CURRENT TRENDS IN THE SURGICAL MANAGEMENT OF CORONARY ARTERY DISEASE

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SUMMARY

Coronary artery surgery as we know it today, has undergone rapaid evolution since its introduction in the late 1960's, resulting, not surprisingly, in much confusion. In an attempt to present the state of the art, a collective review of the literature, coupled with the experiences with 118 patients is presented and discussed.

The modern surgical management of patients with obliterative coronary artery disease is designed to relieve symptoms, prolong life and identify patients at high risk of premature death or myocardial infarction if they were to continue with medical management. Though the most common indication for surgery is persistent limitation of life style by severe symptoms, management is influenced by the strong association between such symptoms and increased mortality.

During the short history of coronary bypass surgery, other indications for operation have been influenced by a combination of lessons from the past and hopes for the future. In addition, advances in technology have produced an enormous impetus for improvements in patient evalution, surgical technique and postoperative care. As these advances continue and as more and more late results of surgery become available, answers to questions about surgery may increasingly be deduced from lessons rather than from hopes. As a result of these changes, an aura of confusion has gradually crept into the scene. It is hoped that this collective review, together with experiences gained from a personal series of 118 patients over a 3 year period from 1984 to 1986, will put into proper perspective, the state of the art of surgical management of coronary artery disease today.

OBJECTIVES OF SURGERY

The objectives, in coronary artery bypass surgery as with all operations, are to relieve symptoms, to improve the quality of life, and to prolong life. In an attempt to answer the question as to what extent these goals have been achieved, a search of the literature provided the following information.

(a) Relief of Angina

A review of the literature[1,2,3,4] has shown that 60–90% of patients do experience either complete or almost complete relief from angina after surgery. In our own series of 118 patients, only one patient is still sufficiently troubled by angina to prevent him from returning to his full time occupation.

Many attempts have been made to discredit these excellent results–factors like perioperative infraction of an agina producing ischaemic area, interruption of neuropathways and the placebo effect of surgery have been put forward as factors contributing to the patients'.postoperation well-being. Because of this scepticism, we now have many studies[3,5] to show that angina relief is definitely related to graft patency and completeness of revascularization. Winer and co-workers[5] have shown that patients who suffer perioperative infarction were more likely to experience postoperative angina and not less so. Likewise, the VA co-operative study[1] on chronic stable angina has demonstrated that in surviving patients, 86% were symptom free one year after surgery whereas only 44% of medically treated patients continue to be free of symptoms and that symptom relief correlated well with graft patency.

(b) **Prolongation of life**

As regards prolongation of life, this is a more difficult question to address. This is especially so in the asymptomatic patient who has suffered an earlier myocardial infarction and who on subsequent investigations has been found to have significant coronary artery disease.

The Coronary Artery Surgical Study (CASS)[6] registry which has added much to the confusion has often been quoted. This study which contained the details of 14,249 patients with documented disease in one, two or three vessels and no previous surgery is purported to have a 4-year survival among medically treated patients, (left main stem disease included), of 80% giving an annual mortality of 5.4%. It has not taken into consideration, the large numbers of patients (58%) who have crossed over onto the surgical series when indications warranted operative intervention. In similar fashion, several recent studies on unstable angina[7,8,9,10,11] showed mortality rates with medical treatment ranging from 10–20% with the substantial crossover from the medical group to surgery being ignored.

Notwithstanding this, CASS has however identified two subgroups of patients who are likely to benefit from surgery. Patients with left main stem disease and triple vessel disease on medical treatment have 40% and 57% 5-year survival respectively whereas their surgically treated counterparts have a 91–96% 5-year survival rate.

Questions are now being raised as to whether classification according to the number of vessels is too restrictive since it does not take into account individual anatomic variations or the physiologic importance of the lesions. To quote Gary D Plotnick,[12] "A rose is a rose ..... but triple vessel disease is not triple vessel disease." Patients with multivessel disease do not constitute a homogeneous population. It would seem therefore, that clinical decision making, based only on the number of vessels with high grade obstructive lesions is a one dimensional approach to a multidimensional problem.

With this in mind, several papers with risk stratification are now available. The concensus is that surgery does reduce mortality in high risk patients.[13,14,15] In the VA study,[13] the benefit of surgery was evaluated with patients categorized into high, mid and low risk groups based on four clinical predictors of risk:

1. ST Segment depression in the resting ECG
2. History of Myocardial Infarction
3. History of Hypertension and
4. NYHA Functional Class III + IV

The authors showed a 5-year survival among the surgically treated and medically treated patients of 82% and 64% respectively in the high risk subgroup, 81% and 79% in the mid-risk group and 82% to 93% in the low risk group. They also showed that anatomy alone did not influence the result as evidenced by

the observation that triple vessel disease in medically treated patients in the low risk group had a 5-year mortality of 11% while similarly treated patients in the high risk group had a 5-year mortality of 42%. Their conclusion was that surgical treatment had better results in high risk patients.

The European Coronary Surgery study[14] uses a somewhat different set of parameters. Their risk criteria include

1. ST abnormalities
2. Edvidence of previous myocardial infarction on ECG
3. History of Intermittent Claudication
4. Increasing age and
5. Development of 1.5mm or greater ST depression on exercise testing

It was shown in this study that patients with few or no risk factors have a low mortality rate. For example, mortality in medically treated patients with triple vessel disease but with no risk factors when compared with those with risk factors, varied from 4% to 60% depending on the number of risk factors present. Surgery made no difference to the mortality rate high risk patients. Taking all these factors into consideration, Neutze and White[15] in studying the Green Lane Hospital, Auckland figures, conclude that coronary artery bypass surgery did lead to a significant reduction in the mortality of their patients.

Finally, the Cleveland Clinic data[16,17] has shown that in most patients undergoing bypass surgery, prognosis was determined by a combination of symptoms and the extent of coronary disease–the prognosis being good in patients with few or no symptoms even when coronary artery disease was extensive. Hence, in conclusion, it would be reasonable to summise that surgery will definitely prolong the life of high risk patients. The task therefore lies with the cardiologist to seek out this group of patients and offer them surgery.

RISK OF SURGERY

Our endeavour to achieve these goals must not make us lose sight of the fact that surgery in itself carries an operative risk. Fortunately for the coronary artery patient, this operative morbidity and mortality has decreased significantly over the past decade (Table 1). The improvement in early results can be attributed to better anaesthetic and haemodynamic management pre, intra and postoperatively; improved myocardial protection with the use of cold potassium and blood cardioplegia; more complete revascularisation and a greater surgical experience. Improvements in oxygenators and cardio-pulmonary

Table 1: Morbidity and Mortality Associated with Myocardial Revascularisation

	Cleveland Clinic Foundation[27] 1978	Personal Series 1984–1986
Perioperative Infarction	1.2%	No Figures
Prostoperative Bleeding (Re-exploration)	3.0%	0.5%
Units of Blood used per case	3.1%	1.0%
Respiratory Complications	0.7%	1%
C. N. S. Complications	1.7%	3%
Wound Problems	0.8%	3.1%
Death (All Cases)	1.0%	1.8%

perfusion techniques have allowed for more complicated and more complete revascularisation to be attempted. They have also led to a decrease in incidence of end organ failure and intra-operative blood trauma.

Inspite of these improvements there are patients in whom a higher operative risk can be anticipated (Table 2). Risk appeared to be directly proportional to age : patients in the 70's and 80's having a significantly higher mortality (3–4%) than did younger patients. Coronary anatomy as regards the diffuseness of the disease and the size of vessels available for bypass played an important role. There was no significant difference in mortality among subgroups classified by number of vessels bypassed. Unlike long term results, severity of symptoms at the time of surgery did not affect operative risks. When left ventricular function was considered however, it was found that patients with moderate to severe left ventricular dysfunction, had a significantly higher operative mortality than those with normal function or mild impairment. The presence of diabetes mellitus and left main stem disease were also incremental risk factors. The likelihood of developing a neurologic injury in the perioperative period, is perhaps the most significant risk to the patient undergoing bypass surgery. Here age, again plays an important part; the incidence of stroke in patients more than 70 years of age being significantly higher than younger individuals. Attempts are still being made to further diminish the incidence of this complication by the use of pulsatile perfusion techniques, preoperative diagnosis of carotid artery disease and prebypass carotid endarterectomy if indicated and by the use of single aortic cross clamping to reduce trauma to the aorta.

INDICATIONS FOR SURGERY

The generally accepted indications for surgery are as shown in Table 3, the commonest being chronic stable angina and unstable angina. Figures in Table 3 summarises over own indications

Table 2: Factors contributing to increase operative risks

Increasing Age
Coronary Anatomy
Left-Ventricular Dysfunction
Left Main Stem Disease
Diabetes mellitus
Neurological Incident

Table 3: Indications for Surgery

Chronic Stable Angina	67	(56.8%)
Unstable Angina	37	(31.4%)
Acute Myocardial Infarction (AMI)	–	
Intractable Arrhythmia	–	
Left Ventricular Aneurysm	2	(1.7%)
Ischaemic Cardiomyopathy	7	(5.9%)
Incidental Coronary Artery Disease	5	(4.2%)
Complications of AMI	–	
Total	118	(100%)

Chronic Stable Angina

The reasons for operating on patients with chronic stable angina are obvious; the aims being, as previously discussed, to relieve symptoms and prolong life.

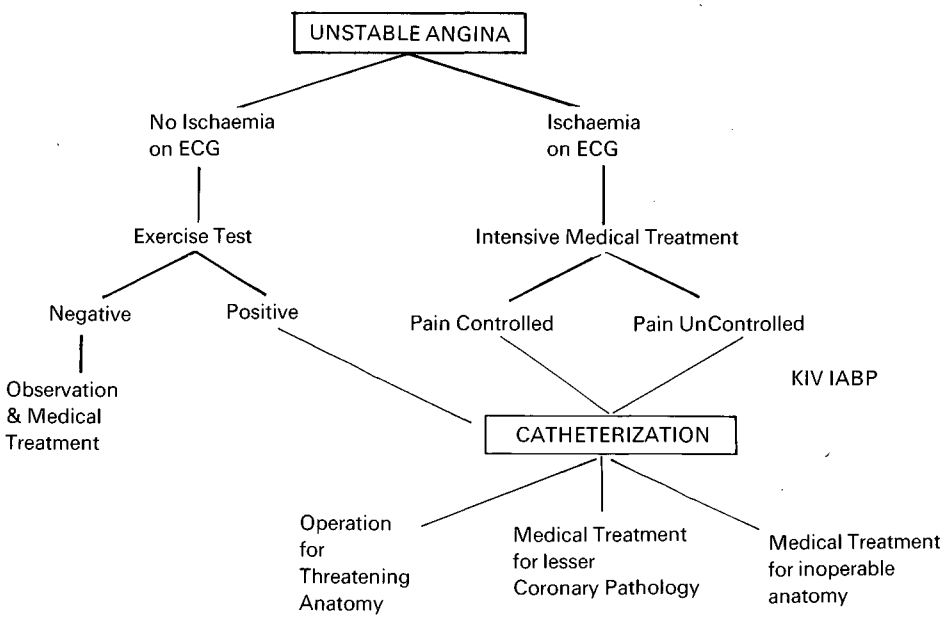
Unstable Angina:[18,19,20,21]

Unstable angina which has been variously called crescendo angina, preinfarction angina, acute coronary insufficiency and intermediate coronary syndrome has been taken as a harbinger of more serious things to come; hence the urgency in knowing what to do with these patients. The criteria for making a diagnosis of unstable angina and the recommended plan of management are summarised in Table 4 and Figure 1 respectively.

Table 4: Clinical Criteria of Unstable Angina

1. Pain : A changing situation
   - (a) Rapidly progressive new onset angina pectoris
   - (b) Acceleration of previously stable angina
   - (c) Severe recurrent rest angina
   - (d) Severe infarction-like pain with no enzyme or ECG evidence of infarction
2. Transient ECG changes associated with angina
3. No new Q waves on ECG in 24 hours
4. No enzyme changes in 24 hours
5. No myocardial infarction within last three months

Fig 1 Flow chart for management of Unstable Angina



In summary, surgery is indicated in

1. Patients who have ECG or myocardial imaging proof of ischaemia if their angiograms show favourable anatomy; especially those with left main stem and triple vessel disease.
2. Patients who require intra-aortic ballon counter pulsation for control of pain if angiograms show favourable anatomy and
3. Patients who have medically refractory angina pectoris and operable anatomy.

Ischaemic Cardiomyopathy

Many patients with ischaemic heart disease do not present with angina but complain instead of breathlessness on exertion, easy fatiguability or even of frank congestive heart failure. Left ventricular aneurysm, mitral regurgitation and ventricular septal defects account for only 30% of these patients, the remaining 70% being due purely to left ventricular dysfunction. Burch et al[22] have coined the term which is well accepted today – Ischaemic cardiomyopathy – when coronary artery disease and left ventricular dysfunction co-exist. The ejection fraction together with the end diastolic pressure have been used to assess the degree of left ventricular dysfunction. Previously, it was felt that patients with ejection fractions of less than 30% and left ventricular end diastolic pressures of more than 25 mmHg had myocardial defects that were irreversible even with bypass grafting. Recent developments, which included better anaesthetic management, improved surgical skills, more complete revascularisation and better intraoperative myocardial preservation, however, have substantially improved the surgical outlook for these patients. It is now accepted that patients with markedly decreased left ventricular function – even those with ejection fractions below 0.2 – can be subjected to aortocoronary saphenous vein bypass with low mortality and good clinical results[23,24].

Left Ventricular Aneurysm

Aneurysms of the left ventricle is the commonest late complication of myocardial infarction, occurring in about 10–38% of patients with acute myocardial infarction. Its natural history with a mortality over a 5-year period of 90% is frightening. Death aside, patients may also present with chronic congestive cardiac failure, systemic embolization or intractable ventricular arrhythmias.

Previously, in view of the poor cardiovascular status of these patients investigations did not go beyond proving the existence of a resectable aneurysm and surgical treatment, in like manner, was limited only to its resection and extraction of mural thrombi. In the past decade, however, with improvements in skills and technology, emphasis has been placed on selective coronary angiography and physiologic assessment of left ventricular function. Surgical treatment, likewise, has been extended to include revascularization of concomitant ischaemic myocardium and ablation of arrhythmogenic scars. These improvements in assessment and surgery have led to an improvement in results.[25,26] Cosgrove and Loop[20] and Cooley and Walker[26] reported an operative mortality ranging from 4–15% depending on the extent of underlying disease, and degree of left ventricular dysfunction. Clearly a more aggressive approach with more complete revascularizaiton has improved the longevity of patients undergoing aneurysmectomy.

Intractable Arrhythmias

Not infrequently an arrhythmogenic area is generated in the scar tissue that has formed as a consequence of a myocardial infarction. The incidence rises to as high as 5–10 per cent of patients with left ventricular aneurysms.[28,29] A large proportion of what was considered intractable arrhythmias previously, however, can be controlled by the newer drugs available today. This is fortunate because simple revascularization and blind aneurysmectomy failed to control the arrhythmias in 50% of patients, because although the irritable focus is most often located near the junction of normal myocardium and scar

tissue, it may sometimes be found quite some distance from the diseased area. Thus, the current treatment of choice for intractable arrhythmias is revascularisation combined with scar resection with the aid of epicardial mapping and endocardial incision in the area closest to the point of epicardial activation.[30,31,31,33]

Acute Complications of Myocardial Infarction

Patients who present with acute complications of a recent myocardial infarction–ventricular septal defects and mitral regurgitation from papillary muscle rupture–are generally extremely ill. Coronary artery grafting in combination with mitral valve replacement or closure of ventricular septal rupture carries a substantial increase in operative mortality.[34,35] Notwithstanding, these poor results, it must be emphasized that surgery offers the only opportunity for survival in the vast majority of these patients.

Acute Myocardial Infarction

In the belief that it my be possible to salvage reversibly damaged myocardiums if patients who have suffered an acute myocardial infarction had their myocardium revascularised early enough, several workers embarked upon operating on such patients.

The time of coronory artery bypass reperfusion appears to be important. It has been shown in some dog models[36,37,38] that reperfusion after three hours of coronary occlusion will reduce ST segment elevation and infarct size. Ventricular function was also noted to improve following reperfusion. Berg and associates[39] using open-chested humans, observed reactive hyperaemia with reperfusion six hours after acute coronary acclusion. Their studies suggest that vein graft reperfusion at 6 hours after acute coronary occlusion can restore severely ischaemic muscle to a functional state. Other workers[40] also confirm that reperfusion within six hours of infarction resulted in the lowest mortality. The initial fear that reperfusion may lead to myocardial haemorrhage and extension of infarction has not been substantiated.[41]

Comparing the results of surgery (in-hospital mortality 2.3% and first year mortality of 1.2%) with those of conventional treatment which has an in-hospital mortality ranging from 12 to 24% and a first year mortality of has high as 14%[42,43,44,45] it would seem reasonable to offer patients with acute myocardial infarction surgery if they presented sufficiently early to benefit from such approach.

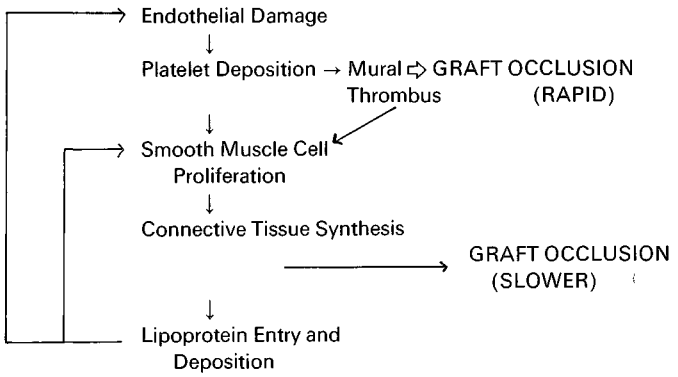
GRAFT PATENCY

One last issue that should be addressed, is that of graft patency. The aim in coronary bypass grafting is to establish flow to vessels distal to the sites of obstruction; hence it is important to ensure that these bypass grafts remain patent. Up to the present time, the standard conduit used for bypass grafting is the saphenous vein so that most patency studies pertain to saphenous veins.[46,47,48] Data extracted from these studies are summarised in Table 5. Various theories have been put forward as to why these grafts thrombose. In summary, the pathogenesis appears to be similar to the development and progression of atherosclerosis except that in the first year, the process is more accelerated (Figure 2).

Table 5 Overall Occlusion Rate

8–18%	Within one month
15–23%	Within 2 to 6 months
16–26%	at 12 months
25–35%	at 5 to 7 years
50%	at 11 years

Figure 2: **PATHOGENESIS OF VEIN GRAFT OCCLUSION (after J.H. Chesebro)**



It is generally agreed that graft occlusion in the first month (early occlusion) is initiated by endothelial damage as may occur during harvesting of the veins by poor handling, exposure to high pressure and significant delay between procurement and anastomosis. The damaged endothelium encourages platelet deposition, adherence and consequent release of platelet factors initiating mural thrombus formation.

Intermediate occlusion (which occurs between one month and one year) has been attributed to smooth muscle and intimal proliferation. This can result from two mechanisms. Firstly, there is smooth muscle proliferation from media to intima and mitogenesis (probably due to plateletderived growth factor). Secondly, there may be secondary proliferation of smooth muscle cells associated with organization of mural thrombi. Statistics show that if proliferation is extensive, total occlusion can result within one year. On the other hand; if proliferation is mild to moderate luminal crosssectional area may be decreased by about 25% by one year.

Late occlusion (after one year) is due to connective tissue synthesis from smooth muscle cells and fibroblasts, followed by incorporation of lipids first intramurally and then extramurally. This process is not unlike that of atherosclerosis. Because of this definite attrition in the number of patent grafts with time, attention has been directed towards measures to prevent or delay graft occlusion and towards finding a more suitable conduit.

Alternative Conduits

Synthetic conduits like the Dacron, Teflon and Goretex grafts have been found to be quite unsatisfactory – especially so when smaller grafts are used. Conversely, since atherosclerosis is rarely observed in the internal mammary artery, the next natural thing to do was to study the patency rate of internal mammary artery bypass grafts. Several studies have shown that after 7–10 years the patency of internal mammary artery grafts is 85–95%.[49,50,51,52,53] A more recent study by loop et al also confrim the above findings. It would seem therefore that the conduit of choice is the internal mammary artery. Unfortunately, not all patients have suitable internal mammary arteries that may be used. Relative contraindications for its use include:

(1) The co-existence of pulmonary dysfunction
(2) Concomitant brachiocephalic artery disease

(3) Poor flow through the internal mammary artery
(4) Small internal mammary artery
(5) Too large a coronary artery (internal mammary artery/coronary artery mismatch) and
(6) The presence of unstable haemodynamics

Technique of Distal Anastomoses

Until recently, the end saphenous vein to side coronary artery anastomoses were the most usual mode of performing the distal anastomosis. Recent data, however, show that sequential grafts remain patent longer provided the most distal anastomosis was to a large vessel. The only disadvantage of this technique is that there should there be a proximal vein occlusion, all the distal anastomoses will be compromised.

Medical Manipulation

While the search for the ideal conduit continues, the onus lies with drug intervention, to delay or prevent graft occlusion. Because platelet deposition appears to trigger the mechanisms of occlusion, drugs capable of interfering with platelet-arterial wall interaction would be the logical choice as opposed to the use of anticoagulants which have no antiplatelet effect. A recent study conducted by the Mayo Clinic Minnesota, utilised Aspirin and Dipyridamole (Persantin). Aspirin blocks the productions of Thromboxane A_2 (a substance that promotes platelet aggregation) while Dipyridamole potentiates the effect of prostacyclin (a substance that inhibits platelet aggregation). Hence the rationale for choosing these two drugs. Since platelet deposition takes place early, even during operation, it was felt by these workers that for any intervention to be maximally effective, it should be begun before surgery. The protocol followed is summarised in Table 6. They showed that these drugs, used in combination, shortened platelet survival in 76% of patients, whereas each drug when used alone will only shorten platelet survival in 38% of patients. It is to be emphasized that potentiation of Persantin by Aspirin is maximised if the aspirin dose is adjusted to 15–20mmg/kg and if both drugs were given simultaneously. This is because aspirin enhances the absorption of Persantin since the absorption of the latter requires an acidic pH (pH less than 4.0). Any manipulation of gastric acidicity by the use of H_2-blockers will therefore inhibit Persantin absorption.

Table 6: Drug Regime for the Prevention of Graft Occlusion

Beginning 2 days before surgery:
Dipyridamole 100mg 6 hourly
On the day of surgery:
2 hours preop: Dipyridamole 100mg
1 hour postop: Dipyridamole 100mg
7 hours postop: Dipyridamole 75mg
and Aspirin 325mg
On the day after surgery and thereafter:
Dipyridamole 75mg TDS
Asprin 325mg TDS

CONCLUSION

This then is the state of the art of surgical myocardial revascularization today. In the short space of three decades we have progressed from an era of blissful ignorance and conjecture to one of masterly activity. The indications for surgery have now become clearer and the broad goals of treatment are being achieved. Initially, the rapid pace of advancement and the multitude of treatment modalities, made evaluation of results difficult if not altogether impossible. Added to this complexity was the fact that we were not dealing with a homogeneous group of patients. As more and more reports appear in the literature however the early and late results of surgery are increasingly predictable. The current status of coronary artery surgery augers well for the coronory patient. Today, with advancements in technology, intraoperative myocardial protection, haemodynamic management and operative skills, the patient with coronary artery disease can look forward to a management protocol that offers him minimal risks and an excellent opportunity for recovery.

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