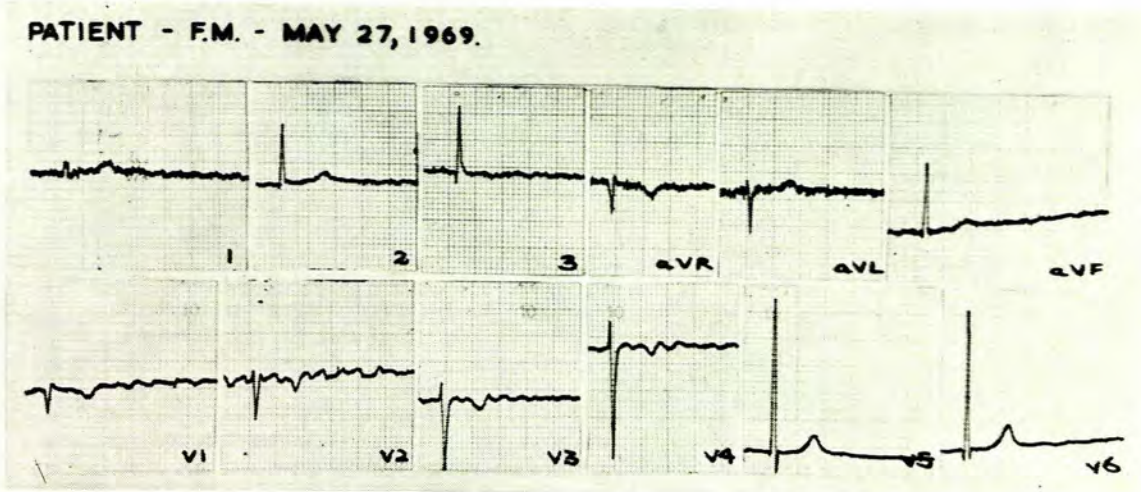

Fig. 3. Electrocardiography to show the development of voltage changes of left ventricular hypertrophy.

The patient was discharged some weeks later with mild residual disability but has since been readmitted to hospital with cardiac failure on 3 further occasions. The ECG pattern of anterolateral infarction is still present. She lives in a remote area far from the hospital so that anticoagulant therapy has not been continued.

DISCUSSION

It is often difficult to prove conclusively that an episode of myocardial ischaemia is due to coronary embolism in those patients who survive and the diagnosis must be inferential. Its incidence in mitral valve disease is uncertain.

This patient sustained a coronary embolus which subsequently disappeared and resolved. The angiographic appearances are unequivocal: the left coronary artery had been normal at earlier aortography, the right and left coronary arteries were otherwise perfectly normal, the

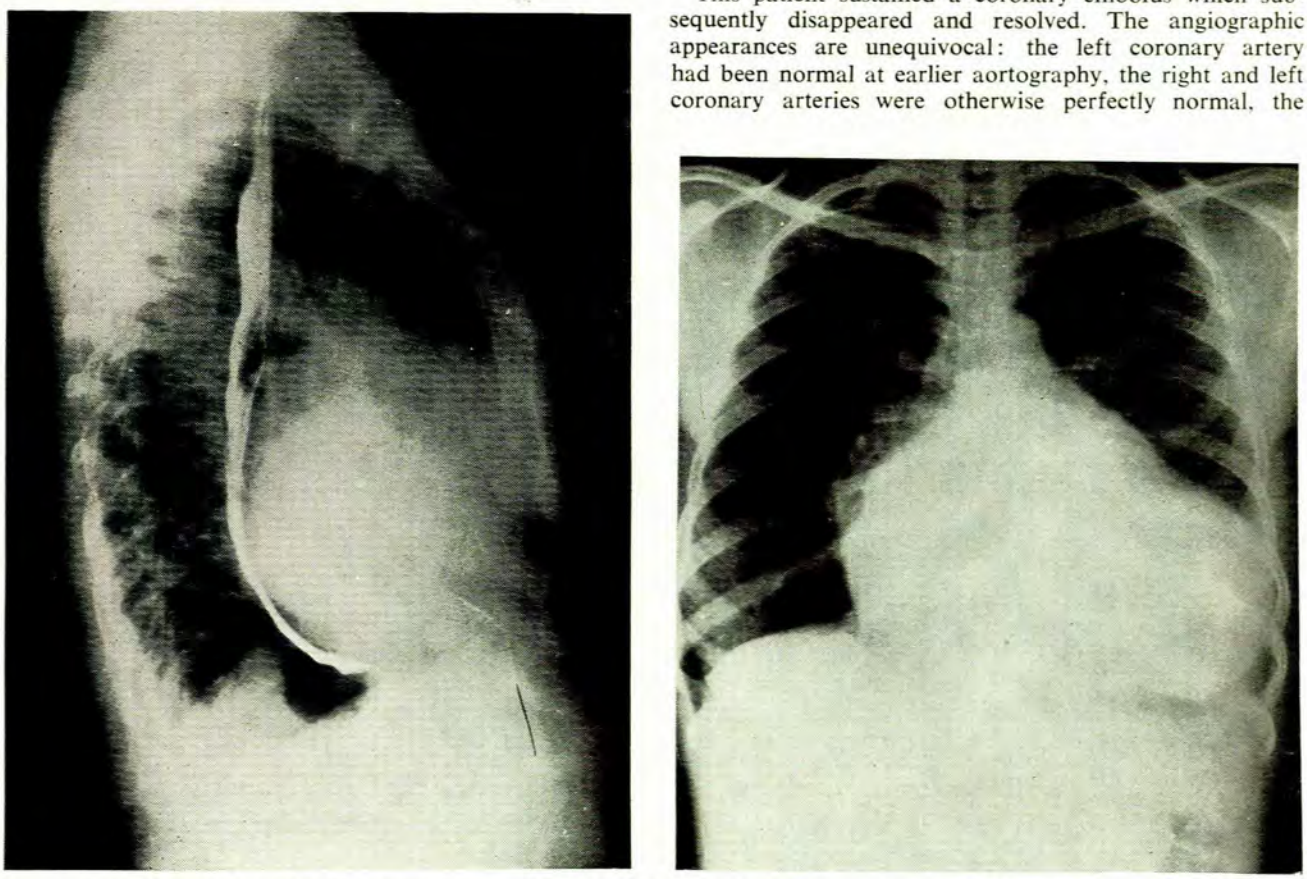

Fig. 4. Chest X-rays (lateral (left) and postero-anterior (right)) before the documented episode of coronary embolism to show unusual left ventricular and left atrial enlargement.

Fig. 5. Selective left coronary arteriography recorded in the right anterior oblique projection. Filling defect shows a saddle-shaped embolus at the bifurcation of the main stem of the left coronary artery into its anterior descending and circumflex branches. The distal vessels are normal.

obstruction was saddle-shaped and located at the bifurcation of the main stem of the left coronary artery and the embolus resolved completely within 4 weeks, leaving an apparently normal artery.

The source of embolization is uncertain. It may have come from the catheter used for coronary arteriography although this had been carefully flushed with heparinized saline while the tip was in the arch of the aorta. Other likely sources were thrombus covering the endocardial surface of the dyskinetic area of the left ventricle or the fibrillating atrium. It is feasible that mural thrombus may have been dislodged from the endocardium of the left ventricle at the time of ventriculography.

Previous subclinical episodes of coronary artery embolization may perhaps have accounted for some of the conflicting clinical features. The symptoms were more severe than the degree of mitral valve disease present, the chest X-ray showed enlargement of the left ventricle which was out of keeping with the degree of mitral incompetence demonstrated by cine-angiography and the latter showed localized abnormalities of contraction of the enlarged left ventricular chamber. It is probable that previously unrecognized emboli had produced small areas of myocardial necrosis and fibrosis and were the cause of the abnormal function and enlargement of the ventricle.

There are few reports which describe the natural history of systemic emboli and most studies have suggested that emboli lyse naturally: the rate of absorption of emboli depends on a number of factors—the size of the embolus, its degree of organization, blood flow around the obstruction, the perfusing pressure and fibrinolytic mechanisms.[8-12]

The present study indicates that coronary artery embolism can occur and can resolve leaving behind normal coronary vessels with residual myocardial dysfunction and infarction.

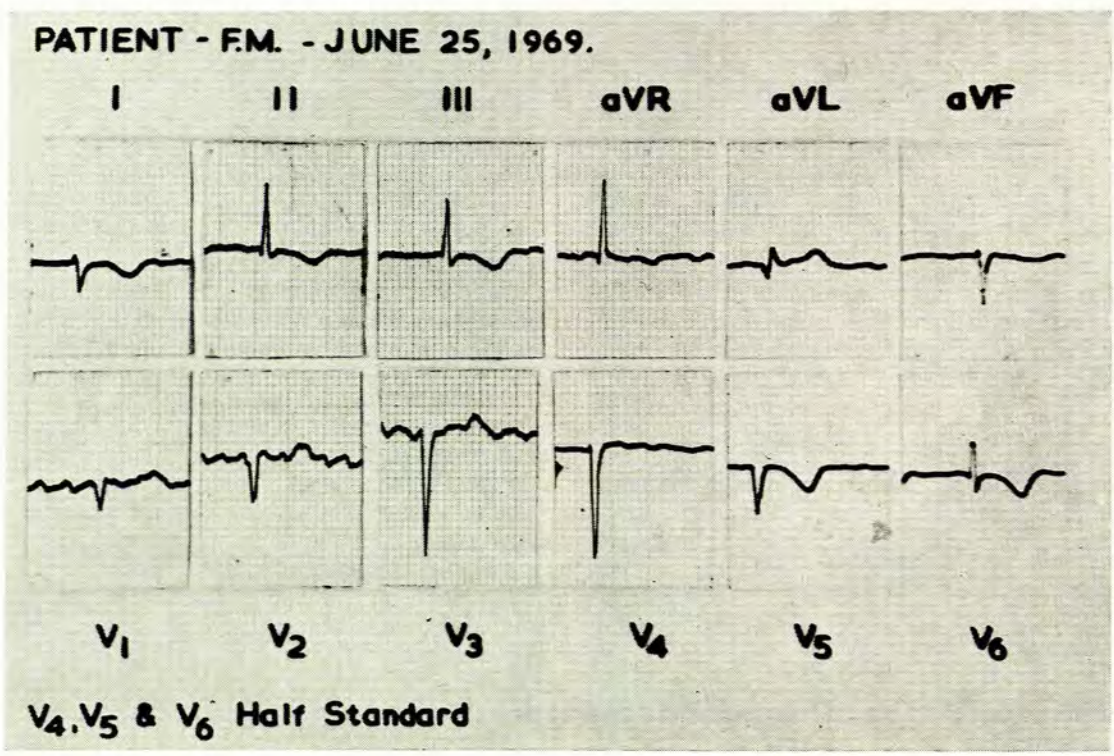

Fig. 6. Electrocardiography to show anterolateral myocardial infarction with diaphragmatic extension.

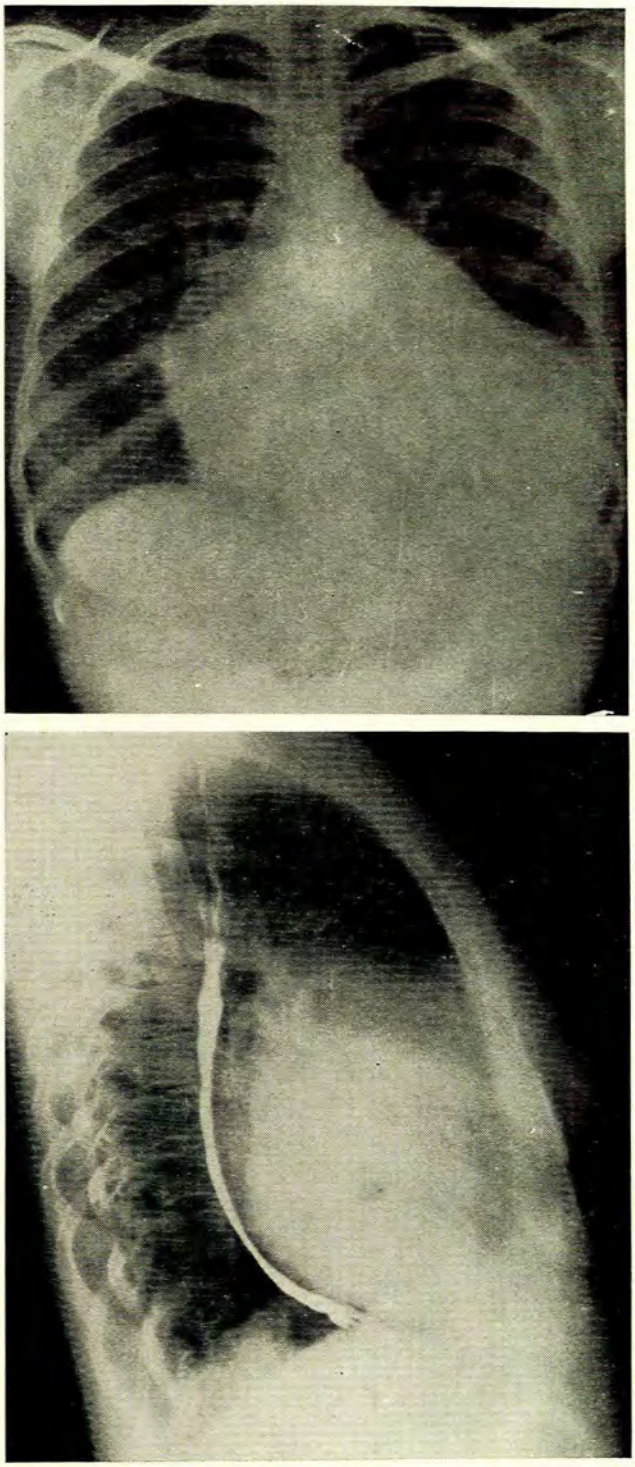

Fig. 7. Chest X-rays (postero-anterior (top) and lateral (bottom)) after the episode of myocardial infarction.

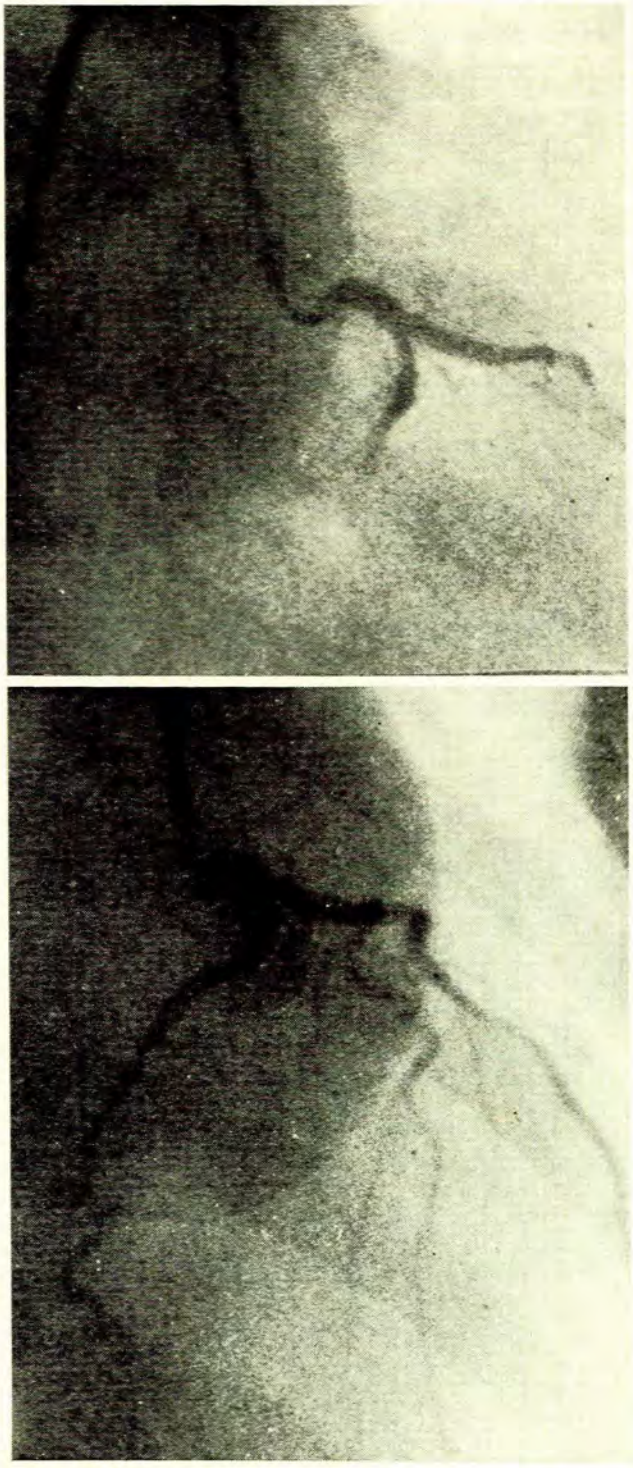

Fig. 8. Selective left coronary arteriography recorded in the right anterior oblique projection (4 weeks after Fig. 5 was recorded) showing the disappearance of the embolus and a normal arterial tree.

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