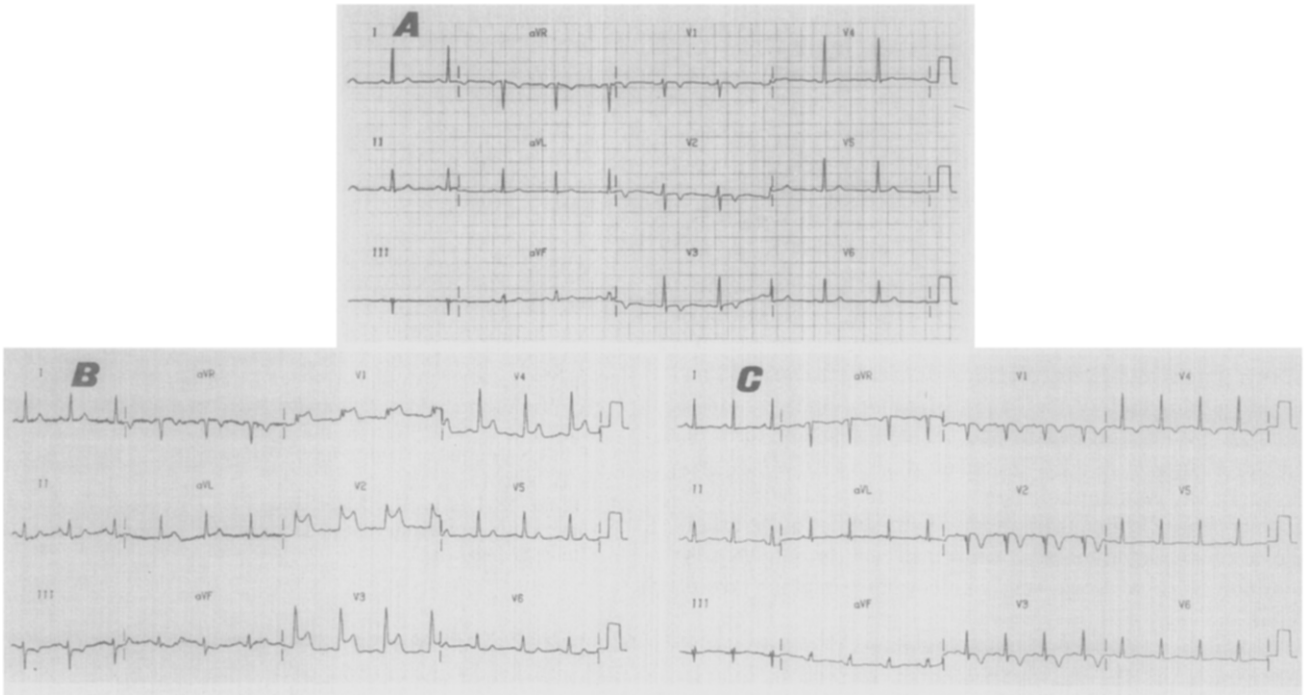

Fig. 1. A, Twelve-lead ECG of the patient at admission, when she was pain free. **B,** Twelve-lead ECG after a half hour of chest pain which had been unresponsive to medication. **C,** Twelve-lead ECG 5 days after admission showing a new Q wave in leads V_1 and V_2 and inverted T waves in leads V_4 and V_5 compared to the ECG taken at admission.

weeks previously, when she developed angina with light effort and angina at rest and was hospitalized. Anginal attacks continued to occur several times a day despite propranolol, 60 mg three times a day; isosorbide dinitrate, 20 mg three times a day; and nifedipine, 10 mg every 4 hours. Cardiac enzymes had been persistently negative, although the T waves in ECG leads V_1 through V_3 were inverted at rest. There were further typical ischemic ST-T changes during attacks of chest pain. On admission to the Thoraxcenter, her blood pressure was 145/95 mm Hg, pulse was 72 bpm and regular, and the cardiopulmonary examination was within normal limits. The ECG taken at the time of admission is shown in Fig. 1, *A*. Shortly thereafter the patient developed her most severe attack of chest pain, with nausea and perspiration and ST elevation on the ECG (Fig. 1, *B*). She was unresponsive to additional nitrates, sublingual nifedipine (10 mg), and intravenous morphine. Forty minutes after onset of pain the ECG slowly reverted to baseline and the pain disappeared, only to recur within 5 minutes with similar ST elevations in leads V_1 through V_3. Since spasm of a coronary artery was suspected, the patient was brought to the catheterization laboratory. She was not randomized in our study of intracoronary streptokinase in acute myocardial infarction, because the diagnosis of infarction could not be made at that early stage—10 minutes after recurrence of chest pain and ECG changes.

Initial injection into the left coronary artery showed total occlusion of the proximal left anterior descending (LAD) coronary artery (Fig. 2, *A*). The right coronary artery had no significant disease. A biplane left ventriculogram showed good ventricular function with a small area of anterior hypokinesis. The LAD remained fully occluded after intracoronary nifedipine. Infusion of intracoronary streptokinase (4000 IU/min) was started, and in 20 minutes the patient's chest pain resolved. Repeat injection showed slight opening of the LAD coronary artery with residual 95% stenosis at the level of the former proximal occlusion (Fig. 2, *B*). At this point in the evening the procedure was halted and the patient returned to the coronary care unit. Creatine phosphokinase (CPK) at the start of the procedure, 1 hour after onset of chest pain, was 18 IU (normal is less than 50 IU). Repeat CPK values were 283 IU at midnight and 287 IU early the following morning. The patient remained free of pain overnight and her ECG was unchanged from the initial recording shown in Fig. 1, *A*.

The patient was then returned to the catheterization laboratory and transluminal coronary angioplasty was performed via the right femoral artery, using the intra-arterial sheath left in place from the streptokinase catheterization the previous evening. The procedure was performed with a transluminal angioplasty catheter (Schneider Medintag AG, Zurich, Switzerland). As with all cases of percutaneous transluminal coronary angioplasty in our institution, the cardiac surgical team was on standby for emergency bypass surgery if necessary. After four 30-second dilatations, the gradient measured over the proximal LAD artery narrowing decreased from 80 to 4 mm Hg, and the stenosis appeared to be reduced to about

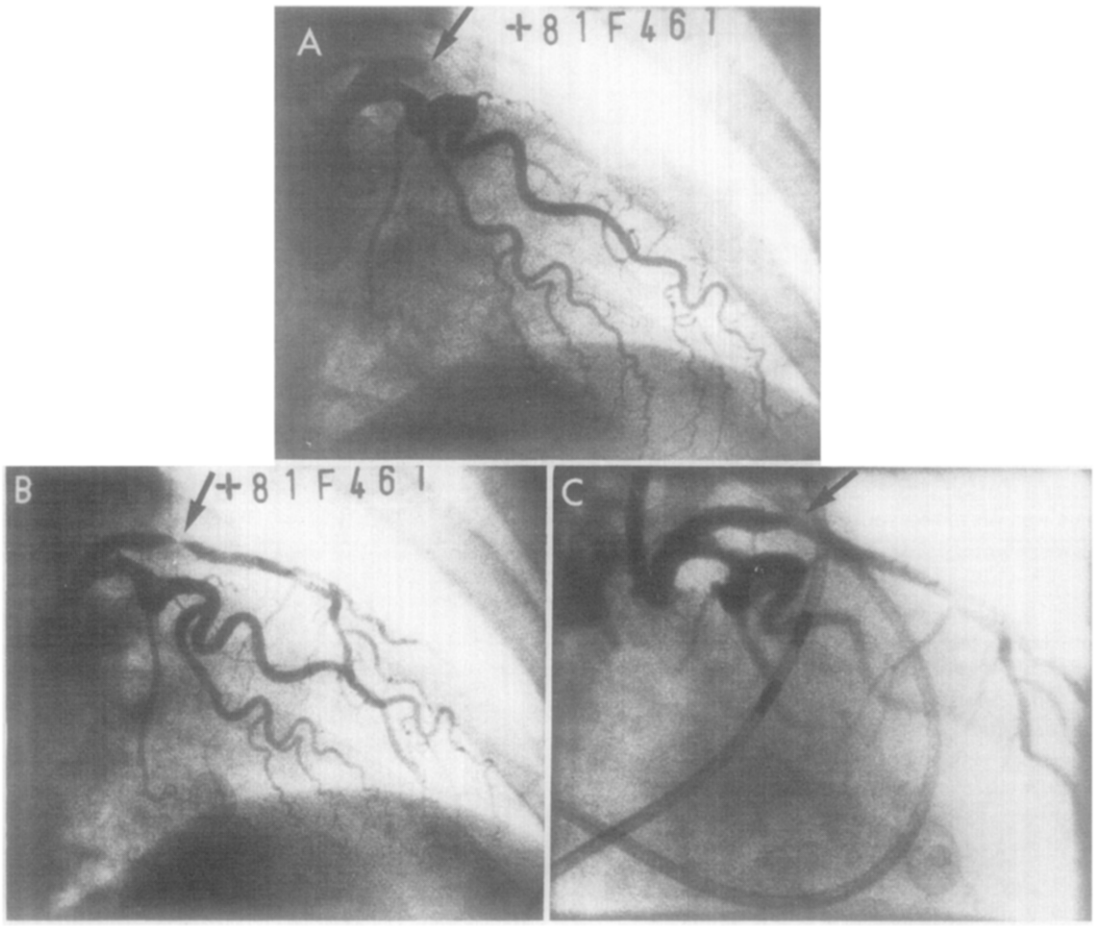

Fig. 2. A, Frame from initial coronary arterial injection showing entirely occluded proximal LAD coronary artery *(arrow)*. **B,** Frame from coronary arteriogram after intracoronary streptokinase infusion showing opening of the former occlusion, but a residual subtotal stenosis *(arrow)*. **C,** Frame from postangioplasty coronary arteriogram showing decreased stenosis *(arrow)*.

60% (Fig. 2, *C*). The patient remained without chest pain during the procedure and afterward. There was a large right inguinal hematoma extending to the thigh as a complication of the procedure, but this gradually resolved. The CPK level returned to normal, and the ECG changes evolved over the next day to show an anteroseptal myocardial infarction with new small Q waves in leads V_1 through V_3 (Fig. 1, *C*).

This case shows that intracoronary streptokinase infusion and percutaneous transluminal coronary angioplasty can be used sequentially to provide reperfusion in the setting of impending myocardial infarction. This approach seems rational if an isolated proximal occlusion opens minimally with streptokinase, since these residual high-grade stenoses may be more likely to reocclude. Furthermore, proximal thrombosis of the LAD coronary artery may be a particularly malignant lesion.[5] At our institution we consider high-grade proximal stenosis of the LAD artery to be a lesion that should eventually require a coronary bypass operation. Thus the transluminal angioplasty spared this patient a thoracotomy. A single case cannot answer the question as to whether the present therapy was better than "traditional" conservative therapy or more experimental and aggressive emergency bypass. However, the satisfactory outcome in this individual patient suggests that this approach may warrant consideration in other cases.

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