Selective Evaluation and Management of Coronary Artery Disease in Patients Undergoing Repair of Abdominal Aortic Aneurysms

A 16-year Experience

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Reduction of cardiac mortality associated with abdominal aortic aneurysm (AAA) repair remains an important goal. Five hundred consecutive urgent or elective operations for infrarenal nonruptured AAA were reviewed. Patients were divided into three groups based on preoperative cardiac status: group I (n = 260, 52%), no clinical or electrocardiographic (ECG) evidence of coronary artery disease (CAD); group II (n = 212, 42.2%), clinical or ECG evidence of CAD considered stable after further evaluation with studies such as dipyridamole-thallium scanning, echocardiography, or coronary arteriography; group III (n = 28, 5.6%), clinical or ECG evidence of CAD considered unstable after further evaluation. Group I had no further cardiac evaluation and groups I and II underwent AAA repair without invasive treatment of CAD. Group III underwent repair of cardiac disease before (n = 21) or coincident with (n = 7) AAA repair. In all instances, perioperative fluid volume management was based on left ventricular performance curves constructed before operation. The 30-day operative mortality rate for AAA repair in all 500 patients was 1.6% (n = 8). There was one (0.4%) cardiac-related operative death in group I, which was significantly less than the five (2.4%) in group II ($p < 0.02$). Total mortality for the two groups were also significantly different, with one group I death (0.4%) and seven group II deaths (3.3%), ($p < 0.02$). These data support the conclusions that (1) the leading cause of perioperative mortality in AAA repair is myocardial infarction, (2) correction of severe or unstable CAD before or coincident with AAA repair is effective in preventing operative mortality, (3) patients with known CAD should be investigated more thoroughly to identify those likely to develop perioperative myocardial ischemia so that their CAD can be corrected before AAA repair, and (4) patients with no clinical or ECG evidence of CAD rarely die of perioperative myocardial infarction, and thus selective evaluation of CAD based on clinical grounds in AAA patients is justified.

REPAIR OF ABDOMINAL aortic aneurysm (AAA) has become a safe operation in the past two decades and clearly has been shown to prolong life. However coronary artery disease (CAD) is often present in patients with AAA, and myocardial infarction (MI) is the major cause of perioperative death. To minimize the risk of cardiac mortality, some authors recommend routine preoperative coronary angiography, with coronary revascularization preceding aneurysm repair when indicated. Others have recommended routine noninvasive preoperative studies to identify patients at risk for perioperative MI. During the past 16 years, we have stratified prospectively patients with AAA into three groups before operation based on preoperative clinical evidence of CAD. Subsequent patient management was determined by this initial stratification. We now report the results of this policy in 500 consecutive patients who underwent repair of nonruptured AAA.

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Methods

Patient Population

The records of 500 consecutive patients with nonruptured, infrarenal AAAs repaired by the senior authors between 1973 and 1989 were retrospectively reviewed (Table 1). There were 94 (19%) women and 406 (81%) men, with average age 69 years (range, 42 to 94 years). The median maximal AAA diameter was 6 cm (range, 4 to 15 cm). Symptoms were present in 168 (34%) and 332 (66%) were asymptomatic. Most of the patients (86.9%) had one or more known operative risk factors, with the most common (48%) being CAD (Table 2).

Preoperative Evaluation of Cardiovascular Risk

Patients were divided into three groups based on their cardiovascular risk status (Table 3). Group I consisted of

TABLE 1. *Patients Operated on for Abdominal Aortic Aneurysm (1973–1989)*

No. Patients	500
Male:Female	406:94
Average age (years)	69 (42–94)
AAA asymptomatic	332
AAA symptomatic	168
Median AAA diameter (cm)	6 (4–15)

260 (52%) patients with no clinical evidence of CAD, as demonstrated by the absence of symptoms and a normal ECG. Group II contained 212 (42.4%) patients who had either symptoms or ECG abnormalities suggestive of CAD, and after further cardiac evaluation were considered to have stable CAD. At the discretion of the vascular surgeon and cardiologist, the severity and stability of the CAD in most of the group II patients were determined by echocardiography, resting gaited pool ejection fraction assessment, dipyridamole-thallium imaging, exercise tolerance testing, exercise thallium imaging, holter monitoring, or coronary angiography. Group III included 28 (5.6%) patients with severe or unstable CAD, as determined by coronary angiography in addition to noninvasive evaluation. The group III patients were further stratified into two groups: (a) asymptomatic AAA (21 patients) and (b) symptomatic AAA (7 patients). Group IIIa patients with asymptomatic AAA underwent staged procedures with initial coronary artery bypass and subsequent AAA repair. Group IIIb patients with symptomatic AAA underwent combined cardiac and AAA repair.

Perioperative Management

The perioperative management of these patients has been described previously.[1] Radial artery and thermodilution pulmonary artery catheters were routinely placed for monitoring of systemic blood pressure, pulmonary capillary wedge pressure, pulmonary artery pressures, and cardiac output. Monitoring lines were inserted the evening before surgery and serial intravenous boluses of crystalloid solution were administered to assess left ventricular performance. These catheters also were used during and after operation to assist in fluid and pharmacologic therapy. Repair of the AAA was accomplished *via* the graft inclusion technique of Creech,[2] as previously described.[1] Tube grafts were placed in 66% of patients, and 34% received bifurcation grafts. Coincident with AAA repair, various secondary procedures also were performed in 83 (17%) patients (Table 4). Immediately preceding AAA repair, seven patients underwent repair of unstable cardiac disease. Nephrectomy of nonfunctioning kidneys was performed in 12 cases with associated renin-mediated hypertension. Renal artery reconstructions were performed in 23 patients. Femoral embolectomy or femoral–femoral bypass were performed in 26 patients to restore lower-extremity perfusion. The inferior mesenteric artery was reimplanted in the arterial circulation in 10 patients for prevention of intestinal ischemia. A clip was used to interrupt the inferior vena cava in two patients to prevent pulmonary emboli. Cholecystectomy was performed in six patients for chronic cholecystitis and intraoperative splenic laceration necessitated splenectomy in five patients.

TABLE 2. *Operative Risk Factors in Patients Operated on for Abdominal Aortic Aneurysm*

Risk Factor	Percentage
Coronary artery disease	48.0
Hypertension	47.6
Chronic pulmonary disease	27.4
Arrhythmia	9.7
Peripheral vascular disease	8.4
Chronic renal disease	8.4
Diabetes	8.0
Congestive heart failure	7.9
Cerebrovascular disease	7.4

TABLE 3. *Cardiac Risk Classification*

Group	Number of Patients	%	Criteria
I	260	52.0	No symptomatic or ECG evidence of CAD
II	212	42.4	Symptomatic or ECG evidence of CAD, stable
III	28	5.6	Severe, unstable CAD
IIIa	21	4.2	Asymptomatic AAA
IIIb	7	1.4	Symptomatic AAA

ECG, electrocardiographic; CAD, coronary artery disease; AAA, abdominal aortic aneurysm.

TABLE 4. *Secondary Procedures During Repair of AAA in 83 Patients*

Procedure	Number of Patients
Cardiac	
Coronary artery bypass (CABG)	5
Aortic valve replacement	1
Aortic valve replacement/CABG	1
Renal	
Nephrectomy	12
Renal artery reconstruction	23
Other vascular	
Femoral embolectomy	23
Femoral–femoral bypass	3
Reimplantation of IMA	10
Inferior vena cava clip	2
Miscellaneous	
Cholecystectomy	6
Splenectomy	5
Resection of pheochromocytoma	1
Total procedures	92

AAA, abdominal aortic aneurysm.

TABLE 5. *Postoperative Morbidity in 500 Patients Operated on for Abdominal Aortic Aneurysm*

Complication	Number of Patients	Percentage
Myocardial infarction	15	3.0
Arrhythmia	116	23.2
Renal failure	28	5.6
Pneumonia	27	5.4
Pulmonary embolus	4	0.8
Ischemic colon	3	0.6
Reoperation for hemorrhage	5	1.0

All patients were followed for complications throughout their hospitalization and as outpatients through the 30-day postoperative period. Data were analyzed for statistical significance using Fishers exact test. The 30-day operative mortality rate for the 500 patients undergoing AAA repair was 1.6% (8 deaths). Myocardial infarction resulted in 6 deaths (1.2%) and another 2 (0.4%) patients died of pulmonary emboli. The operative mortality rate was 2.3% for urgent cases and 1.2% for elective cases.

Postoperative complications, ranging from minor arrhythmias to fatal MI, occurred in 196 (39%) patients (Table 5). Electrocardiographic or cardiac isoenzymes or both revealed evidence of MI in 15 (3%) patients. Arrhythmias were noted in 116 (23%) patients, most of which were inconsequential. Pneumonia complicated the postoperative course of 27 (5.4%) patients, and renal failure requiring serious consideration or actual institution of dialysis occurred in 28 (5.6%) patients. Colon ischemia necessitating bowel resection occurred in three patients (0.6%), and reoperation for postoperative hemorrhage was required in five patients (1%).

Cardiac Risk and Operative Results

In group I only three patients suffered perioperative MI, and one of them died. This represents a 0.4% mortality rate for patients with no preoperative symptoms or ECG evidence of CAD (Table 6).

The 212 patients in group II (42.4%) were thought to have stable cardiac disease before operation. Twelve group II patients experienced a perioperative myocardial infarction, five of whom died, representing a 2.4% cardiac related mortality rate. In addition two group II patients died from pulmonary emboli, making the total group II mortality rate 3.3%. The cardiac related mortality rate was significantly higher for group II compared to group I ($p < 0.02$) patients, as was the total mortality rate for the two groups.

Among the 21 group IIIa patients treated with CABG and staged AAA repair within 6 months for unstable cardiac disease and asymptomatic AAA, there were no MIs or deaths.

The seven patients (1.4%) in group IIIb, defined as having unstable cardiac disease and symptomatic AAA, underwent urgent combined procedures with repair of the cardiac disease followed by repair of the AAA. Five patients with crescendo angina or left main CAD unresponsive to medical therapy underwent CABG and then AAA repair before chest closure. One patient, who had critical aortic stenosis, severe CAD, and a symptomatic AAA, underwent an aortic valve replacement, CABG, and then AAA repair as a combined procedure; another patient with critical aortic stenosis and a symptomatic AAA received an aortic valve replacement and repair of AAA as a combined procedure. No patient in group IIIb suffered MI or death.

Discussion

On March 29, 1951, Charles Dubost used an aortic homograft during the first successful repair of an aortic aneurysm.[3] His achievement, which added 8 years to his patient's life, stood in stark contrast with notoriously poor outcomes derived from previous methods consisting of aortic ligation, wiring, or wrapping. While subsequent availability of prosthetic grafts firmly established this approach to aneurysm repair, initial operative mortality rates approximated 20%.[4–6] Vastly improved anesthetic management and the adoption of the graft inclusion technique advocated by Creech were among several factors responsible for the subsequent decline in operative mortality rate to the 4% to 9% range reported in the early 1970s.[2,8–10] These lower mortality figures resulted primarily from significant reduction in postoperative renal and respiratory

TABLE 6. *Myocardial Infarction and Cardiac Mortality in Patients Operated on for Abdominal Aortic Aneurysm*

Postoperative Morbidity	Group I No CAD (n = 260)	Group II Stable CAD (n = 212)	Group III Unstable CAD (n = 28)	Total (n = 500)
Non Fatal MI	2	7	0	9 (1.8%)
Op mortality, cardiac	1 (0.4%)*	5 (2.4%)*	0	6 (1.2%)
Noncardiac	0	2	0	2 (0.4%)
Total	1 (0.4%)	7 (3.3%)	0	8 (1.6%)

* $p < 0.02$.

CAD, coronary artery disease; MI, myocardial infarction; Op, operative.

failure, while the incidence of cardiac complications remained virtually unchanged.[11]

Reduction in cardiac mortality rate was forthcoming in the 1970s as the physiology of declamping hypotension became better understood and monitored perioperative fluid administration more routine.[1,12-14] Sophisticated management of the compromised myocardium reduced the anticipated operative mortality rate consistently below 6% by 1980, yet the primary cause of death remained cardiac.[1,15-20]

Further reduction in operative mortality rate requires, therefore, identification of those patients with CAD at significant risk for postoperative cardiac complications. Reliable predictors would allow more vigorous investigation of high-risk individuals and guide specific management of CAD before or concurrent with aneurysm repair. Predictive methods have included clinical scoring systems and a variety of laboratory investigations.

Several clinical scoring systems have been used to assess operative risk, perhaps the most rigorous of which is the Goldman risk index, according to which poorly controlled congestive heart failure and recent myocardial infarction represent the most ominous predictors.[21,22] Although proved accurate in several series, the index criteria were developed in the late 1970s and may not accurately reflect current management of even the most severe manifestations of CAD.[23-26] Rivers et al.[27] recently documented the incidence of cardiac morbidity in a group of 30 patients undergoing peripheral vascular surgery a median of 11 days after myocardial infarction. While the Goldman risk index accurately predicted the cardiac complication rate in class II and III patients, the 33% cardiac complication rate associated with class IV patients was significantly lower than the 78% level predicted by Goldman. None of these 30 patients, however, underwent an intra-abdominal aortic repair. Similarly Lette and associates[28] examined 66 patients and found no correlation between postoperative cardiac events as predicted by any of the five clinical scoring systems investigated, including the Goldman index, Cooperman scale, Dripps score, Detsky index, and Eagles clinical markers. The patient population in the latter study also was heterogenous but included 48 vascular procedures, 67% of which required aortic cross-clamping. Thus most clinical scoring systems are probably outdated, lack risk adjustment for the specific operative procedure, and do not seem accurate for the patient with a severely impaired myocardium. Perhaps this is not surprising because silent ischemia may be just as, if not more, important than any other clinical parameter.

The exercise tolerance test, widely used to evoke evidence of myocardial ischemia, is subject to many limitations.[29-33] A significant number of vascular patients (25% to 33%) cannot complete the standard examination, which has useful predictive value only when patients achieve 75% to 85% of their predicted maximal heart rate (PMHR).[29,30,33] Unfortunately as many as 70% of patients examined cannot achieve their PMHR,[33] and even if successfully completed, the severity of CAD cannot be accurately assessed in patients with no prior cardiac history.[32] Furthermore subsequent coronary angiography in patients with significant S-T segment depression not infrequently fails to demonstrate significant CAD.[31] The exercise tolerance test therefore may provide equivocal results requiring further stratification before appropriate referral for coronary angiography.

Resting gaited pool ejection fraction (EF), as determined by radionuclide ventriculography, also is associated with equivocal findings.[34-38] Pasternack recently determined EF in 200 patients, 34% of whom underwent AAA repair.[35] Those patients with an EF less than 40% sustained a 17% incidence of perioperative myocardial infarction, significantly greater than the 3.4% incidence observed in patients with EF in excess of 40%. Other reports refute such a correlation, one of which specifically included 60 patients undergoing AAA repair.[36-38] This is perhaps not unexpected because the EF measures the efficiency of left ventricular function at one time point and may not accurately reflect the extent of CAD and associated risk of sudden coronary occlusion.

Dipyridamole-thallium imaging has been shown to correlate well with cardiac risk.[39,40] Thallium redistribution following coronary vasodilatation induced by dipyridamole accurately reflects myocardial susceptibility to postoperative events. Unfortunately the examination is not readily available in most institutions because drug supplies remain severely rationed in the United States.

These difficulties inherent in accurate stratification of patients with CAD has led to the recommendation of routine coronary angiography and preliminary coronary artery bypass grafting (CABG) when surgically reconstructable disease is identified.[41] In more than 1000 such studies in patients before elective vascular surgery, Hertzer et al.[42] found severe but operable CAD in 25% of the entire group and in 31% of those with AAA. Eighteen per cent of those with AAA had proved and operable CAD, yet no prior history of myocardial ischemia. Of the entire group, 216 patients underwent CABG and sustained a 5.3% mortality rate before vascular surgery. Of those who survived CABG, however, only one death occurred during the subsequent vascular procedure. The added risks imposed by coronary angiography and initial CABG have cast doubt on the overall benefits of this approach.[42-44] More selective use of coronary angiography is currently recommended for patients with a positive clinical history and for those asymptomatic patients who are identified on exercise tolerance testing or radionuclide imaging as high-risk indi-

viduals.[42] Although 44% of the entire group of patients with a positive clinical history had severe operable CAD by angiography, a significant 15% of patients with an entirely normal cardiac history also had severe surgically correctable coronary disease.

Because of the unreliability of clinical scoring systems, objective laboratory tests, and the expense and morbidity associated with coronary angiography, we have implemented the clinical classification system reported in this study. Some patients with AAA without clinical evidence of CAD have significant and operable CAD, as demonstrated by Hertzer et al.[42] Despite this our policy of recommending elective repair of AAA without further cardiac work-up in patients with no clinical evidence of CAD seems justified by the minimal cardiac morbidity and mortality established in our initial experience, confirmed by others since, and reaffirmed in the present report.[45–48]

Patients with unstable CAD manifest by crescendo or nocturnal angina unresponsive to medical therapy, including those associated with symptomatic aneurysms, require coronary revascularization before, or concurrent with, AAA repair. None of our patients managed in this fashion died, even among the group of patients undergoing simultaneous CABG and AAA repair. While our experience with preliminary percutaneous transluminal coronary angioplasty is very limited, this may prove to be a safe and expeditious approach for patients with symptomatic aneurysms and correctable coronary disease.

Patients with clinically evident but stable CAD, however, are at the highest risk for perioperative myocardial events. While Hertzer et al.[42] recommend routine coronary artery angiography and CABG, as indicated for this group, we support the use of a more selective policy in which the cardiac mortality rate was limited to 2.1% of patients with known CAD (groups II and III). This figure compares favorably with the 3.9% mortality rate associated with 250 aneurysm patients reported by Hertzer[42] who underwent preliminary coronary angiography and CABG as required.

Further reduction in the cardiac mortality rate may require recognition of myocardial ischemia in these group II patients with CAD. A significant number of patients with surgically correctable CAD in Hertzer's group were clinically asymptomatic.[42] Of significant myocardial ischemic events documented on ambulatory Holter monitoring of vascular surgical patients, 75% of those reported by Pasternack and nearly all of those reported in our experience were, in fact, entirely silent.[49,50] These observations challenge our ability to accurately determine the relative stability of CAD. Prospective studies using routine ambulatory Holter monitoring may allow further stratification of group II individuals and identify those most likely to sustain cardiac events and therefore most likely to benefit from coronary angiography and subsequent intervention.

In conclusion operative deaths resulting from cardiac events associated with repair of AAA has diminished markedly, yet myocardial infarction remains the leading cause of postoperative death in most series, including this one. Significant perioperative cardiac complications rarely develop in patients with no clincial or ECG evidence of coronary disease and thus they warrant no further cardiac evaluation. In contrast, in those patients with severe or unstable coronary disease, correction of the CAD before, or simultaneous with, AAA repair is required to minimize cardiac deaths. Those individuals with established but stable CAD, however, need aggressive investigation to detect those at highest risk for perioperative ischemia so their associated CAD may be corrected before elective AAA repair. The recognition of silent myocardial ischemia in such individuals may prove to be a most important aid in further reducing the operative mortality rate.

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Discussion

DR. E. STANLEY CRAWFORD (Houston, Texas): These authors deserve congratulations for presenting another study of their methods of managing abdominal aortic aneurysm operation that reduce the operative mortality rate to less than 2%, despite the presence of coronary artery disease in nearly one half of the cases. They accomplished this by simply monitoring and managing cardiac function in patients with stable coronary artery disease and coronary bypass before or at operation for abdominal aortic aneurysms in patients with unstable coronary artery disease. I am sure that all of us would agree with this approach and I would like to add that the long-term (10-year) survival rate for patients with both coronary artery disease and abdominal aortic aneurysms who have had both coronary artery bypass and aneurysm replacement is as good as that in the patient without coronary artery disease who had aneurysm replacement.

DR. WAYNE JOHNSTON (Toronto, Ontario, Canada): The results of this study can be generalized to other groups of surgical patients. Clearly patients having aortic aneurysm repair have a very high incidence of coronary artery disease, but I suspect that many other populations of surgical patients who have an average age of 70 years have a similar high incidence.

The data from the Canadian Aneurysm Study supports the authors' conclusion, that patients without clinical or EKG evidence of coronary artery disease do not require further investigation for aneurysm repair.