Acute Intervention During Myocardial Infarction in Patients with Prior Coronary Bypass Surgery

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Thrombolytic intervention with or without coronary angioplasty is becoming widely accepted as standard therapy for patients who present early with acute myocardial infarction. Thrombolytic therapy has been shown to improve left ventricular function[1] and reduce mortality.[2] Nearly all clinical trials of myocardial reperfusion have excluded patients with a history of coronary artery bypass grafting. Furthermore, the only available data for the use of thrombolytic agents in this patient group consist of case reports of intracoronary thrombolytic agents[3] and a small series of intravenous thrombolytic agents.[4] There are no published data for the use of angioplasty or combined thrombolysis and angioplasty in patients with acute infarction after bypass surgery. Considering the increasing number of patients undergoing coronary artery bypass grafting and their propensity for future cardiac events,[5] important questions regarding the optimal treatment strategy for prior coronary artery bypass patients come to bear. Accordingly, we reviewed our experience in the treatment of patients after coronary artery bypass surgery in the setting of evolving myocardial infarction.

Records from the University of Michigan Cardiac Catheterization Laboratory were reviewed from January 1, 1984, to December 30, 1987, and a database of patients with a history of coronary artery bypass grafting and acute myocardial infarction was tabulated. Acute myocardial infarction was diagnosed when an episode of characteristic chest pain lasting ≥30 minutes was associated with a transient, temporally appropriate increase of the total serum creatine phosphokinase above the upper limit of normal with myocardial isoenzyme fraction greater than twice normal. Data were compiled regarding the presence and type of standard electrocardiogram tracings at the time of infarction. Details of acute interventions, when performed, were accumulated, including those concerning the infarct-related vessel and whether or not the patient was treated with thrombolytic agents, direct angioplasty or a combination of the 2. Status of the infarct-related vessel after intervention was also noted. Follow-up data regarding hospital discharge, repeat catheterization, exercise stress testing and the need for further intervention, including repeat bypass surgery, were also obtained.

Data are expressed as mean ± 1 standard deviation. A comparison of the efficacy of acute intervention as defined by infarct-related vessel patency after intervention was estimated by examining a cohort of patients who presented with acute myocardial infarction and had no previous history of bypass surgery treated with thrombolysis and/or angioplasty in the Thrombolysis and Angioplasty in Myocardial Infarction-1 trial.[6] Estimations of statistical significance were made using a chi-square test.

As listed in Table I, 40 patients met the criteria delineated previously during the time frame stated. The time since coronary artery bypass was 6 ± 3.5 years (range 2 months to 16 years). Age of the patients was 57 ± 10 years (range 33 to 77); 31 of 40 were male (72%). Although all patients presented with symptoms consistent with acute myocardial infarction and subsequently ruled in by enzymatic criteria, only 21 of 40 (53%) had typical ST-segment elevation or new Q waves. The remainder presented with ST depression or nonspecific electrocardiogram changes, thus not allowing localization of the site of myocardial infarction by these means. Thirty-five of the patients (88%) underwent diagnostic cardiac catheterization within close temporal relation to the onset of their symptoms. Of these patients, the infarct-related vessel could be reliably identified in 26 (74%); 12 were native vessel occlusions (46%) and 14 were saphenous vein grafts (54%). Left internal thoracic artery grafts were not present in any of our patients. Acute intervention was attempted in 20 of these patients in whom the infarct-related vessel could be ascertained. The time from onset of symptoms to intervention was 4 ± 1 hours (range 2 to 6).

Intravenous thrombolysis was only used in 4 (10%) of the patients (100 mg of recombinant tissue plasminogen activator in 2 cases, 1.5 million U of intravenous streptokinase in 1 case and 500,000 U of urokinase in 1 case) and was successful in establishing vessel patency in 3 (75%). The infarct-related artery of the patient with unsuccessful reperfusion was a native right coronary artery. Direct coronary angioplasty of the infarct-related vessel was performed in 9 patients (23%) and vessel patency was achieved in 7 (78%). The 2 unsuccessful coronary angioplasties were in saphenous vein grafts, 1 to the left anterior descending artery and 1 to an obtuse marginal branch. Combined angioplasty and thrombolytic therapy (intracoronary urokinase or streptokinase) was performed in 7 patients and resulted in reperfusion in 6 (86%). The unsuccessful combined therapy was in a patient with a native right coronary as the infarct-related vessel. Angiographically demonstrable embolic complications were not noted. The total success rate of all acute interventions in establishing infarct-related vessel patency was 16 of 20 (80%). A similarly treated cohort of patients obtained from the Thrombolysis and Angioplasty in Myocardial Infarction trials revealed a reperfusion rate of 92% in 855 patients with native coronary artery thrombosis receiving thrombolysis and/or coronary angioplasty ($p = 0.06$). Of the patients not undergoing acute percutaneous intervention in this study, 2 had

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TABLE I Demographic Characteristics and Outcomes

Age (yrs), Sex	Years After CABG	Location of MI	Treatment	ECG Localization	Infarct Conduit	Patent Conduit	Outcome
57, M	6	NQMI	Medical	Undetermined	Undetermined	—	Negative ETT
51, M	5	Anterior	PTCA	Anterior	SVG-LAD	+	Died
46, F	0.5	Inferior	Medical	Inferior	Right	—	Patent artery at 1 yr
55, F	1	NQMI	CABG	Undetermined	Undetermined	—	Negative ETT
70, M	16	NQMI	Medical	Inferior	Undetermined	—	Unknown
56, M	7	Inferior	Medical	Inferior	OM	—	Negative ETT
43, F	2	Inferior	Combined	Inferior	Right	0	PTCA of nonIRV
57, M	5	NQMI	Medical	Undetermined	Undetermined	—	Died
43, M	2	Inferior	Combined	Inferior	Right	+	Patent artery at 6 months
51, M	5	Inferior	Combined	Inferior	SVG-RCA	+	Restenosed IRV, unsuccessful PTCA
47, F	5	Inferior	Combined	Inferior	Right	+	Restenosed IRV, successful PTCA
58, M	9	NQMI	Medical	Undetermined	Undetermined	—	Died
58, M	3	Inferior	Medical	Inferior	SVG Right	—	Negative ETT
45, F	11	Inferior	Thrombolysis	Inferior	SVG Right	+	PTCA of Residual Stenosis
76, M	3	NQMI	Medical	Undetermined	Undetermined	—	Negative ETT
61, M	6	NQMI	Medical	Undetermined	LAD	—	Delayed PTCA of IRV
56, M	6	Inferior	Combined	Inferior	SVG-OM	+	Repeat CABG
55, F	8	NQMI	PTCA	Undetermined	SVG-OM	0	Died 3 months later
56, M	8	NQMI	PTCA	Undetermined	SVG-LC	+	Repeat CABG
58, F	5	NQMI	CABG	Undetermined	Undetermined	—	Died
67, M	13	Inferior	Medical	Inferior	SVG-Right	—	Unsuccessful PTCA of IRV
56, M	11	Inferior	Thrombolysis	Inferior	SVG-OM	+	Delayed PTCA of IRV
65, F	6	Anterior	Thrombolysis	Anterior	SVG-LAD	+	IRV patent, negative ETT
56, M	4	NQMI	Medical	Undetermined	LC	—	Repeat CABG
60, M	6	Inferior	Combined	Inferior	Right	+	Patent IRV at repeat angiography
49, M	3	NQMI	PTCA	Undetermined	SVG-LAD	0	Repeat CABG
57, M	6	NQMI	PTCA	Undetermined	SVG-OM	+	Negative ETT
60, M	6	NQMI	PTCA	Undetermined	SVG-OM	+	Negative ETT
57, M	10	NQMI	Medical	Undetermined	Undetermined	—	Repeat CABG
77, M	10	NQMI	Medical	Undetermined	Undetermined	—	Died 2 months later
62, M	0.25	Inferior	PTCA	Inferior	SVG-OM	+	Positive ETT, medical therapy
33, M	0.1	Inferior	PTCA	Inferior	Right	+	Patent IRV at repeat angiography
75, F	6	NQMI	Medical	Undetermined	Undetermined	—	Died 3 months later
64, M	11	NQMI	Medical	Undetermined	Undetermined	—	Unknown
72, M	5	NQMI	Medical	Undetermined	Undetermined	—	Negative ETT
44, M	4	Inferior	PTCA	Inferior	Right	+	Patent IRV at repeat angiography
62, M	5	Inferior	Combined	Inferior	Right	+	Negative ETT
61, M	7	NQMI	Medical	Undetermined	Undetermined	—	Died 4 months later
51, M	6	Inferior	Thrombolysis	Inferior	Right	0	Delayed PTCA of IRV
63, M	10	Inferior	Medical	Inferior	Undetermined	—	Died 3 months later

CABG = coronary artery bypass graft; Combined = treatment with thrombolysis and angioplasty; ECG = electrocardiogram; ETT = exercise tolerance test; IRV = infarct-related vessel; LAD = left anterior descending artery; LC = left circumflex artery; MI = myocardial infarction; NQMI = non-Q-wave myocardial infarction; OM = obtuse marginal; PTCA = percutaneous transluminal coronary angioplasty; SVG = saphenous vein graft; + = yes; 0 = no.

emergency repeat coronary artery bypass grafting and the remaining 18 were treated medically.

Four patients died in the hospital (10%). Two of these patients had been treated medically; 1 died during emergency repeat coronary artery bypass surgery and the other died during acute coronary angioplasty (without combined thrombolysis). The intermediate-term postdischarge outcome was available in 38 of 40 patients. There were 4 deaths within the first 3 months, 3 in patients who had been treated medically and 1 by acute intervention with angioplasty only. Five patients had repeat coronary artery bypass grafting, 2 of whom had been treated medically, 2 by angioplasty only and 1 by combined thrombolysis and angioplasty. Eight patients had further revascularization by coronary angioplasty. Three of the 4 patients treated by thrombolysis alone had angioplasty of a residual stenosis of the infarct-related vessel. Two patients from the group initially treated with combined angioplasty and thrombolysis had restenosis of the infarct-related artery of which one was successfully dilated. One other patient from the combined treatment group had a delayed angioplasty of a noninfarct-related artery. Two patients in the medically treated group had delayed angioplasties; 1 was successful. Thus, 8 of 20 patients (40%) initially treated with acute intervention went on to have successful revascularization procedures after their acute infarct; 1 was lost to follow-up and 1 died. The remainder had patent infarct-related arteries at recatheterization or were asymptomatic clinically.

The number of patients undergoing coronary artery bypass grafting with saphenous vein grafts has been steadily increasing.[7] Considering that the rate of late graft occlusion is approximately 4%/year[8] and the yearly rate of myocardial infarction after coronary artery bypass surgery is approximately 3%,[9] the number of patients at risk for these events continues to increase. Thus, this group of patients represents an increasing proportion of cases of acute myocardial infarction that may benefit from acute intervention. However, the experience on the

use of thrombolytic agents or angioplasty in these patients remains very limited.

This study reports our experience in patients after coronary artery bypass grafting in the setting of acute myocardial infarction. Although the number of patients available for review is small when compared to large scale clinical trials, it represents a large series of such patients reported to date. Furthermore, we report all such patients presenting to our institution regardless of whether or not an intervention was performed.

We noted a limited ability of the standard electrocardiogram to corroborate acute myocardial infarction and aid in ability to determine the infarct area. The preponderance of non-Q-wave myocardial infarcts in our study probably relates to either the finding that the infarct-related vessel is less likely to be a main epicardial artery[10] or to the presence of collaterals. Thus, such infarcts are usually smaller in postcoronary artery bypass surgery patients.[11] This observation may impede the ability to promptly intervene in these patients when they present with acute myocardial infarction.

Furthermore, even with angiography the infarct-related vessel could not be reliably identified in a fairly large proportion of our patients. This phenomenon was due to the presence of >1 vessel occlusion or multiple discordant wall motion abnormalities. With our concern over patient safety intervention with direct angioplasty or intracoronary thrombolysis was not possible in this group.

However, in our patients in whom the infarct-related vessel could be ascertained and acute intervention was performed, the results as assessed by vessel patency compared less favorably with a cohort of patients who never had bypass surgery. Although the lesions encountered in these patients may have been somewhat more "resistant" to thrombolytic therapy or angioplasty, there was yet a high degree of success. We did not encounter overt angiographic evidence of embolic complications as reported by others.[12] Furthermore, more definitive revascularization with redo surgery was then possible in many of these patients at a later date. The limited number of patients in our study preclude recommendations as to the general treatment of such patients. However, based on this experience acute intervention in this setting is a viable option, albeit with a decreased reperfusion success rate, but with a potentially positive impact on the prognosis of these patients. There appears to be no legitimate reason to exclude this important group of patients from prospective reperfusion trials.

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Effects of Verapamil on the Anaerobic Threshold and Peak Oxygen Consumption in Effort Angina Pectoris

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The anaerobic threshold is the submaximal level of exertion above which lactic acid progressively accumulates in the blood and fatigue progressively increases during incremental exercise.[1,2] The anaerobic threshold is a useful clinical measurement of submaximal exercise performance in normal subjects[1–3] and in patients with congestive heart failure,[4] angina pectoris[5] and valvular regurgitation,[6] and has been used to assess drug interventions.[7] In patients with effort angina, ST-segment depression is reduced and exercise time increased after calcium antagonists.[8,9] If relief of myocardial ischemia by verapamil allows an improvement in peak oxygen consumption (VO_{2peak}), there may also be an improvement in oxygen metabolism by exercising muscle and the anaerobic threshold may increase, delaying the onset of fatigue. This study determines whether verapamil alters the oxygen consumption (VO_2) at the anaerobic threshold, or the perception of leg fatigue in patients with effort angina pectoris.

Sixteen patients with stable exertional angina pectoris and positive exercise tests (Bruce protocol with at least 1-mm horizontal ST-segment depression) entered the study. No patient had chronic lung disease, valvular regurgitation, clinical heart failure or peripheral vascular disease. All patients gave written informed consent

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