cm extension. An occlusive thrombus, 1.5 cm long, was found in the proximal segment of the myocardial bridging (Fig. 2), associated with a massive recent infarction in the corresponding myocardium. A typical atherosclerotic plaque, composed of a lipid core with cholesterol crystals surrounded by a fibrous cap, could be seen underlying the thrombus (Fig. 3, *upper panel*). The mural segment of the myocardial bridging was narrowed, and showed an atherosclerotic lesion mainly composed of smooth muscle cells and fibrous tissue (Fig. 3, *lower panel*). Other organs showed no abnormalities, except for pulmonary edema. Familial history of hypercholesterolemia was not established.

Autopsy studies have provided conflicting data concerning the role of myocardial bridges in producing atherosclerotic coronary disease and myocardial ischemia.[2, 8, 9] A significantly greater degree of atherosclerosis has been found in the segments immediately proximal to the bridges compared with the totally epicardial coronary arteries.[9] On the other hand, one theory of atherogenesis is that the atherosclerotic plaque develops as a response to injury of the vascular endothelium, the endothelial injury being the primary event.[10] The hemodynamic disturbance caused by the narrowing of the coronary artery in myocardial bridging may have important pathogenetic implications in the development of atherosclerosis in the proximal segment of the vessel. We observed a greater severity of atherosclerosis in the proximal segment of the myocardial bridging in comparison with other affected areas. The presence of an atherosclerotic plaque at this location, as well as the local flow disturbances, could be directly related to the development of occlusive thrombosis and consequent ischemia and myocardial infarction.

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Critical left main stenosis presenting as diffuse ST segment depression

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There is no standard treatment strategy for the clinical syndrome of unstable angina, which in part reflects the heterogeneity of patients with variable symptoms and electrocardiographic changes. Both stabilization with medical therapy as well as early catheterization and coronary revascularization have been advocated.[1-3] For the most ominous subgroup of patients with severe left main trunk (LMT) stenosis, no clinical pattern of recognition has emerged from the literature. We report our findings in three patients who had diffuse sustained ST depression and evidence of acute left ventricular dysfunction despite normal heart size, who were found to have critical LMT disease. All three patients were given an aspirin suppository in the emergency room, and all three received intravenous heparin. No prior ECGs were available. Patient data are shown in Table I and Figs. 1 to 3.

Case 1. A 78-year-old woman with hypertension awoke with severe dyspnea. Emergency intubation by paramedics was required for acute pulmonary edema. The ECG showed diffuse 1 to 6 mm ST depression (leads I to III, aVF, and V_3-V_6), as well as increased voltage compatible with left ventricular hypertrophy. Intensive therapy with nitrates, morphine, and diuretics was given over a 2-hour period. Subsequently cardiogenic shock developed with a sustained systolic blood pressure of 80 mm Hg despite administration of multiple pressor agents and placement of a balloon pump. Urgent catheterization demonstrated severe diffuse three-vessel disease in addition to eccentric 95% LMT diameter stenosis. Urokinase, 250,000 units, was infused into the LMT preoperatively with no effect. Coronary bypass surgery was performed successfully 5 hours after the onset of symptoms. However, the patient died of refractory heart failure on postoperative day 6. Postoperative ECG findings included diffuse loss of R waves and diffuse 1 to 1.5 mm ST depression. Autopsy findings revealed concentric left ventricular hypertrophy and up to 50% subendocardial left ventricular infarction.

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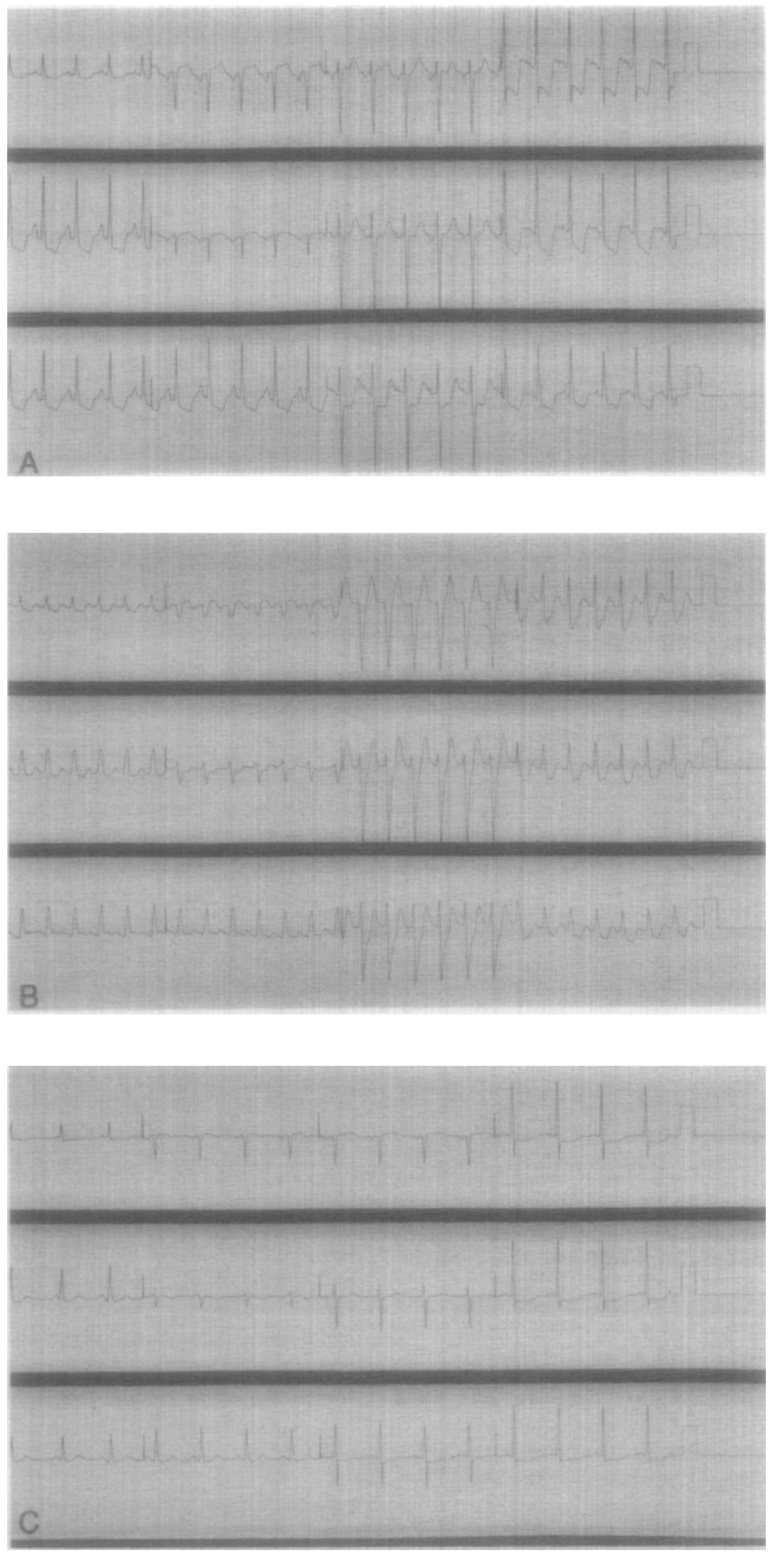

Fig. 1. Preoperative ECGs from cases 1 **(A)**, 2 **(B)**, and 3 **(C)** (see text for analysis).

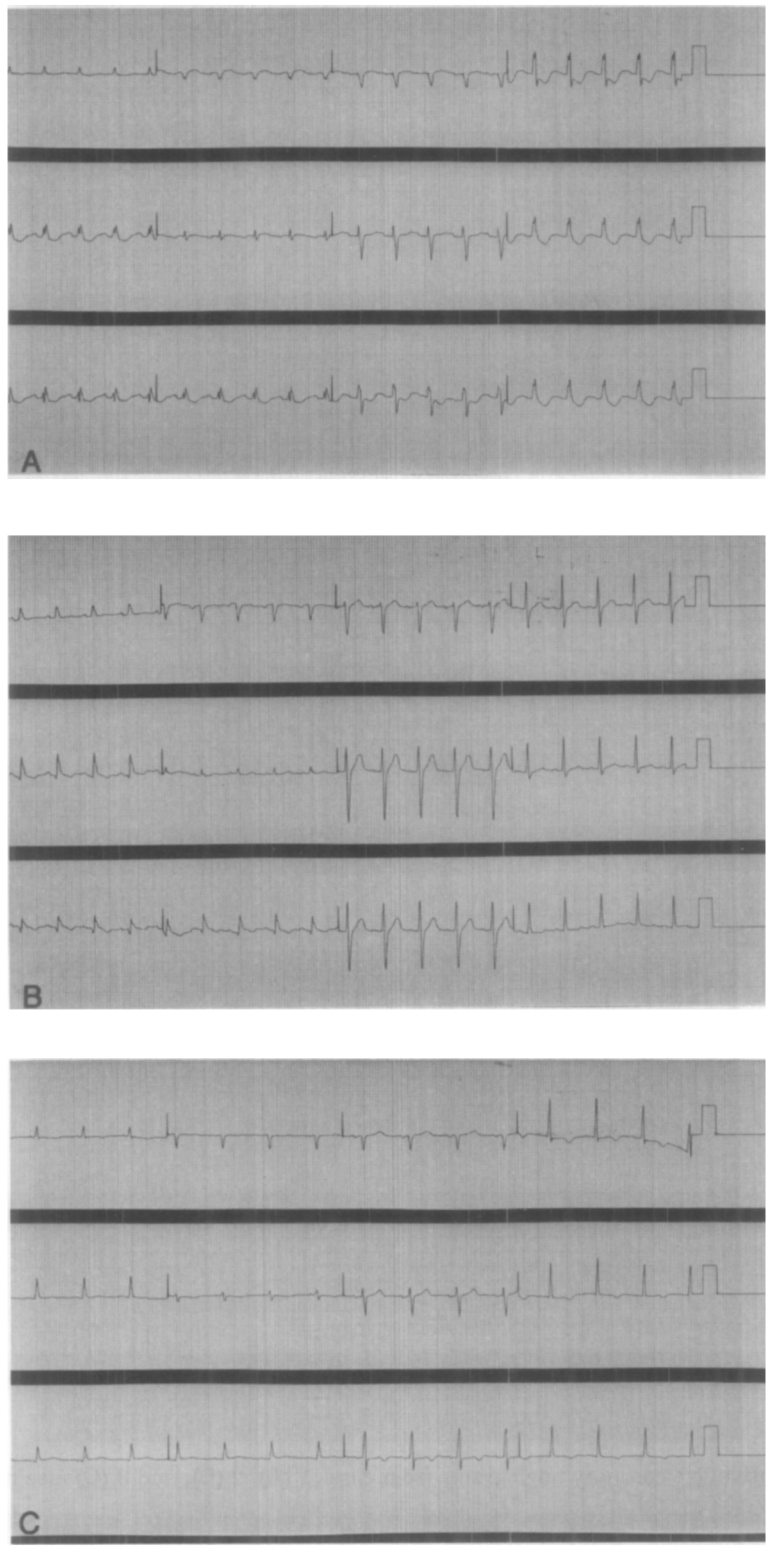

Fig. 2. Postoperative ECGs from cases 1 **(A),** 2 **(B),** and 3 **(C)** (see text for analysis).

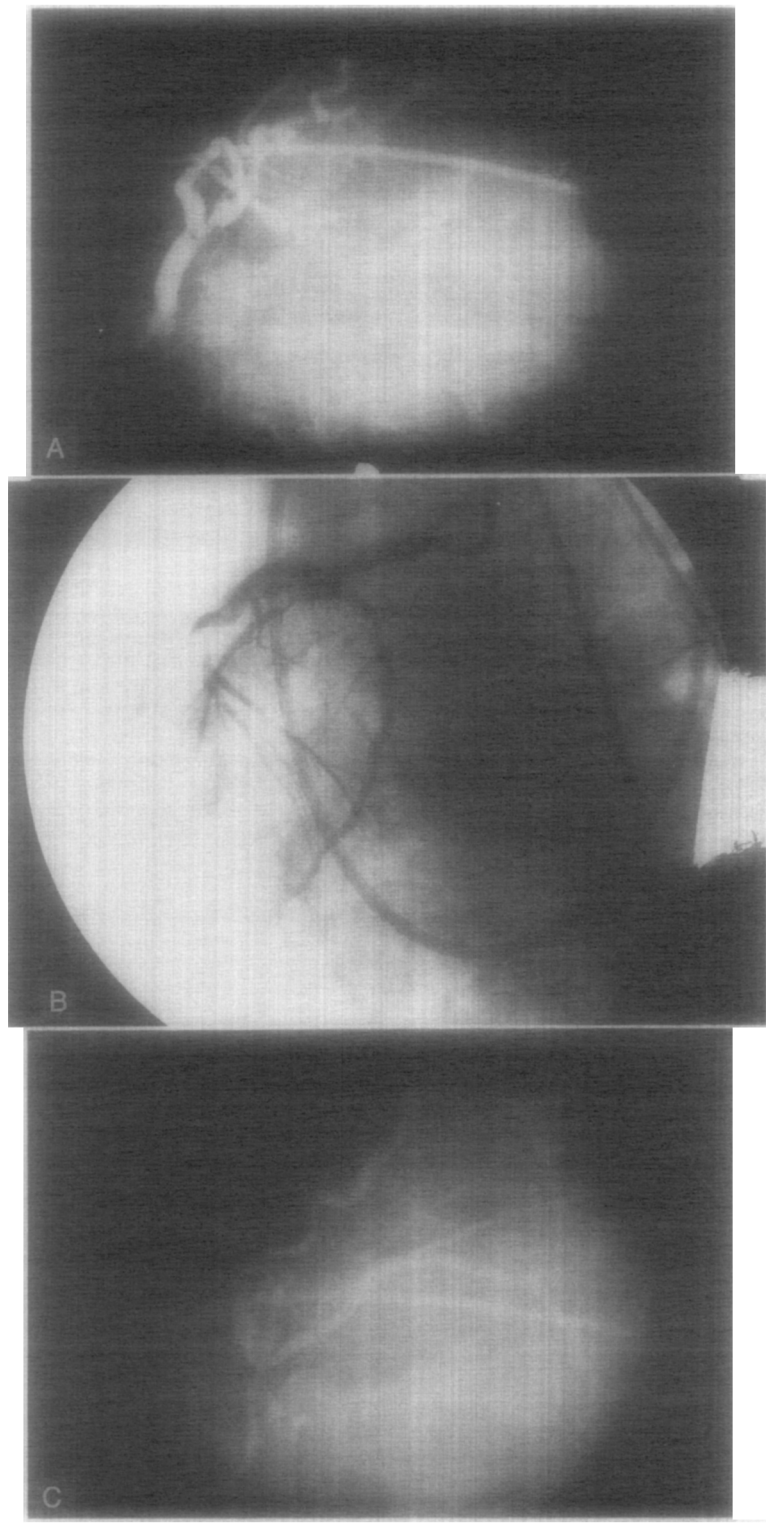

Fig. 3. Preoperative left coronary angiograms from cases 1 **(A)**, 2 **(B)**, and 3 **(C)** (see text for interpretation).

Case 2. A 61-year-old man suddenly collapsed in his front yard. He received cardiopulmonary resuscitation within 5 minutes and was defibrillated within 10 minutes. The patient had stable vital signs but was comatose and on a ventilator. An ECG showed an old inferior infarction and 1 to 1.5 mm anterolateral ST depression. An admission chest x-ray was normal. Two hours later further cardiopulmonary resuscitation was required for recurrent epi-

Table I. Baseline clinical and angiographic data with critical left main stenosis

Case	*Age (yr)*	*Clinical presentation*	*PWP*	*CI*	*LVEF*	*Peak CPK (U/L)*	*Max MB (ng/ml)*
1	78	Acute pulmonary edema	30	2.2	0.35	9,556	304
2	61	Recurrent cardiac arrest	11	2.5	0.30	5,058	134
3	60	Acute pulmonary edema	13	2.1	0.28	1,206	51

PWP, Pulmonary wedge pressure; *CI*, cardiac index; *LVEF*, left ventricular ejection fraction; *CPK*, creatine phosphokinase; *Max MB*, maximum MB isoenzyme fraction.

sodes of rapid sustained ventricular tachycardia. Hemodynamics stabilized after administration of bretylium, additional lidocaine, and brief use of pressor agents. However, serial ECGs demonstrated severe sinus tachycardia with intraventricular conduction delay and diffuse 1 to 5 mm ST depression (leads I, II, aVL, and V_3-V_6). Angiography showed 50% midcircumflex and 100% proximal right coronary artery stenosis, in addition to ostial 90% LMT stenosis. After placement of a balloon pump, the heart rate decreased from 160 to 125 beats/min. Bypass surgery was performed 8 hours after admission, and the patient recovered with no neurologic sequelae. A postoperative ECG showed nonspecific T wave changes and an old inferior infarction. Echocardiography performed 1 month after surgery showed normal left ventricular size and overall systolic function.

Case 3. A 60-year-old man awoke with chest pain and severe dyspnea. He required intubation and sedation in the emergency room for acute pulmonary edema, which was unresponsive to nitrates, diuretics, and morphine. Apsac was given by the emergency room physician for possible acute anterior infarction, based on an artifact-filled ECG reflecting patient movement. A repeat ECG showed 1 to 2 mm anterolateral (V_3-V_6) and 0.5 mm inferior ST depression, with 1 mm ST elevation in lead V_1. Blood pressure remained stable except for transient hypotension during catheterization, for which a balloon pump was placed. Angiography demonstrated 75% stenosis of the lateral dominant circumflex artery, in addition to diffuse 90% LMT stenosis. Subsequently, prolonged hypotension occurred as a result of refractory pyloric ulcer bleeding, which stabilized after administration of pressors, blood products, intravenous fluids, and endoscopic cauterization. At 18 hours after the onset of symptoms, bypass surgery was performed followed by complete recovery. Echocardiography performed 3 days after surgery demonstrated normal left ventricular size with a left ventricular ejection fraction of 0.45. The postoperative ECG showed diffuse T wave changes.

In patients first seen with symptoms of acute unstable angina and ST segment depression, it is uncertain whether initial medical management will adequately relieve coronary ischemia.[1-3] Previous studies have shown that LMT or severe three-vessel disease is significantly more common in patients with ST depression at baseline or during anginal symptoms.[3,4] Even so, initial medical stabilization followed by in-hospital catheterization has been recommended unless refractory symptoms develop, since urgent bypass surgery has not been shown to improve outcome for most patients with unstable angina.[1-2,5] Regarding thrombolytic therapy, no clear benefit has been demonstrated in patients with unstable angina.[6] However, an important subgroup that may benefit from early revascularization is severe LMT disease presenting with acute unstable angina, although no relatively specific clinical markers have been reported in the literature.[1,2,5]

In this report three patients with severe cardiac symptoms had diffuse ST depression. All had normal heart size despite evidence of severe left ventricular dysfunction. Of the two patients who had acute pulmonary edema, one initially stabilized with medical therapy and a balloon pump. The third patient, who had recurrent ventricular tachycardia after cardiac arrest, had rapid sinus tachycardia without clinical heart failure. Angiography demonstrated severe LMT stenosis in all three patients with either dominant circumflex or severe dominant right coronary artery disease. Bypass surgery was delayed only in the one patient who received thrombolytic therapy and had severe preoperative gastrointestinal bleeding. All patients had non–Q wave myocardial infarctions, and the one nonsurvivor had extensive subendocardial infarction at autopsy. The latter patient may have been at increased risk because of additional concentric left ventricular hypertrophy and severe three-vessel coronary disease. In the two surviving patients a large improvement in left ventricular function was documented. Thus in the setting of sustained diffuse ST depression with evidence of acutely impaired left ventricular function, urgent catheterization should be performed to identify possible severe LMT disease.

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