Children are also apt to ingest large doses of these drugs which may have been prescribed for their parents or for other adults living in the same household. The cardiovascular effects of toxic doses have not been as widely known as the effects on the nervous system. Conduction defects such as those seen in the present patient make it likely that complete heart block will also occasionally be seen; therefore, constant electrocardiographic monitoring and ready availability of transvenous pacing equipment are advisable in caring for such patients.

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Myocardial Infarction and Ventricular Aneurysm in a Patient with Normal Coronary Arteries*

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A 64-year-old man is reported with a clinical history and electrocardiographic evidence of myocardial infarction. He was found to have a ventricular aneurysm and normal coronary arteries at cardiac catheterization.

Three findings usually considered pathognomonic of atherosclerotic coronary artery disease are angina pectoris, myocardial infarction, and ventricular aneurysm. Angina pectoris* and myocardial infarction* were described recently in patients with normal coronary arteries as visualized by selective cineangiography. The present case report recounts in detail the occurrence of a ventricular aneurysm in an elderly man with a clinical history consistent with atherosclerotic coronary artery disease supported by electrocardiographic evidence of myocardial infarction in which normal coronary arteries were demonstrated by coronary arteriography.

CASE REPORT
A 64-year-old Greek man was admitted to this hospital for the first time on May 1, 1972 complaining of chest pain. In 1961, while living in Venezuela, he first developed chest pain for which he was hospitalized. He was told he had suffered a "heart attack." He was asymptomatic until 1966 when exertional chest pain prompted hospitalization and another diagnosis of heart attack. In 1969, while working, he developed severe chest pain and collapsed, and once more he was hospitalized with a diagnosis of myocardial infarction. From 1969 to 1971 the patient was admitted to Greek hospitals at least seven times for recurrent chest pain and shortness of breath. He continued to have chest pain three to four times per week and more severe attacks every two weeks, with some relief from nitroglycerin therapy. In July, 1971, with no specific alterations in his therapeutic regimen or lifestyle, the symptoms improved dramatically. Mild chest pain occurred only rarely until this admission, which occurred two days...

![Electrocardiogram](https://example.com/figure1.png)

**Figure 1.** Electrocardiogram taken during present admission is typical of those of patient since 1969. It reveals Q waves in leads V3-V4, with coving of elevated ST segments indicative of anterior wall infarction.
Physical examination revealed a moderately obese white man in no distress. Blood pressure was 100/80 mm Hg. The pulse rate was 80/min and regular; respiration rate was 16/min. Funduscopic examination revealed grade 1 arteriosclerotic changes. There was no venous distention in the neck. The lungs revealed minimal rales at the left base. Cardiac examination disclosed a diffuse point of maximum intensity (PMI) just outside the midclavicular line in the fifth intercostal space. The heart sounds were somewhat diminished; the aortic and pulmonic components of the second heart sound were of equal intensity at the pulmonic area. A fourth heart sound was heard at the apex. There was a 2/6 systolic ejection-type murmur heard in the fourth intercostal space at the left sternal border. The abdominal examination showed no abnormalities. Peripheral pulses were of good quality and equal throughout, and there was no peripheral edema.

Laboratory data included normal values for serum electrolytes, complete blood cell count, platelets, serum enzymes, cholesterol, fasting and two-hour postprandial blood sugar levels. The blood urea nitrogen value was initially elevated to 60 mg percent but subsequently fell to 19 mg percent. Creatinine clearance was 82 ml/min, and the triglyceride level was 164 mg percent.

The chest x-ray film showed a normal cardiac silhouette. The electrocardiogram (Fig 1) disclosed changes in the precordial leads consistent with an old anterior myocardial infarction. The patient brought his serial electrocardiograms dating from 1969, all of which were unchanged from the present one.

Two weeks after admission, cardiac catheterization was performed. The pressures in the right side of the heart were at the upper limits of normal. The left ventricular end-diastolic pressure was 17 mm Hg, which included an "A" wave of 8 mm Hg. Following angiography, left ventricular end-diastolic pressure increased to 25 mm Hg. The cardiac index by dye-dilution technique was calculated to be 2.6 liters/min/M². The stroke index of 43 ml/beat/M² was within normal limits. The left ventricular angiogram demonstrated a minimally enlarged left ventricle, with a discrete anterior wall ventricular aneurysm (Fig 2). The end-diastolic volume index was 118 ml/M², a value slightly above the upper limits of normal for this laboratory (73-109 ml/M²), with an ejection fraction of 36 percent. Selective right and left coronary arteriography was performed by the Sones technique in both left and right anterior oblique views. The coronary arteries were normal, showing no evidence of vascular cut-off, pruning or intimal disruption (Fig 3).

FIGURE 2. End-systolic left ventricular angiogram in right anterior oblique view revealing discrete midanterior wall aneurysm.

FIGURE 3. (1) Anterior descending; (2) circumflex; (3) diagonal coronary arteriogram in right anterior oblique view (a) revealing normal anterior descending artery without luminal irregularities. Septal perforating branches are present. Similar anatomic observations can be confirmed in posterior-anterior view (b, lower).

DISCUSSION

The most common manifestations of coronary artery disease are angina pectoris and myocardial infarction; when either is present, a diagnosis of coronary artery disease is presumed. In recent years an increased frequency of evaluation by coronary arteriography has revealed a considerable percentage of patients with these features but who have normal coronary arteriograms.

One complication of myocardial infarction, namely ventricular aneurysm, has generally been associated with severe coronary artery disease and electrocardiographic evidence of a transmural infarction. This communication relates our experience with a patient who gave a history of myocardial infarction and showed evidence of a transmural anterior infarction on the electrocardiogram as well as angiographic evidence of an anterior wall aneurysm. However, no evidence of coronary artery disease was found on the coronary arteriogram.

Several recent case reports have described patients with unquestionable clinical and electrocardiographic evidence of myocardial infarction in whom subsequent coronary arteriography has revealed normal coronary vessels. Varying degrees of regional hypokinesis were demonstrated in some of these patients but only rarely has a ventricular aneurysm been reported. Bruschke et al described five patients with acute myocardial infarc-
tion with subsequent normal coronary arteriograms. One of these patients demonstrated paradoxical motion of the anterior wall during left ventriculography. In one other patient recanalization was documented by serial coronary angiograms. A coronary artery embolus with subsequent thrombolysis offers one possible explanation of the finding of a normal anterior descending artery in our patient. Subacute bacterial endocarditis, marantic endocarditis, cardiac catheterization, or prosthetic valve insertion can result in coronary artery embolus, but there is no clinical or historic evidence to support any of these diagnoses in this patient.

One possibility that must be considered is that the cineangiograms failed to detect coronary arterial lesions which were sufficient to cause localized myocardial death and aneurysm formation. The angiograms of this patient demonstrated multiple branches of the anterior descending coronary artery in three views without a starburst pattern, which left no area underperfused. Ross and Friesinger stated that luminal narrowings of 20 percent or more may be overlooked, particularly if the lesion is circumferential and discrete, and that twig lesions may not be detected. However, a 20 percent narrowing or distal occlusion, even if overlooked, is unlikely to result in aneurysm formation.

This report should serve as a caution that most if not all of the diagnostic characteristics of coronary artery disease may be seen in the presence of normal coronary arteriograms. The etiology of these disorders remains to be clarified. Occurrence of a regional myocardial infarction or ventricular aneurysm suggests prolonged interruption of coronary flow to that region. In this regard, prolonged vasospasm or an embolus with subsequent lysis are the most likely causes. Unfortunately, objective evidence of such occurrences is difficult to obtain.

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