Intraaortic Balloon Pumping for Control of Recurrent Myocardial Ischemia

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SUMMARY
Eleven patients were studied because of recurrent angina at rest. In five cases ischemic pain developed 3–10 days after acute myocardial infarction. Pain recurred in all cases despite therapy with bedrest, oxygen, heparin anticoagulation, nitrates, and whenever possible, beta blockade. Intraaortic balloon pumping with a 30-cc balloon prevented ischemia in nine patients and markedly reduced the frequency of ischemic attacks in two. Coronary angiograms were obtained during circulatory assistance without complication and confirmed proximal coronary artery obstructions in all patients. Saphenous vein aortocoronary bypass grafting was then performed. Intraaortic balloon pumping will effectively control myocardial ischemia resistant to medical therapy.

Additional Indexing Words:
Angina pectoris Mechanical circulatory assistance Myocardial infarction

There are two forms of myocardial ischemia which require aggressive management. The first is recurrent angina in the early phase of acute myocardial infarction. The second is accelerated angina at rest, which may complicate a previously stable ischemia pattern. Both conditions are treated in the hospital with oxygen, anticoagulation, and measures designed to reduce myocardial oxygen demand, namely bedrest, vasodilators, and whenever possible, beta-adrenergic blockade. Some patients respond to this combined therapy and become pain free. In others the intensity or frequency of ischemic attacks increase and may result in serious ventricular arrhythmias or progress to frank infarction.

In this group with resistant ischemic pain, circulatory assistance with the intraaortic balloon pump (IABP) can further reduce myocardial oxygen demand by diminishing left ventricular systolic pressure while at the same time increasing coronary perfusion pressure. Furthermore, diastolic counterpulsation has been reported to augment collateral circulation. This study was undertaken to determine (1) whether IABP would interrupt ischemic pain in the absence of cardiogenic shock and (2) whether continuous pumping would prevent recurrent attacks.

Materials and Methods
Eleven patients were studied. Six had repetitive ischemic attacks at rest without evidence of recent infarction (preinfarction group, table 1). Five had recurrent ischemic attacks at rest in the recovery phase of their first transmural myocardial infarction (postinfarction group, table 2).

Prior to study, all preinfarction patients had been under hospital observation for 4–9 days. During this period, pain increased in frequency from an average of three to seven episodes per day while on therapy with heparin anticoagulation, beta blockade, long-acting nitrates, oxygen, and bedrest. Despite the increasing frequency of pain, none showed enzyme elevation or electrocardiographic evidence of myocardial infarction.

In all postinfarction patients, the occurrence of myocardial infarction was documented by a typical history, electrocardiographic changes, and serial enzymes. The average time from infarction to recurrent ischemic pain was 4 days. In three patients, pain recurred despite therapy with heparin anticoagulation, beta-blocking agents, long-acting nitrates, oxygen, and bedrest. In two, beta blockade and long-acting nitrates
were withheld because of left ventricular failure and hypotension. Observation under drug therapy was continued for an average of 4 days (range 5–11 days, table 2).

Patients in both groups were transferred to the Myocardial Infarction Research Unit (MIRU) Intensive Study Area where hemodynamic monitoring was performed with radial artery cannulae and Swan-Ganz pulmonary artery catheters. Serial enzymes and 12-lead electrocardiograms were taken, and cardiac output measurements were obtained by the dye-dilution technic.

In the MIRU the basic medical program was not altered. In preinfarction patients the dose of oral isosorbide dinitrate was 10 mg every 6 hours. Propranolol was administered in an average dose of 120 mg/day (range 60–200 mg/day). Doses were selected to reduce the resting heart rate below 70 beats/min without producing significant myocardial depression. Propranolol was discontinued in one patient because of hypotension and bradycardia. The dose of propranolol in three postinfarction patients was 120 mg/day and isosorbide dinitrate 10 mg every 6 hours. In all patients heparin dosage averaged 50 mg i.v. every 4 hours adjusted to keep the partial thromboplastin time twice control.

After the initial hemodynamic measurements on therapy, all preinfarction patients were monitored for at least two episodes of rest pain which were documented by 12-lead electrocardiogram, pressure measurements,
Hemodynamic effect of myocardial ischemia on patients without previous myocardial infarction. The asterisks represent the means. MAP = mean arterial pressure; HR = heart rate; P values: MAP = P < 0.01; HR = NS; wedge pressure = P < 0.01.

A graphic representation of the ischemia pattern in a patient without previous infarction. Each open square represents an episode of ischemic pain. Pain increased in frequency on therapy from three to 18 episodes per day. On day 9 after five episodes of pain IABP was begun. The one subsequent episode of pain followed cardiac catheterization.

Figure 1

Figure 2

Table 3

Preinfarction Group

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changes returned to baseline after an average of 3 min.

All preinfarction patients underwent coronary and left ventricular cineangiography during uninterrupted circulatory assistance without significant complication. These studies demonstrated high-grade proximal obstruction of the left anterior descending coronary artery in every case. In two this was the only lesion. The remaining four showed three-vessel disease. All patients were considered operable and underwent aortocoronary saphenous vein bypass grafting with IABP support during induction of anesthesia and in the postoperative period. All survived.

Postinfarction Group

The hemodynamic effects of ischemic pain before IABP are shown in figure 4. Prior to pain heart rate averaged 79 beats/min, mean arterial pressure 80 mm Hg, and pulmonary capillary wedge pressure 13 mm Hg. In contrast to the preinfarction group, ischemia produced no change in mean arterial pressure but a greater change in heart rate. No cardiac output determinations were performed.

The electrocardiogram in four postinfarction patients showed S-T segment elevation during pain in the area of recent infarction. In three this was associated with S-T depression in the leads reflecting the opposite myocardial wall. The fifth patient showed generalized S-T segment depression. Three showed significant ventricular irritability during pain. Serial electrocardiograms and enzymes showed no evidence of new infarction.

IABP produced a marked change in pain pattern (fig. 5). Four showed no recurrence of pain during pumping and in the fifth patient the frequency of attacks was greatly reduced. During interruption of IABP two showed recurrence of ischemic pain within 5 min (table 4). Resumption of IABP was effective with prompt resolution of ischemic signs and symptoms (fig. 6).

**Figure 3**

The effect of IABP on the arterial pressure, pulmonary capillary wedge (PCW) pressure, and lead V<sub>4</sub> of the electrocardiogram in a patient with severe chest pain. After the first four beats the paper speed is slowed. On the left the pressures and electrocardiogram are displayed during pain. At the arrow IABP is begun. Within 3 min the patient is pain free. Note the return of the PCW pressure to normal and the continuous fall in mean arterial pressure during balloon pumping.

**Figure 4**

Hemodynamic effect of pain in postinfarction patients. MAP = mean arterial pressure; HR = heart rate.
IABP TO CONTROL ISCHEMIA

Table 4

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catheterization and was then reversed with IABP. Serial enzymes suggested further myocardial infarction. As expected, all patients in this group showed high-grade obstruction of the coronary artery in the area of previous infarction. In addition all showed high-grade proximal obstruction of the artery supplying the opposite noninfarcted wall. In four this involved the left anterior descending and right coronary arteries, and in one patient with inferior infarction and right coronary artery obstruction, a high-grade main left coronary artery stenosis was found.

All patients were considered operable and underwent vein-bypass grafting with IABP support during induction of anesthesia and in the postoperative period. Four of five survived. The operative death occurred in the patient in whom cardiac catheterization without IABP was complicated by myocardial infarction.

Discussion

Aggressive management of myocardial ischemia in the group of patients without previous myocardial infarction was required because medical therapy had failed. Attacks increased in frequency and often in severity. Furthermore the ischemic nature of the pain was always documented by hemodynamic and electrocardiographic S-T segment changes. The presence and severity of

Figure 5

A graphic representation of the ischemia pattern in a postinfarction patient. Open squares represent pain responsive to nitroglycerin; shaded squares represent pain resistant to nitroglycerin; and the black square indicates infarction. Acute infarction occurred after an increase in severity of ischemic attacks. Following infarction, ischemia recurred with increased frequency and persistent severity until initiation of IABP. There was one subsequent episode of pain during IABP which was again responsive to nitroglycerin.

Five of six patients in this group underwent coronary and left ventricular cineangiography during IABP without complication. The sixth patient underwent angiography without circulatory assistance. The study in this patient was complicated by recurrent ischemia, ventricular tachycardia, and hypotension. Pain and hypotension persisted after

Figure 6

Continuous electrocardiogram, arterial pressure, and wedge pressure in a postinfarction patient during abrupt cessation of IABP. Paper speed changes are indicated by the 1-sec markers. With discontinuation of IABP wedge pressure rose and the S-T segments became more depressed. Three min after resumption of IABP, both signs of ischemia were reversed and pain was relieved.
coronary artery disease was confirmed by angiography. In this category of patients the 1-year mortality has been reported to be 43%.13

In patients without previous myocardial infarction, ischemic pain recurred despite beta blockade. Only moderate doses of propranolol were employed. Resting heart rate was reduced, and during pain the maximum heart rate recorded was 83 beats/min. The dosage of propranolol was not increased to maximum tolerable oral levels because of the potential of myocardial depression which can be especially hazardous when ischemia progresses to frank infarction.

Although ischemic pain could be temporarily interrupted by nitroglycerin in all but one patient, recurrent ischemic attacks could not be prevented by oral isosorbide dinitrate. Serious ventricular irritability occurred during ischemic pain in four patients and could not be suppressed by intravenous xylocaine infusion. In order to halt this progression of myocardial ischemia, more direct therapy was required.

The patients with myocardial infarction and recurrent ischemic pain at rest required more urgent application of mechanical circulatory assistance. All had recently suffered a moderate-sized transmural infarction. Electrocardiograms recorded during the pain suggested either renewed injury in the area of infarction or ischemia involving the opposite left ventricular wall. Infarction of the opposite left ventricular wall would have been either debilitating or fatal. Moreover, medical therapy in this group is more limited because of the recognized hazards in the administration of nitrates and beta-blocking agents in this setting.

The interruption of ischemic attacks by IABP allowed patients a pain-free interval during which coronary angiography could be performed at low risk. The studies were performed with careful attention to balloon timing and with continuously monitored arterial pressure. Even with uninterupted balloon assistance, pain occurred in several patients after coronary injection. In two cases there was severe hypotension transiently following repeated injections of the left coronary artery. This could be reversed by continued IABP and brief catecholamine infusion. There was no evidence of myocardial injury after angiography and no patients developed ventricular fibrillation.

After an average of 24 hours of balloon pumping, there was no clear evidence that the underlying coronary pathology had been altered such that mechanical circulatory assistance was no longer necessary. Five of 11 patients showed recurrent ischemic pain within minutes of cessation of IABP. The remaining six did not show recurrent pain but were not stressed by prolonged interruption of circulatory assistance. The data herein presented does not rule out the possibility that a longer course of pumping in some patients might be beneficial.

All patients in this series were treated without morbidity from IABP. There was no clinical evidence of peripheral embolism, significant platelet depression, aortic or femoral artery trauma. Thus, IABP is an effective and safe device for interrupting recurrent myocardial ischemia resistant to medical therapy. Beneficial effects are seen even when systemic pressure is elevated. By preventing the recurrent ischemia it provides a period of clinical stability allowing deliberate angiographic evaluation and selection of definitive therapy.

Acknowledgment

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