CARDIAC

BRC 2011

Contents

Overview of the current Guidelines for Revascularisation

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The delegate should be familiar with the limitations of the historical evidence concerning revascularisation. A thorough understanding of the evidence that has been drawn together to support the ESC/EACTS guidelines and the NICE recommendations is mandatory. Particular attention should be paid to the SYNTAX trial.

References

- EACTS/ESC guidelines can be downloaded from respective web sites
- NICE guidance. This has just been agreed in July 2011
- Three year syntax results

Other references

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Assessment of ischaemic heart disease

NICE have recently focussed on and published guidelines on the assessment and diagnosis of patients with chest pain that are suspected to be of cardiac origin.

There are two separate diagnostic pathways

1. Patients with acute chest pain and an acute coronary syndrome (ACS)
2. Patients with stable angina

Acute chest pain (in-hospital assessment)

A 12 lead ECG should be performed at the earliest possible timepoint. The diagnosis of an ACS should not be excluded on a normal resting ECG. In addition, blood should be sent for measurement of either troponin I or T (a second sample should be sent 10-12 hours after onset of symptoms).

An ACS represents a group of clinical conditions secondary to acute myocardial ischaemia. They are differentiated by ECG changes and biochemical markers of myocardial injury and include unstable angina, non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI).

- Unstable angina – Troponin or CK normal, transient ST and T-wave changes or normal ECG
- NSTEMI – Rise in troponin or CK with transient ST and T-wave changes or normal ECG
- STEMI - Rise in troponin or CK, ST elevation or Q waves

UA and NSTEMI are the clinical manifestation of acute plaque rupture and intracoronary thrombosis, but usually without sustained vessel occlusion and hence without significant myocardial damage As soon as a diagnosis of UA or NSTEMI is reached, aspirin and ant-thrombin therapy should be instituted. Individual risk of future cardiovascular events can be established using the Global Registry of Acute Cardiac Events (GRACE) score. Six month mortality for ACS ranges between 3.6-

6.2% and up to 20% are re-hospitalised during the same time period. Patients who are classified as intermediate or high risk of future adverse cardiovascular events should be offered in-hospital angiography with appropriate revascularisation.

In the setting of STEMI, acute plaque rupture and intracoronary thrombosis occur but there is sustained vessel occlusion. Therefore target vessel revascularisation should be considered in this group of patients. This should be achieved by primary PCI or if this facility is not available by thrombolysis. A meta-analysis has demonstrated that PCI is associated with a 22% reduction in mortality, a 57% reduction in re-infarction, and a 50% reduction in stroke compared to thrombolysis and therefore should be the treatment of choice.

Stable angina

Diagnosis of stable angina requires clinical assessment with or without diagnostic testing. Currently available diagnostic tests demonstrate either anatomically obstructive coronary artery disease (CAD) and/or functional testing for myocardial ischaemia. Estimates of the likelihood of CAD are based upon

- Typicality of chest pain symptoms
- Age
- Sex
- Diabetes
- Smoking
- Hyperlipidaemia
- Hypertension
- Early history of familial CAD

Following a full clinical assessment NICE guidelines recommend the following strategies

Coronary angiography

For those patients with a 61-90% likelihood of CAD who would be suitable for revascularisation

Non-invasive functional imaging

For those patients with a 61-90% likelihood of CAD who would not be considered for revascularisation or angiography is not acceptable to the patient.

For patients with a 30-60% likelihood of CAD. Investigations appropriate for non-functional imaging include

- Myocardial perfusion imaging (MPI)
- Stress echocardiography
- Magnetic resonance perfusion or imaging for stress induced wall abnormalities

CT calcium scoring

In patients with a 10-29% likelihood of CAD. If the calcium score is

- Zero, consider other causes
- 1-400 offer CT coronary angiography
- >400 invasive angiography

Notes on methods of detection of CAD

Coronary angiography

Coronary angiography is an invasive cardiological investigation that delineates coronary anatomy via direct injection of contrast into the coronary ostia. It provides a 2D representation of a 3D structure. As well as the coronary arteries, the aortic and mitral valves, ascending aorta and LV function can be assessed. Significant stenosis is defined as a >70% reduction in luminal diameter in an epicardial vessel or in for

the left main stem a 50% reduction. These correspond to a 75% and 91% reduction in cross sectional area respectively.

Alternatively fractional flow reserve (FFR) can be used to determine the haemodynamic significance of a lesion. This is calculated by using a pressure wire that measures the pressure proximal and distal to the lesion after maximal pharmacological vasodilatation. An FFR of <0.8 indicates a significant lesion.

Myocardial perfusion imaging

MPI is used to evaluate patients with either known or suspected CAD, it is able to evaluate patients with symptoms suggestive of ischaemia and to risk-stratify patients with known CAD. During rest myocardial perfusion is governed by the coronary resistance vessels, vasodilatation of these vessels occurs during periods of increased work to increase flow and meet the metabolic demands of the myocardium. In diseased coronary arteries, flow is increased at rest by dilatation of these vessels and therefore during periods of increased workload when there is critical stenosis, there is impairment of coronary flow reserve and the metabolic demands of the myocardium cannot be met.

Myocardial perfusion utilises radiopharmaceutical agents that accumulate rapidly in the myocardium in proportion to myocardial blood flow including technetium labelled compounds (sestamibi and tetrofosmin) and thallium. As the tracer decays, photons are emitted and detected by a gamma camera. This is single photon emission computed tomography (SPECT). Images are acquired at rest and the maximal uptake in the myocardium is set as the background normal level. Stress images are acquired after either exercise or pharmacological stress (adenosine, dipyridamole, regadenoson or dobutamine). Perfusion defects detected after stress not present on the initial scan are consistent with stress induced ischaemia within that territory and defects detected on both rest and stress are fixed defects consistent with

scar/infarcted myocardium. It is also possible to measure LV function and volumes with these techniques.

Conventional ECG exercise testing relies on interpretation of the ECG for a positive result (difficult in LBBB etc) and has a sensitivity and specificity of ~70% and 80% respectively. MPI has a sensitivity and specificity of 90% and 70% respectively. All of the currently utilised pharmacological stressors have a similar ability to produce flow heterogeneity in the presence of (50-70%) stenosis of the coronary arteries, with sensitivities for adenosine and dipyramidole ~90% and dobutamine ~80%.

Even in patients with known CAD, for those with a negative MPI, the annual rate of cardiac death or non-fatal MI is <1%. For patients with a severely abnormal scan managed medically the risk of cardiac death compared to those who undergo revascularisation is significantly higher. There are several variables associated with a poorer prognosis

Perfusion	**Non-perfusion**
Multi-vessel disease	Poor exercise capacity
Large reversible defect	Angina at low workload
Scar > 14% of LV	ST depression >3mm with exercise
LV dilatation with stress	Exercise induced arrhythmias
RV uptake	Vasodilator stress induced ST depression >1mm
Resting LV dysfunction	Hypotensive blood pressure response
Pulmonary uptake of thallium	

CT calcium scoring

Coronary artery calcification is part of the disease process of atherosclerosis. It occurs in small amounts in early lesions although it is more predominant in later lesions. Presence of coronary artery calcification has been reported to increase the risk of death from CAD or MI by four-fold over a 3-5 year period whilst those without calcification have a low rate of death or MI. In addition, there is a rise in risk associated with incremental increases in calcification scores.

Further reading

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- NICE clinical guideline 95: Chest pain of recent onset
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the Society of Cardiovascular Computed Tomography. J Am Coll Cardiol. 2007 Jan 23;49(3):378-402.

Coronary revascularisation

CABG is offered to patients on

1. Symptomatic grounds
2. Prognostic grounds
3. Both symptomatic and prognostic grounds

There are a number of RCTs (some historical) which define the evidence base for coronary revascularisation

Medicine vs. surgery

Three large RCTs were performed of medical vs. surgical therapy in the 1970s. These each recruited and randomised ~700 patients and were the VA, ECSS and CASS study. The patient profile recruited to these studies has changed compared to the populations seen today and they consisted of mainly young (mid 50s), males with the majority having good ventricular function. In summary, these trials demonstrated a survival advantage in the first 5-7 years with convergence over time (possibly due to cross-over from medical to surgical arms with an intention-to-treat analysis and the relative lack of LIMA grafting compared to current practice) and a survival advantage for those with LMS, proximal LAD and triple vessel disease. A survival was also noted for the small subset with impaired ventricular function.

Subsequently Yusuf performed a meta-analysis of 7 trials (including the big 3) a total of 2649 patients were analysed. At 10 years 41% of patients in the medical group had crossed over to surgery. Patients randomised to the surgical arms had a significant survival benefit at 5, 7 and 10 years. Extension of survival was noted for the following sub-groups at 10 years

- Triple vessel and LMS
- Abnormal LV function
- Early +ve ETT

- CCS II/IV

PCI vs. surgery

The vast majority of patients recruited to these trials were single/double-vessel disease with preserved ventricular function. The majority of trials excluded patients with LMS stenosis and only ~5% of all potentially eligible patients were recruited. The only trial to report a survival advantage for CABG was the SoS trial and this unsurprising given the nature of the patients recruited. However, patients undergoing PCI were more likely to require repeat intervention and had higher recurrence of angina. In the subgroup analysis of the BARI trial even low-risk diabetic patients exhibited a survival advantage at 10 years with CABG vs. PCI as well as a significantly reduced need for re-intervention.

SYNTAX

The *Syn*ergy between PCI with *TAX*US and cardiac surgery (SYNTAX) Trial randomised 1800 patients with TVD/LMS to either DES (897) of CABG (903). Patients were reviewed by a heart team. If it was felt that full revascularisation could be achieved by either PCI or CABG then patients were eligible to be randomised. For patients whom it was felt were only suitable for a single intervention they were entered into the SYNTAX registry.

The primary endpoint at one year was non-inferiority between PCI and CABG and this was not met with CABG proving superior. As non-inferiority was not shown all subsequent sub-group analyses should be considered observational and hypothesis generating.

The SYNTAX score was devised to score complexity of coronary artery disease. On the basis of this 3 groups were identified low (0-22), intermediate (23-32) and high (>32) SYNTAX scores.

The three year follow-up data was presented in 2010. For all randomised patients at 3 years the rate of MI, cardiac death and repeat revascularisation was increased for PCI vs. CABG, no difference was detected for either CVA or all cause death (CVA was initially higher in the CABG group). The overall rate of MACCE was 28.0% vs. 20.2% for PCI vs. CABG at 3 years (mainly driven by the need for repeat revascularisation). There was no difference in MACCE for patients with a low score at 3 years and for those patients with intermediate or high scores, PCI had significantly higher rates of MACCE than CABG.

In pre-specified analyses for patients with TVD, MACCE, mortality and combined safety endpoint of death, MI and stroke were increased with PCI. For patients with LMS disease there were no significant differences in these outcomes with PCI

Therefore CABG remains the standard of care for patients with complex disease (TVD intermediate/high and LMS high SYNTAX scores).

Further reading

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Coronary Artery Bypass Grafting following an Acute Coronary Syndrome

Acute Coronary Syndrome (ACS) encompasses the following diagnoses:

- Non-ST segment elevation myocardial infarction (NSTEMI) – patients presenting with acute chest pain but without persistent ST-segment elevation. These patients may have persistent or transient ST-segment depression, T-wave inversion, flat T-waves, pseudo-normalization of T-waves or no ECG changes. These patients would be confirmed NSTEMI if troponin positive.
- Unstable angina – patients with chest pain without persistent electrocardiographic changes or an enzyme rise.
- ST segment elevation myocardial infarction (STEMI) – patients with typical acute chest pain and persistent (>20mins) ST-segment elevation with an associated troponin rise.

Patients diagnosed with NSTEMI comprise 25% of patients with an ACS in the Global Registry of Acute Coronary Events (GRACE). However, the incidence of NSTEMI has increased and now thought to be higher than STEMI. These patients form a heterogeneous population with a variable prognosis. It is therefore important that these patients are appropriately risk stratified and selected for appropriate treatment strategies (OMT vs. revascularisation).

Strategies for NSTEMI/UA

An interventional strategy over a conservative approach is preferred in managing NSTEMI patients. A meta-analysis in 2010 has shown that an early invasive strategy reduces ischaemic endpoints by reducing recurrent ischaemia and cardiovascular death and MI at up to five years of follow-up. Rates of cardiovascular death or non-fatal MI were 14.7% in the routine invasive (RI) group compared to 17.9% in the selective invasive (SI) (conservative) group. Similarly the rate of MI was lower in the

routine invasive group at 10% compared to 12.9% in the selective invasive group. The number of cardiovascular deaths and death from any cause in the RI group was 6.8% and 10.6% respectively, lower than in the SI group which was 8.1% and 11.7% respectively, but not statistically significant. Furthermore, patients at most risk potentially gain the most benefit from an aggressive interventional strategy, with an 11.1% absolute risk reduction in cardiovascular death or MI in the highest risk patients compared to 2 to 3.8% in the low and intermediate risk groups. The ESC guidelines recommend the GRACE risk score as a means of helping to decide whether to proceed with an early or late invasive strategy. The GRACE risk score is calculated using a number of clinical variables at the time of admission; age, systolic blood pressure, creatinine, class of congestive heart failure, the presence of cardiac arrest at the time of admission, ST segment deviation, or elevated cardiac enzymes. A patient with a GRACE risk score of > 140 is considered high risk and predicts an in-hospital mortality of >3%. The ESC/EACTS guidelines recommend an invasive strategy in patients with a GRACE score >140 or at least one high-risk criterion, recurrent symptoms or inducible ischaemia on stress testing and angiography should be performed in this group within 24hrs if possible.

An invasive strategy always starts with angiography and following this the most suitable strategy should be decided based on coronary anatomy, severity and distribution of disease as outlined in the AHA/ACC and ESC/EACTS guidelines. No RCT has yet addressed the selection of mode of intervention between PCI and CABG in patients with NSTE-ACS.

The Veterans Affairs Non-Q-Wave Infarction Strategies in Hospital (VANQWISH) trial was a multi-centre randomised control trial that enrolled patients with an acute non-Q-wave MI to compare an invasive with a conservative treatment strategy. Eligible patients had to have an evolving acute MI, with a level of Creatine Kinase MB (CK-MB) > 1.5 times the upper limit of normal for each hospital. VANQWISH randomly

assigned 462 patients to an invasive strategy versus 458 to a conservative strategy. Of the 204 patients that underwent myocardial revascularization in the invasive group, 95 patients had CABG surgery. Study patients with ACS who had early surgery (median of 8 days for overall revascularization) had an in-hospital mortality of 12% compared to 3% in those who had delayed revascularization (median of 26 days for overall revascularization). Up to 40% of patients in this study were > 65 years, were high risk and all patients recruited including surgical patients had a significant rise in myocardial enzymes indicating a larger infarct.

Subsequent trials comparing an invasive and a conservative strategy (FRISC II, TACTICS-TIMI 18, RITA-3) allocated patients at the discretion of the investigator into either PCI or CABG within the invasive arm. In the FRISC II trial, within the invasive sub-group, CABG had a mortality, myocardial infarction and rate of revascularisation of 3%, 9% and 2% respectively at one year in comparison to PCI which demonstrated rates of 1%, 15% and 14% respectively; a considerably higher rate of MI and revascularization in the PCI group. These results were consistent with the TACTICS-TIMI-18 trial where the rate of re-vascularisation was 7% following CABG compared to 15% with PCI at 1 year. The long term outcomes are difficult to interpret as all trials do not separate the outcome of CABG, but group them into the invasive arm. However, similar trends in the rate of revascularization are seen in trials comparing CABG to stents with a subset analysis of ACS. The ARTS trial reported a revascularization rate of 3.6% compared to 16.9% with PCI.

In TACTICS-TIMI-18 the benefit of an early invasive strategy was greater in those patients with a higher cardiac enzyme release (Troponin rise > 0.01), with a significant benefit in this group of patients in comparison to the conservative therapy arm. Similar trends were observed in the FRISC-II trial. This benefit was seen with rise in troponin; a very sensitive marker of myocardial injury. Hence it is difficult to compare this, against the outcomes observed in the VANQWISH trial where

everyone had a marked rise in CK-MB. In these trials the proportion of patients receiving CABG following an ACS was 26%. This is only achievable in centres where the resources allow for an urgent CABG and actual numbers may be lower in real life. The Prospective Registry of Acute Ischaemic Syndromes in the UK (PRAIS-UK), which determines the clinical outcomes, risk stratification and practice patterns of unstable angina and myocardial infarction has shown that at six months, in patients with Non-ST elevation MI, only 27% of patients undergo coronary angiography, 8% receive PCI and 7% receive CABG. It is vital that patients with an ACS, particularly those with NSTEMI whose treatment pathway is not straightforward is discussed at an MDT, risk stratified and selected for the most appropriate invasive strategy as part of the 'heart team' approach.

The CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of the American College of Cardiology/American Heart Association Guidelines) and ACTION registry-GWTG (Acute Coronary Treatment and Intervention Outcomes Network Registry-Get with the Guidelines) both collect data on unstable angina and NSTEMI patients in the United States from 2001. A study looking at these registries, showed that 11-13% of in-patients undergo CABG for NSTEMI of which, 70% of these patients undergo late CABG as an in-patient, a trend that has not changed from 2002 to 2008. In this study early CABG was defined as CABG occurring ≤ 48hrs and late as occurring ≥ 48hrs. Looking at just the ACTION registry, this study showed that although the group undergoing late surgery had a high risk profile, there was no difference in in-hospital mortality (3.8%) or in composite outcome of death, myocardial infarction, congestive heart failure or cardiogenic shock (12.4%) compared to patients that underwent early CABG; mortality and composite outcome of 3.6% and 12.6% respectively.

The timing of intervention is different for PCI and for CABG, where the benefit from CABG is greatest after several days of medical stabilization. However the ideal

timing of surgical revascularization following NSTEMI is conflicting. Delaying surgery 3-5 days after a non-transmural MI and 5-7 days after a transmural MI showed a mortality of 2.6% and 0% respectively, similar to outcomes of patients undergoing elective CABG. Hence this study advocates a waiting strategy to allow the myocardium to recover.

The timing of CABG may be determined by the clinical urgency based on the patients' ongoing clinical condition or that of medications used in the treatment of NSTEMI. The period of use of clopidogrel and low molecular weight heparin both would influence the timing of surgery. Clopidogrel is usually stopped for five days prior to CABG surgery to allow for the anti-platelets effects of clopidogrel to wean and reduce the risk of peri-operative bleeding.

Strategies for STEMI

The mainstay of management of STEMI is early reperfusion with either thrombolysis or primary angioplasty. Primary PCI has proven to be more effective than fibrinolytic treatment for STEMI and the trend is to provide a primary PCI service as 1st line therapy considering the time windows and expertise available. Outcomes at five years comparing PCI to thrombolysis have shown a favourable reduction in mortality and re-infarction, 13% versus 24% and 6% versus 22% for PCI vs. thrombolysis respectively. Primary PCI is ideally performed within the first two hours from the onset of symptoms. If PCI cannot be delivered < 2 hrs or <90 minutes patients <75 years patients should receive immediate fibrinolysis and then transferred to a PCI centre, where angiography and PCI can be performed between 3-24 hrs. Adopting these criteria has resulted in decrease in in-hospital mortality from 16% to 9.5%.

In cases were PCI fails or coronary anatomy is not suitable for PCI, surgical revascularization may be considered. Emergency CABG may be potentially indicated during an evolving STEMI, when there is a large area of myocardium at risk and surgical revascularization can be complete before this area becomes necrotic (in the

initial 3-5 hours). Considering the times required for surgical set-up this is a rare occurrence. Urgent CABG is indicated in patients with multi vessel disease following an STEMI, where the culprit lesion has already been addressed by primary PCI or urgent post-fibrinolysis. Surgical mortality is inversely proportional to time elapsed since STEMI. Therefore in patients without persistent pain or haemodynamic compromise a period of 3-7 days after STEMI is suitable before surgical intervention.

Cardiogenic shock

Cardiogenic shock may occur as a complication of acute myocardial infarction. Despite advances in medical treatment, early invasive therapy and CABG, overall mortality rates are as high as 60% and half of these patients die within 48 hours once cardiogenic shock is established.

The SHOCK trial looking at early revascularization in acute myocardial infarction complicated by cardiogenic shock randomised 152 patients to revascularisation and 150 to medical treatment. Revascularization was performed <6hrs form randomisation. The SHOCK trial showed a beneficial effect from early revascularization for patients in cardiogenic shock. This early revascularization strategy in the SHOCK trial resulted in 132 lives saved at 1 year per 1000 treated. Most patients in the SHOCK trial had severe multivessel disease and CABG surgery was performed in 40% of patients in the early revascularization strategy. The majority (79%) of patients with cardiogenic shock developed left ventricular failure. Although 30-day mortality showed no difference between the revascularization and medical therapy groups (47% versus 56%), 6 month mortality was significantly in favour of early revascularization (50% versus 63%). Immediate stabilization and early revascularization is indicated when cardiogenic shock develops secondary to pump failure and complicates acute MI.

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Choice of conduit for CABG

There are a number of conduits available for performing CABG. The most commonly used are the internal mammary arteries (left and right), radial artery and long saphenous vein. Other less commonly used conduits include the short saphenous and vein and arterial conduits right gastroepiploic and the inferior epigastric artery.

Long saphenous vein

The LSV has an internal elastic lamina with a smooth muscle containing media. Due to the relative wall thickness, once the vasa vasorum are deprived of inflow, transluminal provision of nutrients is not possible and this leads to replacement of the smooth muscle with fibrous tissue. Whilst the endothelium is able to produce NO, its ability to do so is less than that of the arteries and this is further reduced after grafting to the arterial system. Historical studies have shown that SVG patency may be as low as 50% at 10 years and could potentially be lower (due to the fact that only survivors are able to be studied and death may be secondary to graft occlusion). However, it should be remembered that with current modern day pharmacological secondary prevention and improved conduit handling, some randomised controlled trials are demonstrating better than expected SVG patency rates.

Radial artery

The brachial artery terminates at the antecubital fossa as the radial and ulnar arteries. It lies inferior to fascia in the distal third, brachioradialis in the middle third and fascia in the proximal third. Two nerves are related to it and these may be damaged on harvest. These are the lateral antebrachial cutaneous nerve in the upper forearm (supplies sensation to dorsoradial aspect of the forearm) and the superficial radial nerve, which lies on the ulnar side of the radial artery and gives sensation to the thenar eminence, first metacarpal and proximal phalanx of the thumb. It is a muscular artery and can be prone to intimal hyperplasia, atheroma and

calcification. Reports have suggested that at the time of harvest, only 0.7% of radial arteries are histologically normal with 94% exhibiting intimal hyperplasia, 5.3% being atherosclerotic and up to 13.3% may have medial calcification. Before harvest of the RA it is imperative to check and document the collateral circulation of the hand. This is done using an Allen's test in which the radial and ulnar arteries are occluded at the wrist and exsanguinating the hand. The ulnar artery is released and the capillary refill of the palm is documented. Various cut-offs have been described in the literature with up to 12 seconds being deemed acceptable. The radial artery is sensitive to vasodilators and topical and intraluminal papaverine may be administered in conjunction with low dose systemic milrinone.

Internal mammary arteries

Within the muscular media of the IMA are a number of elastic lamellae. The subintima lies on an elastic lamina, with a few small fenestrations. These smaller fenestrations potentially allow for less ingress of smooth muscles cells to the subintima, thereby inhibiting plaque formation. In addition, the reduction in smooth muscle mass means that the artery is less prone to spasm on exposure to vasospastic drugs.

LEFT – Arises from the left SCA opposite the vertebral artery. It is an elastic artery until its distal portion where it becomes more muscular. At the level of the 6th ICS it divides into the musculophrenic and superior epigastric arteries. It is less prone to spasm and may be dilated with both topical and intraluminal papaverine.

RIGHT – Is similar in anatomical and functional characteristics to the left.

Reporting of patency rates in trials

It is important to have an understanding of how patency rates are reported within trials so that data can be interpreted in an appropriate manner. Grafts may be defined as totally occluded, greater than 75% or50% stenosis or string sign. In

addition, the Fitzgibbon classification is used, which classifies graft patency as A – patent or stenosis < 50%, B stenosis > 50% and O – occluded. Frequently these classifications are merged as an endpoint. Physiological classification can also be used such as TIMI flow) 0 – no flow, 1 – minimal dye, 2 – partial dye and 3 – normal. The gold standard for reporting of graft patency remains coronary angiography. CT angiography is an alternative method of assessing patency.

Kaplan Meier assessment of graft patency assumes what is termed as “right censoring”. It cannot be known at what point a graft became occluded between assessment times and the KM method assumes that this happens at the time of last analysis i.e. results with KM analysis may falsely elevate graft patency times.

Symptom directed angiography is performed in patients with evidence of ischaemia and in these trials, graft failure rates may be up to twice as high as in studies with planned angiography.

Internal mammary artery – Cleveland Clinic studies and ART

The left internal mammary artery to the LAD is the gold standard for conduit and target vessels. Patency rates at 1, 10 and15 years are reported to be 95, 95 and 88% respectively. Furthermore, the data from the Cleveland Clinic published in 1986 in a an observational study demonstrated significant survival for patients receiving LIMA (vs. SVGs only) in particular for those patients with multivessel disease and this survival benefit started to become apparent after 5-6 years. Patients receiving a LIMA to LAD also have increased freedom from angina, myocardial infarction and re-intervention.

The Cleveland Clinic in non-randomised retrospective analyses utilising propensity score matching and matched pairs analysis have suggested that there may be a benefit with the use of BIMA vs. SIMA with improved overall and re-operation free survival. The ART trials which has randomised 3102 patients to either BIMA or SIMA

has reported early results with no difference in 30-day mortality 1.2% each and one-year mortality 2.3vs. 2.5% but with an increase incidence of sternal wound reconstruction 0.6 vs. 1.9% for SIMA vs. BIMA respectively. This study is powered for the 10-year survival results.

RAPCO

RAPCO is a randomised controlled trials with two distinct populations group 1- <70 years randomised to free RIMA or radial group 2 - >70 years randomised to RA or SVG. LIMA to LAD was the primary graft and the "study graft" was placed to the next biggest target with a stenosis > 70%. Interim analyses of protocol directed angiograms performed at random intervals in 57% of the study population document a 5-year patency rate for group 1 89.8% vs. 83.2%, RA vs. fRIMA and 90% vs.87%, RA vs. SV. The number of angiograms to be performed was weighted to the second half of the study period and 10-year graft patency rates will be reported. Grafts were defined as failed if occluded, string sign or >80% stenosis.

RAPS

The RAPS study randomised patients with high grade right/circumflex stenoses (>70%) to receive either a RA or SVG to that vessel. The LIMA was grafted to the LAD. Failure of a graft was classified as TIMI 0 flow. One year data demonstrated a significant difference, 8.2% vs. a 13.6% occlusion rate for the RA vs. SVG (TIMI 0). For functional graft occlusion TIMI 0-2, there was no difference in patency rates 12.3% vs. 14.3% (RA vs. SVG). Recent five year data shows a significant increase in both TIMI 0 graft occlusion 8.9% vs. 17.8%, functional graft occlusion 12.0% vs. 18.8% and stenosis > 25% or TIMI 0, 21.9 vs. 33.8% for RA vs. SVG respectively.

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Off-Pump Coronary Artery Bypass Surgery

The interest in off-pump coronary artery bypass (OPCAB) surgery has largely been driven by the increased awareness of the deleterious effects of cardiopulmonary bypass (CPB) and aortic manipulation. OPCAB utilization appears to have reached a plateau in recent years. In 2008 OPCAB accounted for approximately 22% of coronary artery bypass cases in the US and 17% in the UK, with the remainder being performed with the use of CPB (ONCAB). For many surgeons, the lack of compelling evidence in randomized trials supporting OPCAB over ONCAB, combined with concerns about the quality of the anastomoses and the effect of this on long-term survival, has been an impediment to implementing this strategy in routine practice. Undoubtedly, OPCAB is more technically challenging, particularly for the lateral wall, and consequently some surgeons prefer to implement this technique only in low-risk patients or in patients requiring only one or two-vessel bypass grafting, the very patient group who are likely to benefit the least from it.

Meta-analyses

There have been a number of meta-analyses performed investigating ONCAB vs. OPCAB.

Takagi et al focused on randomized controlled trials that published follow-up data beyond one year. They identified 11 results of 12 randomised trials of 4,326 patients. Pooled analysis demonstrated a significant increase in mid-term all cause mortality with OPCAB vs. ONCAB (RR 1.37; 95% CI 1.043-1.808). The authors confirmed that after exclusion of the ROOBY trial (which is critiqued in detail below), that no significant difference between groups was observed.

Wijeysundera et al identified 37 RCTs (n=3449) and 22 risk-adjusted observational studies (n=293,617). 10 RCTs reported ≥1 death within 30 days of surgery, mortality

1.7%, with no difference between groups. 14 observational studies suggested that OPCAB was associated with reduced 30-day mortality (OR 0.72, 95% CI 0.66-0.78), CVA (OR 0.62, 95% CI 0.55-0.69), MI (OR 0.66, 95% CI 0.50-0.88), and AF (OR 0.78, 95% CI 0.74-0.82), and it was suggested that these discrepancies between RCTs and observational studies may be due to differing patient-selection and study methodology. 24 RCTS reported a mean number of bypass grafts. The mean number was 0.19 lower in the OPCAB group, p<0.001.

Randomized trials

ROOBY

The ROOBY study represents the largest RCT of OPCAB vs. ONCAB to date. A total of 9663 patients were assessed for eligibility following which 2203 patients underwent 1:1 randomization to receive either OPCAB or ONCAB. Participating surgeons had to have performed at least 20 OPCAB cases. Pre-study OPCAB experience was a median of 50 cases. 15.2% of patients randomized required urgent surgery. 66.1% were documented as having triple vessel disease. The primary short-term end-point was a composite of death or major complications (reoperation, new mechanical support, cardiac arrest, coma, CVA, renal failure requiring dialysis within 30 days) and the primary long-term end-point was a composite of death from any cause, repeat revascularization or non-fatal MI within 1 year.

No significant difference in the rate of primary short term composite end-point was demonstrated between OPCAB and ONCAB, 7.0% *vs.* 5.6%. 30 day mortality was low in both groups, 1.6% *vs.* 1.2%. The primary long term composite outcome was higher in OPCAB than ONCAB, 9.9% vs. 7.4%, p=0.04. There were no significant differences in any of the individual components of the one year end point. Kaplan-Meier analysis for any cause mortality showed no significant difference between groups.

In 73.1% of the OPCAB group and 75.6% of the ONCAB the number of grafts planned was equal to the number of grafts performed. However, in 17.8% of OPCAB versus 11.1% the number planned was greater than the number performed.

1335 patients (62.7%) had a 12 month angiogram and graft patency was evaluated by Fitz Gibbon criteria. Significantly lower graft patency was demonstrated in the OPCAB versus ONCAB, 82.6% vs. 87.8%, $p<0.001$.

This trial was widely criticized as being fatally flawed for 2 reasons.

1. The surgical teams were relatively inexperienced at OPCAB as demonstrated by a five-fold increase in the expected intra-operative conversion from OPCAB to ONCAB (12.4%) with a median experience of only 50 OPCAB cases per surgeon (this being within the learning curve of approx 200 cases).
2. It enrolled low-risk male patients in whom avoidance of CPB was unlikely to greatly improve the expected excellent outcome. It should be noted that there was a 3.6% conversion rate from ONCAB to OPCAB, and that if the group was re-analysed excluding patients who did not receive their randomized intervention, then no significant difference in the primary late end point was observed.

SMART Trial

Puskas first reported the results of the SMART Trial in 2004. Of 297 non-consecutive patients referred to a single surgeon, 200 were randomised to OPCAB vs. ONCAB (197 included). There were 4 crossovers, 3 from ONCAB to OPCAB and 1 from OPCAB to ONCAB. 93.4% of patients underwent angiography prior to hospital discharge, both FitzGibbon and TIMI scores were not different between groups. A total of 1.6% of grafts were occluded at catheter prior to discharge. Of the 189 patients alive at one year, 81% underwent angiography. A total of 93.6% of grafts

were patent for OPCAB compared with 95.8% of grafts for ONCAB. No significant differences were detected between groups in the incidence of death, MI, stroke, recurrent angina, readmission from cardiac or non-cardiac events. Patients undergoing OPCAB had reduced release of biochemical markers of myocardial injury within the first 48 hours. The number of grafts per patient (3.39 OPCAB versus 3.40 CPB) and the index of completeness of revascularization (1.01 OPCAB versus 1.00 CPB) were similar between groups.

In mid-term follow-up of this study there were 140 survivors (71%) at a mean of 7.5 years of follow-up. There were 26 deaths in the OPCAB and 31 deaths in the ONCAB groups. Of the 140, a total of 87 (44% of entire study – 43 OPCAB vs. 44 ONCAB) volunteered to return for assessment of graft patency (CT angiography and myocardial ischaemia (CPET). Although only small numbers of grafts were re-studied, there was no difference in overall graft patency between groups 78.4% vs. 84.4%, OPCAB vs. ONCAB

In this study 200 unselected patients were randomised to OPCAB vs. ONCAB. Again similar in-hospital and 30-day outcomes were reported with a similar number of grafts (OPCAB 3.39 *vs.* ONCAB 3.40) and completeness of revascularization index (OPCAB 1.0 *vs.* ONCAB 1.01). They did however demonstrate a reduced length of stay, transfusion requirements and myocardial injury with OPCAB.

Observational studies

Puskas' group examined the benefit in high risk patients undergoing OPCAB in 14,766 patients comparing STS Predicted Risk of Mortality (PROM) to observed mortality. It demonstrated that patients in highest risk quartile had significant reduction in mortality with OPCAB compared with ONCAB (3.2% vs. 6.7%, $p<0.0001$; OR 0.45, 95% CI 0.33-0.63). Logistic regression analysis confirmed a significant

interaction between surgery type and PROM (p=0.005) suggesting that the benefits of OPCAB are greater in patients with a higher STS-Prom. This benefit was seen to be greatest for patients with PROM values above 2.5%, where mortality curves began to diverge sharply.

Hannan's New York group analysed 49,830 patients from the New York State registry between 2001 and 2004. Outcomes were risk-adjusted analysis using a Cox proportional hazard models and propensity analysis. OPCAB had lower 30-day mortality (adjusted OR 0.81, 95% CI 0.68-0.97, p=0.0022) and CVA (OR 0.70, 95% CI 0.57-0.86), however, no difference in 3-year mortality (hazard ratio 1.08, 95% CI 0.96-1.22) but importantly a higher rate of subsequent revascularization (hazard ratio 1.55, 95% CI 1.33-1.80) in OPCAB.

Further reading

Two comprehensive reviews on the subject have been published by Taggart's and Puskas's groups

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Treatment of Chronic Left Ventricular Failure

John Dark

Whilst coronary disease continues to decline, there is an epidemic of heart failure. The progress of medical management has been spectacular; a series of randomized trials have demonstrated the advantages, at least in terms of survival, of ACE inhibitors and receptor blockers, B blockers, aldosterone antagonists and re-synchronisation. There have been failures – we remember the oral phosphodiesterase inhibitors that allowed the patient to “burn brightly but briefly”

Does the surgeon have a role here? Many patients with “heart failure” benefit from our attentions – correction of aortic stenosis, mitral regurgitation. But what do we have to offer when the problem not mechanical, but myocardial? A number of recent studies have given us new and not entirely welcome information.

The STICH trial was a valiant attempt to establish surgical superiority over medical management in patients with coronary disease and impaired LV function. There were two strata, one addressing just CABG, the other the role of “ventricular restoration” when there was LV enlargement and anterior akinesia. There have been numerous single institution, retrospective studies of this approach, all suggesting improved survival. In this study with very large number of patients randomised[1] there was a measurable reduction in LV size but absolutely no survival advantage or reduction in re-admissions after resecting the scarred area of the ventricle. An accompanying editorial quite reasonably suggests that with the optimal medical therapy enjoyed by patients in this trial, there was no added advantage to surgery.

Similarly, and again in contrast with uncontrolled single centre studies, there was no advantage to CABG in this group of patients with multi-vessel disease and an EF <35%[2]. Even more interestingly, although patients with viable myocardium overall did better than those with just scar, surgery did not increase this advantage[3]. An

accepted truth from a previous age, that patients with surviving but impaired myocardium downstream from an obstructed coronary *must* do better with revascularization, must now be held open to doubt. There are inevitable criticisms of the trials: less than half had viability studies (left to the discretion of the clinicians). With 96 centres and a 1000 patients, average recruitment was only 10 per centre over 3 years! But to operate on the ventricle in addition to revascularization is now difficult to justify for many patients

What about the mitral valve in LV dysfunction? Patients with bad MR do badly with medical therapy alone, particularly if there is a reduced EF. The rationale for eliminating MR with a reduction annuloplasty, reducing the volume load on the ventricle, makes complete sense. There are no randomized studies, but many authors described low mortality and reduction in LV size. However, Wu and colleagues from Michigan, incidentally the same centre as Steve Bolling, one of the most articulate proponents of reduction annuloplasty, compared medical and surgical cohorts[4]. Whilst surgery improved symptoms in some, there was absolutely no difference in survival! This study came from 1995 -2002, when surgical mortality was already low, but medical therapy has probably made significant advances since then. This albeit retrospective comparison is probably still valid 6 years later.

Nevertheless, this study and others have shown both strikingly low operative mortality (1.6% in the Acorn Trial, for instance) and symptomatic improvement. So the operation is worthwhile as long as the patient understands the benefits. It is of course essential to achieve durable valve competency, and some variations on simple annuloplasty (following which up to 35% of patients have late, recurrent MR) , such as the String and Ring approach[5] warrant consideration

This technique goes someway to addressing issue that the pathology is in the ventricle, not the valve! A very perceptive piece of work from Robert Dion, one of the more thoughtful mitral surgeons, sheds more light on this problem. In a large series

with significant LV impairment, LV size was a major determinant of survival and improvement[6]. There was a cut-off at an LV end-Diastolic Diameter of 65mm, with larger LV's doing badly whilst those with smaller hearts had a strikingly better outcome.

If the large, failing ventricle can't be improved by indirect approaches such as CABG or valve repair, what about direct approaches?

Cardiomyoplasty, wrapping the heart in paced skeletal muscle, although much refined, has not proven successful. Indeed, its greatest benefit was to show that ventricular constraint – achieved by the muscle wrap even if not paced, was potentially useful, leading to the Acorn device. The Batista operation went the same way, with no proven benefit.

Stem cells might be a means of reversing the inevitable decline once the heart is scarred or dilating. In animal models the results are spectacular, but clinical trials, in various settings and with a range of cells, have not reproduced what is found in the lab[7]. A number of trials have shown a small advantage, often persisting after the new cells can no longer be found. The benefits may be paracrine rather than the direct benefit of contracting elements

Cardiac transplantation and left ventricular devices are the most radical but possibly the most successful options left for the surgeon to help the very worst affected patients. Transplantation is the gold standard, returning the recipient close to a normal lifestyle. But it is only available for a tiny handful of patients – c100 per year in the UK – and the number is falling[8].

By contrast, mechanical assist devices have an applicability only limited by money. There has been a continuing evolution, with striking technical advances and constantly improving bio-compatibility. Results with the totally implantable centrifugal devices, still used principally as a bridge to transplant, are now comparable with

transplant itsel – survival rates of 85% at one year are reported in major registries[9], and may be even better at major institutions. Modern devices are now comparable with transplant and will become the definitive therapy for many with end-stage heart failure[10]

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Surgery for ischaemic heart failure

Surgical ventricular restoration

Surgical ventricular restoration (SVR) is a technique to restore LV geometry and volume, thereby reducing LV wall stress. Following infarction, LV remodelling leads to LV dilatation and increase in LV wall tension. Increased wall stress leads to increased myocardial oxygen consumption, reduced subendocardial blood flow and reduced systolic shortening. The procedure of SVR commonly includes three procedures CABG, mitral repair (when required) and restoration of LV size and geometry.

RESTORE group

The RESTORE group examined the result of 1198 patients undergoing SVR. Patients had to have prior anterior infarction with significant LV dilatation (LVESVi >60ml.m^{-2}) and an asynergic (non-contractile) LV circumference > 35%. Just under 70% of patients were in NYHA III/IV. Akinesia was present in 66% and dyskinesia in 34%. Akinetic ventricles had larger LVESVi. Concomitant procedures performed included CABG (95%), MV repair (22%) and MV replacement (1%). Patients undergoing concomitant MV surgery had reduced EF and increased LVESVi compared to those not undergoing MV surgery. The 30-day operative mortality was 5.3% (8.7% for those undergoing MV surgery). Post-operative LVEF was improved and LVESVi was reduced from 80.4 to 56.6ml.m-2. Overall five year survival was 68.6%. Survival was better for those with dyskinetic rather than akinetic segments. Risk factors for death after surgery included LVESVi> 80ml.m-2, LVEF< 30%, age> 75 and advanced NYHA class. When compared with the available literature on survival of patients with ischaemic heart failure treated by OMT, lone CABG or MV surgery, SVR (in this non-randomised study) has superior outcomes.

STICH Trial – hypothesis 2

The Surgical Treatment for Ischaemic Heart Failure (STICH trial) hypothesis 2 randomised 1000 patients with an LVEF < 35% and revascularisable coronary artery disease and anterior LV dysfunction to CABG alone or SVR + CABG. Both CABG and SVR led to reductions in LVESVi 6% and 19% respectively. Both therapies led to improvements in cardiac symptoms and exercise tolerance. There was however, no improvement in the primary outcome measure (composite of death and hospitalisation from cardiac causes). The results of the STICH trial hypothesis 2 have questioned the validity of SVR but the trial has been criticised by a number of proponents of the technique for the following reasons

1. A proportion of patients had not had prior MI and therefore were unlikely to have LV wall scar
2. LV volume measures were planned to be by CMR, however, only 38% of patients had LV volumes measured
3. SVR is only performed if the LVESVi > 60ml.m-2, however, volume measurements were not reported.
4. A 30% reduction in LVESVi is believed by many to be an adequate reduction in LVESVi at four months, but in those few patients who had LVESVi measured, the reduction was only 19%, calling into question the operative technique.

Thus, the role of SVR still needs to be further defined.

CABG for patients with left ventricular dysfunction

Based on analysis of data from the CASS registry examining the outcomes of patients with an LVEF < 35%, demonstrated improved outcomes for CABG compared with OMT. The STICH hypothesis 1 set out to investigate the role of

CABG in patients with an LVEF < 35% compared with modern day OMT with a primary outcome measure of death from any cause.

STICH Trial - hypothesis 1

In this arm of the STICH trial 1212 patients were randomised to either CABG or OMT. The majority of the patients (95%) had CCS class ≤ II. There was no difference in rate of any cause death but a significant benefit was seen in the CABG group for a composite endpoint of any cause death or hospitalisation for cardiovascular causes. However, 17% of patients in the OMT group underwent CABG at a median of 142 days (mainly due to symptoms). In as-treated and per-protocol analyses, CABG was demonstrated to be superior to OMT with respect to the primary outcome measure.

In a third publication from the STICH investigators, a non-random sub-group of patients (49%) undergoing viability studies were followed to ascertain the survival benefit associated with revascularisation in the setting of viable myocardium. The investigators demonstrated greater survival benefits for those patients with viable vs. non-viable myocardium, however, no significant interaction between viability status and treatment assignment with respect to mortality was seen.

The authors noted several important limitations including, the small, non-random sample of patients undergoing viability testing, only 19% of the patients with viability testing had non-viable myocardium, differing techniques to assess viability and the low death rate of patients with viable myocardium assigned to current day OMT compared with historical studies.

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Mixed Scenarios in Coronary Artery Bypass Grafting

GJ Murphy

Aortic Stenosis

Patients undergoing CABG who have severe or moderate aortic stenosis (AS) should undergo Aortic Valve Replacement (AVR) at the time of revascularization. Decision making is less clear in patients who have CAD that requires CABG when these patients have mild AS. The natural history of mild AS is variable although some patients manifest a relatively rapid progression of AS that is defined as a decrease in valve area of up to 0.3 cm^2 per year and an increase in pressure gradient of up to 15 to 19 mm Hg per

year. Patients with mild AS who exhibit recent progression or those with leaflet calcification or restricted leaflet motion warrant replacement if they have an expected life expectancy of >5 years.

The ACC/AHA Guidelines for the management of aortic stenosis in patients undergoing coronary artery bypass grafting are as follows:

Section 10.4 Aortic Valve Replacement in Patients Undergoing Coronary Artery Bypass Surgery

CLASS I

1. AVR is indicated in patients undergoing CABG who have severe AS (**area less than 1.0 cm2, mean gradient greater than 40 mm Hg, or jet velocity greater than 4.0 m per second**).

(Level of Evidence: C)

CLASS IIa

1. AVR is reasonable in patients undergoing CABG who have moderate

AS (**area 1.0 to 1.5 cm2, mean gradient 25 to 40 mm Hg, or jet velocity 3.0 to 4.0 m per second**). (Level of Evidence: B)

CLASS IIb

1. AVR may be considered in patients undergoing CABG who have mild

AS (**area >1.5 cm2, mean gradient less than 25 mm Hg or jet velocity less than 3.0 m per second**) when there is evidence, such as moderate-severe

valve calcification, that progression may be rapid. (Level of Evidence:

C)

Carotid Disease

Carotid disease increases the risk of perioperative stroke. This may be reduced by either staged or synchronous carotid revascularisation where the risk of stroke is high (in patient with severe carotid stenosis; 50–99% in men and 70–99% in women *and* the patient has a had a previous TIA or non-disabling stroke) and the risk of carotid revascularisation is low; it is performed by teams with death-stroke rates <6%. Carotid revascularisation may be considered for asymptomatic male patients if there is severe bilateral disease or severe disease with contralateral occlusion and the risk of death-stroke is <3% and life expectancy is > 5years. There is evidence of benefit in asymptomatic female patients. If there is doubt as to benefit each case should be considered by a multidisciplinary tam.

Carotid Endarterectomy (CEA) remains the procedure of choice for carotid revascularisation with proven benefits over Carotid Artery Stenting (CAS) in meta-analyses of RCTs but selection of CEA versus CAS depends on multidisciplinary assessment and local expertise. There is no clear benefit for staged over synchronous carotid revascularisation.

The EACTS/ECC Guidelines for the Management of Carotid Disease are as Follows:

CLASS I

1. CEA or CAS should be performed only by teams with demonstrated 30 day combined death-stroke rate: <3% in patients without previous neurological symptoms <6% in patients with previous neurological symptoms. (Level of Evidence A)

2. The indication for carotid revascularization should be individualized after discussion by a multidisciplinary team including a neurologist. (Level of Evidence: C)

3. The timing of the procedures (synchronous or staged) should be dictated by local expertise and clinical presentation targeting the most symptomatic territory first. (Level of Evidence:C)

In patients with previous TIA/non-disabling stroke, carotid revascularization:

CLASS I

Is recommended in 70–99% carotid stenosis. (Level of Evidence: C)

CLASS IIb

May be considered in 50–69% carotid stenosis in men with symptoms <6 months. (Level of Evidence: C)

Class III

CEA is not recommended if carotid stenosis <50% in men and <70% in

women. (Level of Evidence: C)

In patients with no previous TIA/stroke, carotid revascularization:

CLASS IIb

May be considered in men with bilateral 70–99% carotid stenosis or 70–99% carotid stenosis + contralateral occlusion. (Level of Evidence: C)

CLASS III

Is not recommended in women or patients with a life expectancy <5

years. (Level of Evidence: C)

Atrial Fibrillation

Atrial fibrillation (AF) in patients undergoing cardiac surgery is associated with poor short and long-term outcomes. In patients undergoing mitral valve surgery conversion to sinus rhythm improves outcomes and survival. Ablation, whether surgical or percutaneous is more effective at converting patients to sinus rhythm and is associated with better outcomes, although there is a documented failure rate for every technique. Factors reducing success of the procedure include large LA size, advanced age, longer duration of AF (permanent vs. paroxysmal AF), hypertension, and sleep apnoea

It is reasonable to suggest that surgical treatment of AF at the time of surgery will improve clinical outcomes. Whilst there is observational data to support this there is no data from randomised trials. There is no evidence to support Ganglionated plexus ablation and vagal denervation beyond the benefits gained from pulmonary vein isolation and exclusion of the left atrial appendage. Post surgical ablation results are improved if antiarrhythmic and anticoagulation drugs are continued for at least 3 months with subsequent withdrawal based on clinical, ECG, and echocardiographic assessment.

There are several established surgical modalities for the treatment of AF.

The MAZE Procedure: This is the longest established surgical treatment for AF however the results of surgery are operator dependent. Freedom from AF is 75–95% up to 15 years after the procedure.

Radiofrequency: sinus rhythm is restored in 85% of cases at 1 year and 52% at 5 years. The duration of AF and the LA size are predictive of recurrence.

Cryoablation: induces transmural lesions by freezing atrial tissue. Freedom from AF is 87% at 1 year.

High-intensity focused ultrasound: results in deep heating, coagulation necrosis, and conduction block. Freedom from AF or flutter is 86% at 18 months.

The European Society of Cardiology Guidelines for the management of atrial Fibrillation in patients referred for surgical revascularisation are as follows:

CLASS IIA

1. Surgical ablation of AF should be considered in patients with symptomatic AF undergoing cardiac surgery. (Level of Evidence: A)

CLASS IIB

Surgical ablation of AF may be performed in patients with asymptomatic

1. AF undergoing cardiac surgery if feasible with minimal risk. (Level of Evidence: C)

2. Minimally invasive surgical ablation of AF without concomitant cardiac

surgery is feasible and may be performed in patients with symptomatic

AF after failure of catheter ablation. (Level of Evidence: C)

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Functional anatomy of the aortic valve

Ian Wilson

Location of the aortic root

Although forming the outlet from the left ventricle, when viewed in the context of the heart as it lies within the chest, the aortic root is positioned to the right and posterior relative to the subpulmonary infundibulum. The subpulmonary infundibulum is a complete muscular funnel which supports in uniform fashion the leaflets of the pulmonary valve. In contrast, the leaflets of the aortic valve are attached only in part to the muscular walls of the left ventricle. This is because the aortic and mitral valvar orifices are fitted alongside each other within the circular short axis profile of the left ventricle, as compared to the tricuspid and pulmonary valves which occupy opposite ends of the banana shaped right ventricle When the posterior margins of the aortic root are examined, then the valvar leaflets are seen to be wedged between the orifices of the two atrioventricular valves. Sections in long axis of the left ventricle then reveal the full extent of the root, which is from the proximal attachment of the valvar leaflets within the left ventricle to their distal attachments at the junction between the sinus and tubular parts of the aorta.

The aortic root

Forming the outflow tract from the left ventricle, the aortic root functions as the supporting structure for the aortic valve. As such, it forms a bridge between the left ventricle and the ascending aorta. The anatomic boundary between the left ventricle and the aorta, however, is found at the point where the ventricular structures change to the fibroelastic wall of the arterial trunk. This locus is not coincident with the attachment of the leaflets of the aortic valve. The leaflets are attached within a cylinder extending to the sinutubular junction of the aorta. The semilunar attachments of the leaflets themselves from the haemodynamic junction between left ventricle and

aorta. All structures distal to these attachments are subject to arterial pressures, whereas all parts proximal to the attachments are subjected to ventricular pressures.

The structures distal to the semilunar attachments are the valvar sinuses, into which the semilunar leaflets themselves open during ventricular systole. Two of these valvar sinuses give rise to the coronary arteries, usually at or below the level of the sinutubular junction. The arrangement of the coronary arteries permits these two sinuses to be called the right and left coronary aortic sinuses. When their structure is examined, it can then be seen that, for the greater part, the sinuses are made up of the wall of the aorta. At the base of each of these coronary sinuses, however, a crescent of ventricular musculature is incorporated as part of the arterial segment. This does not happen within the third, non-coronary sinus. This is because the base of this sinus is exclusively fibrous in consequence of the continuity between the leaflets of the aortic and mitral valves.

Examination of the area of the root proximal to the attachment of the valvar leaflets also reveals unexpected findings. Because of the semilunar nature of the attachments, there are three triangular extensions of the left ventricular outflow tract which reach to the level of the sinutubular junction. These extensions, however, are bounded not by ventricular musculature, but by the thinned fibrous walls of the aorta between the expanded sinuses. Each of these triangular extensions places the most distal parts of the left ventricle in potential communication with the pericardial space or, in the case of the triangle between the two coronary aortic valvar sinuses, with the tissue plane between the back of the subpulmonary infundibulum and the front of the aorta. The triangle between the left coronary and the non-coronary aortic valvar sinuses forms part of the aortic-mitral valvar curtain, with the apex of the triangle bounding the transverse pericardial sinus. The triangle between the non-coronary and the right coronary aortic valvar sinuses incorporates within it the membranous part of the septum. This fibrous part of the septum is crossed on its right side by the

hinge of the tricuspid valve, which divides the septum into atrioventricular and interventricular components. The apex of the triangle, however, continuous with the atrioventricular part of the septum, separates the left ventricular outflow tract from the right side of the transverse pericardial sinus, extending above the attachment of the supraventricular crest of the right ventricle.

When considered as a whole, therefore, the aortic root is divided by the semilunar attachment of the leaflets into supravalvar and subvalvar components. The supravalvar components, in essence, are the aortic sinuses, but they contain at their base structures of ventricular origin. The supporting subvalvar parts are primarily ventricular, but extend as three triangles to the level of the sinutubular junction. Stenosis at the level of the sinutubular junction is usually described as being "supravalvar". In that the peripheral attachments of the leaflets are found at this level, the junction is also an integral part of the valvar mechanism. Indeed, stretching of the sinutubular junction is one of the cardinal causes of valvar incompetence.

Aortic valve annulus

The aortic annulus is the fibrous tissue to which the leaflets are attached. This is formed in the shape of a cylindrical aortic root, with the valvar leaflets supported in crown-like fashion.

Aortomitral Continuity

The central fibrous skeleton of the heart includes the right fibrous trigone (the central fibrous body), the left fibrous trigone and the membranous septum.

The non coronary leaflet straddles the central fibrous body overlying the anterior leaflet of the mitral valve. The conduction tissue traverses the membranous septum between the right coronary and non coronary leaflets.

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Assessment of Aortic Stenosis and Indications for Surgery

Jean-Louis Vanoverschelde

Echocardiography has become the standard means for evaluation of aortic stenosis (AS) severity. Cardiac catheterization is no longer recommended, except in rare cases when echocardiography is non-diagnostic or discrepant with clinical data.

Echocardiographic assessment.

Anatomic evaluation of the aortic valve is based on a combination of short- and long-axis images to identify the number of leaflets, and to describe leaflet mobility, thickness, and calcification. In addition, the combination of imaging and Doppler allows the determination of the level of obstruction; subvalvular, valvular, or supravalvular. Transthoracic imaging usually is adequate, although transesophageal echocardiography (TEE) may be helpful when image quality is suboptimal.

The primary hemodynamic parameters recommended for clinical evaluation of AS severity are the AS jet velocity, the mean transaortic gradient and the valve area by the continuity equation.

The antegrade systolic velocity across the narrowed aortic valve, or aortic jet velocity, is measuredusing continuous-wave (CW) Doppler. Accurate data recording mandates multiple acoustic windows in order to determine the highest velocity (apical and suprasternal or right parasternal most frequently yield the highest velocity; rarely subcostal or supraclavicular windows may be required). AS jet velocity is defined as the highest velocity signal obtained from any window after a careful examination. A maximal jet velocity > 4 m/s is usually associated with severe AS.

The difference in pressure between the LV and aorta in systole, or transvalvular aortic gradient, is another standard measure of stenosis severity. Gradients are calculated from velocity information using the simplified Bernoulli equation, and peak gradient obtained from the peak velocity does therefore not add additional

information as compared with peak velocity. However, the calculation of the mean gradient, the average gradient across the valve occurring during the entire systole, has potential advantages and should be reported. Potential causes of discrepancies between Doppler and catheterization estimates of transaortic gradients include: failure to take subvalvular velocities into account when these exceed 1 m/s, malalignment of jet and ultrasound beam, comparison between maximal instantaneous pressure gradient by echo and peak-to-peak pressure gradient by cath, significant pressure recovery (essentially when the oartic root is small) and confusion between a concomitant MR jet and the AS jet with Doppler.

Calculation of the stenotic orifice area or aortic valve area is helpful when flow rates are very low or very high, although even the degree of valve opening varies to some degree with flow rate. Aortic valve area is calculated based on the continuity equation, which requires three measurements: the AS jet velocity by CW-Doppler, the LVOT diameter, and the LVOT velocity by pulsed Doppler. The clinical measurement variability for continuity equation valve area depends on the variability in each of the 3 measurements, including both the variability in acquiring the data and variability in measuring the recorded data.

When LV systolic dysfunction co-exists with severe AS, the AS velocity and gradient may be low, despite a small valve area; a condition termed ‘low-flow low-gradient AS’. A widely used definition of low-flow low-gradient AS includes the following conditions: an effective orifice area < 1 cm^2, a LV ejection fraction < 40% and a mean pressure gradient < 30-40 mmHg. *Dobutamine stress* provides information on the changes in aortic velocity, mean gradient, and valve area as flow rate increases, and also provides a measure of the contractile response to dobutamine, measured by the change in SV or ejection fraction.

Indications for surgery.

Early valve replacement should be strongly recommended in all symptomatic patients with severe AS who are otherwise candidates for surgery. As long as mean gradient is still > 40 mmHg, there is virtually no lower EF limit for surgery.

On the other hand, the management of patients with low-flow, low-gradient AS (severely reduced EF and mean gradient < 40 mmHg) is more controversial. In patients with low gradient and with evidence of contractile reserve, surgery is advised since it carries an acceptable risk and improves long-term outcome in most patients. Conversely, the outcome of patients without contractile reserve is compromised by a high operative mortality despite a trend towards better survival after surgery. Surgery can, nonetheless, be performed in these patients but decision-making should take into account clinical condition (in particular, the presence of comorbidity), extent of coronary disease, and feasibility of revascularization.

Management of asymptomatic patients with severe AS remains a matter of controversy. The decision to operate on asymptomatic patients requires careful weighing of benefits against risks. Early elective surgery, at the asymptomatic stage, can only be recommended in selected patients, at low operative risk. This could be the case in the rare asymptomatic patients with depressed LV function not due to another cause, those with echocardiographic predictors of poor outcome suggested by the combination of a markedly calcified valve with a rapid increase in peak aortic velocity of > 0.3 m/s per year, or when an exercise test is abnormal, particularly if it shows symptom development, which is a strong indication for surgery in physically active patients.

Transcatheter aortic valve implantation

A.P. Kappetein

Degenerative aortic stenosis is the most common valvular heart disease in developed countries. The prevalence of severe aortic stenosis increases with age from 2-4% of people below 65 years of age to nearly 6% in people over the age of 85. Since the population life expectancy continues to rise, severe aortic stenosis represents a growing health problem: the global annual need for aortic valve replacement (AVR) is expected to triple to approximately 850,000 by the year 2050

According to the guidelines of the American College of Cardiology (ACC), the American Heart Association (AHA) and the European Society of Cardiology (ESC) for the management of patients with valvular heart disease, symptomatic patients with severe aortic stenosis should have AVR because prognosis is poor when treated conservatively.

Surgical aortic valve replacement (AVR) offers excellent long-term results even in elderly patients However, up to 60% of symptomatic patients are denied AVR because of high age or severe co-morbidity 7-10. Furthermore, the proportion of symptomatic patients is likely to be underestimated: up to 37% of the patients, who claim to be asymptomatic, are in fact symptomatic when an exercise test is performed. New transcatheter techniques to implant a prosthetic aortic valve in the diseased native location have only emerged recently and may be an alternative for patients at high risk for surgery.

About a year ago, the preliminary results of the Placement of Aortic Transcatheter Valves (PARTNER) trial Cohort B were published in the New England Journal of Medicine. A group of high-risk patients with severe aortic stenosis (AS) deemed non-surgical candidates were randomized to either transcatheter aortic valve implantation (TAVI) or standard medical therapy including balloon aortic valvuloplasty (BAV). The

one-year results showed a reduced rate of death to 30.7% in the TAVI group, compared to 50.7% in the standard therapy group. Safety assessment was however less in favor of the percutaneous technique, as 6.7% suffered a stroke or TIA 30 days within randomization, compared to only 1.7% in the standard therapy patients (p=0.03). After 1 year, this difference was still significant (10.6% vs 4.5%, p=0.04).Despite this increased incidence of thromboembolic events, the authors conclude that TAVI is the new golden standard for patients with severe AS who are too sick for surgery. It is important to note that all patients receiving TAVI in the Cohort B of The PARTNER Trial were treated using the TF approach.

Results from PARTNER cohort A, comparing transcatheter-valve implantation with surgery for severe aortic stenosis, show that the new catheter-based procedure is just as good as surgery in surgery-eligible patients for the primary end point of mortality.

At 30 days, deaths were numerically lower in the TAVI group, but not statistically different. By one year, deaths in both groups were nearly identical and met the predefined definition of noninferiority.

End point	TAVI (%)	Surgery (%)
30-d mortality	3.4	6.5
1-y mortality	24.2	26.8

Major strokes were higher in the TAVI-treated patients, both at 30 days and one year. In a combined end point of all stroke or transient ischemic attack (TIA), the difference between groups was statistically significant

Major bleeding was more than twice as common in surgery-treated subjects (19.5% vs 9.3%, p<0.001); new-onset atrial fibrillation was also nearly double in the surgery group (8.6% vs 16%, p<0.001). Vascular complications were nearly four times higher

in the TAVI group (11% vs 3.2%, p<0.001). Symptom improvement (NYHA class and six-minute-walk test) was greater in the TAVI group than the surgery group at 30 days, but no different at one year.

In all, 699 elderly patients (median age 84.1) with severe aortic stenosis were randomized to either TAVI or conventional surgery at one of 26 centers in the US, Canada (three centers), or Germany (one center). In the TAVI group, patients received the experimental device via the fully transfemoral route (244 patients) or via a transapical procedure (104 patients), which is typically used when the femoral artery is deemed unsuited to the TAVI catheters. Not surprisingly, patients in the transapical group were slightly higher risk than patients in either the transfemoral TAVI group or the aortic-valve-surgery group. Of note, 28 patients in the surgery arm either refused treatment or withdrew from the study after initial randomization; by contrast only one person in the TAVI arm refused treatment, and none withdrew between the time of trial enrolment and treatment. As such, findings in the intention-to-treat analyses were slightly different from those in the as-treated analyses, although Smith stressed that these differences did not materially affect the main trial findings.

Future studies focusing on intermediate risk patients

SURTAVI trial

So far, trials and registries on transcatheter heart valves have only included patients who were inoperable or at extremely high risk for surgery. But the use of transcatheter heart valves is expanding with more than 25,000 implants to date. Three types of intermediate risk patients will be discussed for randomisation by the heart team: 80 year olds without comorbidities; 75 year olds with more than 1 cormorbidity; and 70 year olds with 2 comorbidities. Patients under 70 years of age

will not be randomised because of insufficient evidence on the durability of the percutaneous heart valves.

SURTAVI needs to randomise around 1100 patients, which means 550 for surgery and 550 for transcatheter heart valves. Only centres with sufficient experience of transcatheter heart valves (at least 30 valve implants) will be eligible to participate and around 35-40 sites in Europe will be needed. The aim is to start enrolling patients in the second quarter of 2011.

US sites may be added later.

The primary endpoint in the SURTAVI trial is mortality and stroke. Secondary endpoints include valve failure, endocarditis and regression of the left ventricle. Valve registries have shown a higher pacemaker implantation rate (up to 25%) compared to surgery (1-2%) and this will also be monitored.

The study aims to show that TAVI treatment is noninferior to surgery. If it is shown to be noninferior, TAVI could be preferable for an 80 year old who would avoid having the chest opened and being put on a heart lung machine. Patients who receive surgery stay in hospital longer and take 3-4 months to recover which is quite a long time within an average of 7 years to live. It is hoped that the intermediate risk population will recover more quickly after TAVI than surgery and be discharged earlier from hospital.

The SURTAVI trial includes a cost effectiveness analysis which will look at number of days in hospital, number of ICU days, cost of the valve, other medications used, etc.

The recruitment period will take some time and the primary endpoint is therefore expected in 3 years from now. Hopefully the SURTAVI trial will offer us guidance who to treat intermediate risk patients: with surgical aortic valve replacement or with transcatheter valve implantation.

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Transcatheter aortic valve implantation

Aortic valve stenosis (AS) is the most prevalent valve disorder in the adult population in developed countries affecting approximately 2 to 4% of people over 65 years of age. This corresponds to approximately 3 million people with AS in Europe alone. One in five will eventually progress to symptomatic AS representing 600 000 patients.

Patients with severe AS face a grim prognosis once they become symptomatic. The landmark paper on symptomatic AS by Ross and Braunwald in 1968 highlighted this premise: median survival averages only 2, 3 and 5 years after symptom onset of angina, syncope and heart failure respectively. Both, the ESC and ACC/AHA cardiology societies have endorsed guidelines on valvular heart disease emphasizing the need for surgical aortic valve replacement once symptoms develop or in case of impaired LV function. Despite these well-established guidelines, one in every three patients with symptomatic AS is denied surgery mostly because of age, left ventricular dysfunction and co-morbidities. Nevertheless no medical treatment has any impact on survival. If we would assume that only two in every three patients with symptomatic AS would be referred for AVR and there are 600 000 patients with symptomatic AS in Europe alone, this means that hypothetically about 200 000 patients would not be considered for intervention. This unmistakably underscores an unmet clinical need. Undoubtedly this reality and patients' and physicians' preferences for lesser invasive strategies have fuelled the ongoing interest in developing minimally invasive transcatheter therapies.

The PARTNER trial (Placement of AoRTic TraNscathetER Valve Trial) is the first to randomize patients with high or prohibitive operative risk to AVR, TAVI or medical therapy following an operative assessment of the patient: inoperable patients are randomized to TAVI or medical therapy whereas patients with high operative risk are randomized to AVR or TAVI.

PARTNER Trial

Inclusion criteria

Severe symptomatic AS (AVA < 0.8cm^2, mean gradient >40mmHg, peak velocity >4m.s^{-1}). There were two pre-defined cohorts, those felt to be suitable for surgery but at high risk (STS score >10) or co-morbid conditions with a 30-day post-operative mortality >15%.

Exclusion criteria

Bicuspid or non-calcified AoV, AMI, CAD requiring revascularization, LVEF < 20%, aortic annulus <18mm or >25mm, severe MR/AR, TIA or stroke within 6 months.

Cohort A

In cohort A, a total of 699 patients were randomized 1:1 to AVR or TAVI (trans-femoral or –apical). The primary end-point at one year was death from any cause and the trial was powered on the assumption that TAVI was non-inferior to conventional surgical AVR.

The 30-day mortality was 3.4% vs. 6.5%, TAVI vs. AVR (non-significant) and this was also non-significant at one year 24.2% (TAVI) vs. 26.8% (AVR). Stroke rates were similar between the two groups 3.8% vs. 2.1% at 30-days. There were significant differences in the peri-procedural risks, major vascular complications were more frequent with TAVI 11.0% vs. 3.2% but major bleeding and AF were more frequent in the conventional AVR group 9.3% vs. 19.5% and 8.6% vs. 16.0%, respectively. Although patients undergoing TAVI were symptomatically better at 30-days, this difference disappeared at one year.

Cohort B

In addition, for inclusion within this cohort, at least two surgeons and a cardiologist had to agree that the patient was not a surgical candidate. Based on pre-existing conditions with a predicted 50% 30-day post-operative mortality.

Patients with severe symptomatic AS who were felt to be at too high risk for conventional surgery were randomized to either TAVI or optimal medical therapy (OMT). All patients were followed up for at least one year. Cross-over from OMT to TAVI was not allowed. Co-primary endpoint was the rate of a hierarchical composite of the time to death from any cause or repeat hospitalisation due to valve or procedure related clinical deterioration.

A total of 358 patients were randomized. STS score was 11.2 vs. 12.6 and logistic EuroSCORE 26.4 vs. 30.4 (TAVI vs. OMT). In the OMT group 84% underwent balloon valvuloplasty, 6.7% AVR and 2.2% TAVI (at non-participating centres).

At 30-days, the rate of death 5% in TAVI group and 2.8% in the OMT. The one-year all cause mortality was significantly lower for the TAVI vs. OMT as was the cardiovascular mortality 30.7 vs. 50.7% and 20.5% vs. 44.6% respectively. Major stokes were seen more frequently in the TAVI group (non-significant) at 30-days and 1 year 5.0% vs. 1.1% and 7.8% vs. 3.9%, TAVI vs. OMT. However the rate of composite stroke or any cause death was still significantly lower at one-year 33.0% vs. 51.3%, TAVI vs. OMT. Symptomatic improvement was =significantly greater for the TAVI group at 30 days, 6 months and one year.

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Patient Prosthesis Mismatch

The concept of patient prosthesis mismatch was first introduced by Rahimtoola to describe a condition when the effective orifice of an aortic prosthesis is less than that of the normal human valve. When applied in this fashion, the vast majority of implanted valves exhibit PPM. Pibarot was the first physician to attempt to quantify the degree of mismatch by analysing the effective orifice area of implanted valve and indexing it to the BSA to produced the EOAi. He quantified moderate mismatch as being present with the EOAi was between 0.65 – 0.85 $cm^2.m^{-2}$ and severe mismatch as being present when the EOAi was less than 0.65 $cm^2.m^{-2}$. A step-wise increase in mortality with increasing severity of mismatch was demonstrated.

Further work comparing the projected EOA from in-vitro manufacturer data demonstrated that this correlated well with the EOA measured by echocardiography. It should however be noted that due to the phenomenon of pressure recruitment, the EOA is seen to increase for the first six months as both the LV and aorta remodel with the change in orifice area following surgery.

Blackstone then produced a paper examining the impact of PPM. Instead of analysing the results on the basis of either measured or projected EOA, his group instead used the geometric orifice area (GOA). This method simply uses the manufacturer's quoted internal orifice diameter of the valve to calculate an orifice area on the basis of it being a perfect circle. Although he demonstrated an increase in early and late mortality in patients with the most severe mismatch, subsequent studies have demonstrated that the GOA correlates very poorly with the in vivo EOA and this method has subsequently been abandoned.

The concept of PPM was that a residual gradient would result in adverse haemodynamics, failure of left ventricular hypertrophy to regress and therefore lead to an increase in late events and reduced survival. Although a number of studies have demonstrated a close relationship between the predicted EOA and the in vivo

EOA, an interesting paper by Hanayama et al demonstrated that low EOAis did not necessarily correlate with high trans-valvular gradients. Although in part this was attributed to a degree of pressure recruitment, it is clear that our understanding of the haemodynamics of the remodelling ventricle is oversimplified. Many patients with severe concentric LVH secondary to pure AS have small stroke volumes and even small EOA may be able to accommodate such volumes without generating large gradients. It should also be remembered that whilst a smaller valve may present a degree of fixed obstruction it will have no or at least very little opening gradient, and therefore the intensely energy consuming processes of isovolumetric contraction will not be prolonged as is seen in native valve stenosis.

In response to Pibarot's series of papers suggesting a correlation with PPM and adverse outcomes a number of centres performed retrospective analyses using the projected EOAi. These studies were carried out by surgeons rather than cardiologists and all agreed that whilst moderate PPM was common affecting between 40 -80% of implanted valves, severe PPM was rare (<10%). It was also noted that PPM was more common in elderly and in female patients and when this was taken into account, no increased mortality was identified in the PPM groups. Further work examining LV reverse remodelling found that although this process was slower than in patients without mismatch, by 18 months the degree of reverse remodelling was similar and is probably as dependent on genetic patient factors, control of blood pressure, use of ACE inhibitors and chronic kidney disease as it is on valve area.

Following numerous risk adjusted reports, none of which found a correlation between PPM and survival, Pibarot and other proponents of this hypothesis started examining the effect of PPM in certain sub-groups including younger patients, patients with impaired LV function and patients with severe hypertrophy. Whilst the logic behind these ideas is sound, the small number of patients examined means it has been difficult to draw any clear conclusions.

With the advent of TAVI, the issue of PPM has again become topical. Walther and Pibarot have now both published anecdotal reports that TAVI, by over-sizing the annulus leads to the implantation of larger valve sizes, improving haemodynamics and therefore a reduction in the incidence of mismatch and on these grounds is therefore favourable in the smaller aortic root. It should also be noted that these papers also stated that TAVI resulting in better haemodynamics results in improvements in ejection fraction. This conclusion however failed to take into account the increased incidence of ≥ moderate para-valvular leak, which in itself will increase stroke volume and will therefore lead to an overestimation of true EF.

Summary

By the strict definition of Rahimtoola, PPM is common following AVR. Severe PPM (EOAi <0.65$cm^2.m^{-2}$) is however rare and when it does occur, is more common in female patients and the elderly. Whilst there is no good data to support an adverse effect on survival in general, it may be more important in patients with poor left ventricular function and it may reduce the symptomatic benefit of AVR. The evidence for this does however need corroboration and as yet, here is no evidence to support the use of TAVI as a strategy to avoid mismatch.

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Aortic Valve and Root Repair

Prof. J. Mark Redmond

Experience is growing with valve-sparing techniques to treat pathology of the aortic valve and root. While valve-sparing aortic root replacement pioneered by David and Yacoub has standardised treatment of aortic root pathology, it has also laid the foundation for repair techniques of the aortic valve.

As a functional entity, the aortic valve consists of the Sinotubular junction (STJ) and the Aorto-Ventricular junction (AVJ) which together form the functional aortic annulus (FAA), and the valve cusps; thus a fundamental principle of in aortic valve (AV) repair is that lesions of the cusps and the FAA must be identified and addressed at the time of repair.

A functional classification of aortic incompetence identifies all the contributing lesions of the cusps and the FAA and facilitates a reconstructive approach to the aortic valve (table 1): type 1 disease is caused by lesions of the FAA with normal cusp motion; type 2 disease is caused by excessive cusp motion due to cusp prolapse; type 3 disease is caused by restrictive cusp disease.

The goal of AV repair is to restore a normal surface of coaptation by restoring normal geometry to the leaflets and the FAA, while preserving normal mobility of the AV cusps. The mobility of the valve cusps is a ratio between the free margin length and the length of the base of the cusp insertion. Cusp prolapse occurs as a result of an increase in the free margin length compared with the length of the cusp insertion, resulting in a decrease in the height of the prolapsing cusp; a callipers can be used to measure the effective cusp height, as described by Schafers[2,3], to guide free margin plication and free margin resuspension techniques for cusp prolapse repair (figure 1).

Regurgitant bicuspid aortic valves usually present with dilatation of the aortic annulus, aortic root or ascending aorta. Cusp morphology varies; in Type 1 bicuspid valves, there is a median raphe on the conjoined cusp and an asymmetric distribution of the aortic sinuses; in type 0, there is no median raphe and 2 symmetric sinuses, 2 commissures and symmetric cusp insertions. In regurgitant type 1 valves, the median raphe is resected when restrictive and calcified, and the leaflet edges approximated primarily or with the use of autologous or bovine pericardium, followed by correction of the effective cusp height using free margin plication or resuspension techniques. Additional lesions of the FAA are then addressed.

For tricuspid or bicuspid regurgitant valves, following cusp repair, repair of the FAA is often required; this may require sinotubular reconstruction, aortic root remodelling (to address the STJ and sinuses, Yacoub technique[5]), or aortic root reimplantation (to address the STJ, AVJ and sinuses, David technique[6]).

Patients undergoing cusp repair in tricuspid valves have an 8 year freedom from reoperation of 96% and freedom from recurrent AI of > 2+ of 90%; cusp repair in bicuspid valve results in freedom from AV reoperation of 87% at 8 years[1]. Results are independent of cusp repair technique in most studies. For aortic valve reimplantation (David technique) for root dilatation, freedom from AV replacement is 95% at 10 years[6], while for aortic root remodelling (Yacoub technique), freedom from AV reoperation is 89% at both 5 and 10 years[5]. A consistent finding in all major studies of AV repair is the low risk of valve-related morbidity including thromboembolism, bleeding and endocarditis (<0.5%/patient year)

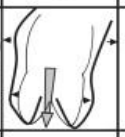
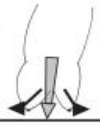
AI Class	Type I Normal cusp motion with FAA dilatation or cusp perforation				Type II Cusp Prolapse	Type III Cusp Restriction
	Ia	Ib	Ic	Id		
Mechanism						
Repair Techniques (Primary)	STJ remodeling *Ascending aortic graft*	Aortic Valve sparing: *Reimplantation or Remodeling with SCA*	SCA	Patch Repair *Autologous or bovine pericardium*	Prolapse Repair *Plication Triangular resection Free margin Resuspension Patch*	Leaflet Repair *Shaving Decalcificatio Patch*
(Secondary)	SCA		STJ Annuloplasty	SCA	SCA	SCA

Table 1 [1]

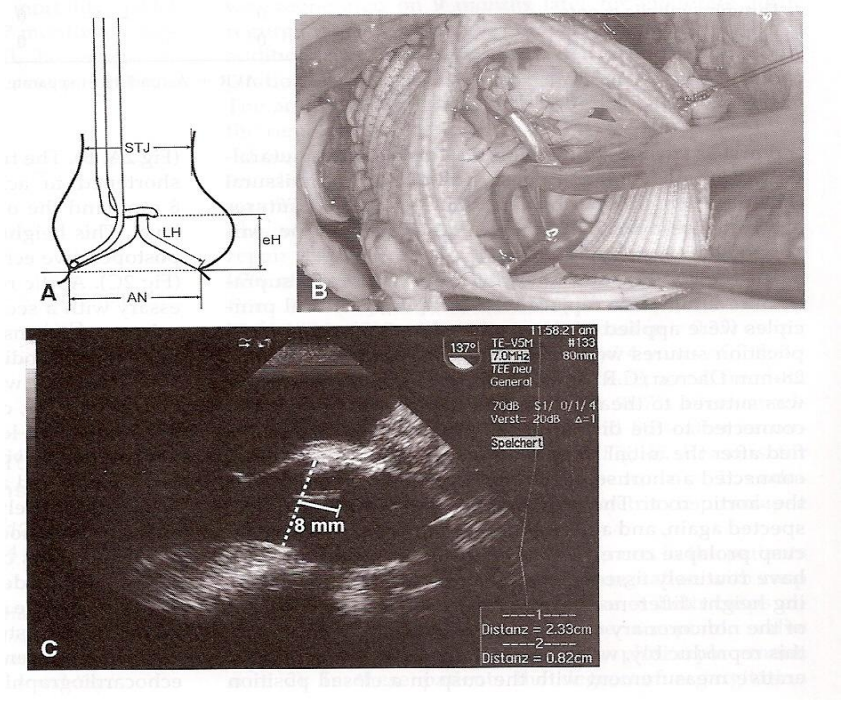


Figure 1

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Aortic dissection

Aortic dissection occurs when there is disruption of the aortic media, most commonly associated with an intimal tear with separation of the intima and medial layers. This dissection may propagate throughout the aorta and penetrate through the adventitia or return to the aortic lumen via the intima. Accurate anatomical classification of aortic dissection is of primary importance as it is on this initial classification that the decision for operative intervention or medical therapy is decided. The two most commonly used classifications for aortic dissection are the Stanford and DeBakey classifications. The Stanford classification divides dissections into types A (involving ascending aorta ± descending aorta) and B (descending aorta only). The DeBakey classification divides dissections into type I (ascending with distal extension), type II (ascending only) and type III (descending thoracic aorta). The true incidence of aortic dissection is difficult to ascertain but its prevalence may be increasing. The estimated total incidence of acute (type A and B) dissection is 30-43 per million of population per annum and acute type A aortic dissection (ATAAD) constitutes >50% of all cases.

There are few randomised controlled trials investigating interventions in thoracic aortic disease and the majority of the data comes from large series as well as the recent data collected by the International Registry of Acute Aortic Dissections (IRAD). The AHA/ACC have recently published guidelines on the management of thoracic aortic disease including dissection.

New insights into an old disease

The IRAD group assessed the presentation, management and outcomes of acute aortic dissection. A total of 464 patients from 12 participating IRAD centres. The majority of patients present with abrupt onset (85%), severe (91%) chest pain (73%).

The majority of patients were male (65%) in the sixth decade of life with a history of hypertension (72%).

Type A

Of the cohort 62% had a type A dissection, the site of chest pain was most commonly anterior and aortic regurgitation was present in 44%. Nearly all (89%) of patients had an abnormal CXR most commonly with widening of the mediastinum. ECG findings were most commonly non-specific ST-segment/T wave abnormalities and LVH.

In-hospital mortality for patients treated by surgery was 26% and except for notable specialist centres this mortality is representative of the current risk. Conservative therapy was employed in 28% of patients and their in-hospital mortality was 58% with the highest risk of mortality in the first seven days. Mortality was most commonly due to rupture and tamponade (42%).

Type B

Patients with type B dissection presented with posterior chest, back and abdominal pain. The majority of the patients were hypertensive at presentation 70% vs. 36% (type A). A total of 20% of patients underwent surgery with an in-hospital mortality of 31%. Medically managed patients had an in-hospital mortality of 11%.

Contemporary results of surgery

IRAD analysed 290 variables in 526 patients undergoing surgery for type A dissection with an in-hospital mortality of 25.1%. Patients were categorised as Group 1 (unstable – tamponade, shock, neurological deficit, myocardial ischaemia, malperfusion, acute renal failure) or Group 2 (stable). In hospital mortality was 31% vs. 17% (1 vs. 2, $p<0.001$). In non-survivors, death occurred in 13% within six hours,

in 34% within 24 hours and within 41% in 48 hours after the beginning of the operation.

Stepwise logistic regression was performed and the following independent risk factors were noted:

History of AVR (OR= 3.1), migrating chest pain (OR= 2.8), presenting hypotension (OR= 2.0), presenting shock/tamponade (OR= 2.7), pre-operative tamponade (OR= 2.2) and pre-operative limb ischaemia (OR= 2.1).

Long-term survival type A

IRAD analysed the outcomes of 303 acute type A aortic dissection (within 14 days of symptom onset) at a median follow-up of 2.8 years. Within this cohort 90.1% were managed surgically and the rest medically. All cause mortality during the 5-year follow-up period occurred in 16.2% of patients with a mean survival of 2.4 years. Surgically managed patients had a follow-up mortality of 13.9%, mean survival 2.5 years and medically managed patients had a mortality of 36.7%, mean survival 2.1 years. Unadjusted survival rates were 96.1±2.4% vs. 88.6±12.2% at 1 year and 90.5±3.9% vs. 68.7±19.8% at 3 years (surgery vs. medical, p= 0.009).

Independent predictors of follow-up mortality with age (≥70) and gender adjusted Cox proportional hazard analysis were atherosclerosis (OR, 2.17) and history of previous cardiac surgery (OR, 2.54).

Importance of false lumen thrombosis in type B

Two hundred and one patients with acute type B aortic dissection (surviving to hospital discharge were stratified according to false lumen status (patent, partial thrombosis, complete thrombosis) as determined during the index hospitalisation. The false lumen was patent in 56.7%, partially thrombosed in 33.8% and completely

thrombosed in 9.5%. All patients were initially managed medically, 72.6% had medical management alone, 17.9% had surgery and 9.5% an endovascular intervention (no difference between groups). Median follow-up was for 2.8 years. Mortality was highest in patients with a partially thrombosed false lumen, 15.4% and 31.6%, 5.4% and 13.7% (patent false lumen) and 0% and 22.6% (completely thrombosed) at 1 and 3 years respectively. Partial thrombosis vs. a patent false lumen was an independent predictor of post-discharge mortality, when patients who received either surgey or endovascular treatment were excluded from the analysis, partial thrombosis still remained a significant independent predictor of mortalilty.

INSTEAD

In this RCT, 140 clinically stable patients at least two weeks following index dissection were randomised to OMT/TEVAR (n= 72) or OMT (n= 68). There was no difference in baseline characteristics or time to randomisation. Median time to TEVAR following randomisation was 12 days. TEVAR was completed successfully in 70 patients and 82.9% had 1 stent graft, 11.4% had 2 stent grafts and 5.7% 3 stent grafts. No patients required conversion to surgery. Survival at 2 years was 88.9% vs. 95.6 % (TEVAR vs. OMT, *p*= 0.15). Cumulative freedom from aorta related deaths were 94.4% vs. 97.0% and for a combined end-point of aorta-related death, crossover for expansion and ancillary procedures was 77.2% vs. 72.5% (TEVAR vs. OMT, p= 0.44 and 0.65, respectively). Aortic expansion to a diameter > 60mm occurred more frequently in the OMT group and 16.2% crossed over to TEVAR and 4.4% to surgery. In the TEVAR group4.2% underwent surgery and 8.3% stent graft extension. TEVAR led to a 91.3% false lumen thrombosis rate vs. 9.1% for OMT (p= 0.001).

Despite favourable aortic remodelling in stable patients with type B dissection, TEVAR did not improve adverse event rates or survival despite favourable aortic remodelling.

Hybrid stenting at open repair of type A

In a non-randomised study, 78 patients with acute DeBakey type 1 dissection, 42 underwent conventional open repair and 36 additional stent grafting by the open arch. Baseline demographics were similar between groups. CPB times were similar 229 vs. 245 minutes and HCA times longer 18 vs. 60 (SACP utilised) minutes (non-stented vs. stented, p= 0.09 and <0.001 respectively). In-hospital mortality was 14% in both groups. Mean follow-up was 15.9 months. There were no late deaths in the stented and 2 late deaths in non-stented group. Transient paraparesis occurred in 3 stented patients, and 1 conventional, but resolved completely in all. Complete thrombosis of the false lumen was achieved in 17% of patients in both groups and partial thrombosis in 60% of stented and 8% of non-stented. Mid- and long-term results of such novel therapies are awaited.

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A sample operation note for aortic dissection repair

Patient: Mr I. A. MILL Surgeon: Mr. I.N. Competent

DIAGNOSIS: Type A aortic dissection. Aortic regurgitation. Acute renal dysfunction.

Thrombolysis 48 hours pre-operation

OPERATION: Aortic valve resuspension, ascending aortic replacement

PRESENTATION AND INDICATIONS: 55 year old male presenting with sudden onset chest and back pain. Initially inferior ST segment changes – Thrombolysed *(indicative of right coronary artery malperfusion)*. Continuing pain - CT scan demonstrated Stanford type A dissection (DeBakey type 1). Echo demonstrated good LV, moderate aortic regurgitation and 2cm pericardial effusion. Increasing pre-operative renal dysfunction on transfer - creatinine 350umol/L *(indicative of low cardiac output state).*

Clinical examination demonstrated an elevated jugular venous pressure *(?tamponade)* and a right arm blood pressure of 95/45. The patient had cool peripheries *(?low cardiac output state/tamponade)* but all peripheral pulses were palpable. The right brachial pulse was of large volume *(?collapsing)(indicative of aortic regurgitation*). Neck auscultation revealed bilateral carotid bruits *(indicative of epiaortic vessel ostium involvement)*. There was some mental obtundation but no focal neurological signs. The patient was immediately transferred to the operating theatre.

MONITORING: Bilateral radial artery cannulae were inserted revealing equivalent pressures (R = L). A standard quad-lumen central venous cannula was inserted into the right internal jugular vein together with a sheath and pulmonary artery flotation catheter.

INCISION: The patient was fully prepped and draped allowing full leg and groin access. A right vertical groin incision, crossing the inguinal ligament was performed over the right femoral pulse. The RCFA was isolated and taped from the inguinal ligament to just proximal to the profunda femoris take-off. The patient was then heparinised and the RCFA cannulated via a longitudinal arteriotomy for initial arterial return. The arterial pressure within the cannula was pulsatile. The RCFA was later repaired with a running suture of 5'O' prolene. The wound was closed with a polydiaxone suture.

A median sternotomy was performed leaving the pleurae intact. The appearance of the pericardium suggested a haemopericardium. Following a longitudinal pericardiotomy a purse-string suture was applied to the right atrial appendage.

BYPASS TECHNIQUE: Right atrial drainage and RCFA return. Satisfactory flows obtained and no difference in right and left radial artery pressure noted on commencement of bypass. Later, the arterial return was transferred to a graft side-arm following construction of a distal aortic anastomosis. The LV was vented. The patient was then cooled to 15^{o}C using a 7^{o}C maximum temperature differential and α-stat pH control. When a naso-pharyngeal temperature of 15^{o}C was attained, cooling was stopped and the 15^{o}C naso-pharyngeal temperature maintained before committing to circulatory arrest.

MYOCARDIAL AND CEREBRAL PROTECTION: Intermittent antegrade cold blood cardioplegia applied approximately at 20 minute intervals. Cooled to 15^{o}C. Pre-treatment with dexamethasone 100mg and mannitol 1g/kg prior to circulatory arrest. Prior to circulatory arrest, the patient was placed in a 15^{o} Trendelenberg position to allow an open distal anastomosis construction during a 27 minute period of hypothermic circulatory arrest. During cooling, the head was packed in ice. At circulatory arrest, anaesthetic infusions were discontinued and restarted following the arrest period. Rewarming was undertaken with similar maximal temperature

gradients and α-stat pH management strategy. The maximal blood outflow temperature was not allowed to exceed 37°C. Bypass was discontinued after a naso-pharyngeal temperature of 36°C had been reached and maintained for 10 minutes.

FINDINGS: There was a haemopericardium with approximately 300ml of blood and clot evacuated on opening the pericardium. Heart action appeared satisfactory apart from some minor depression of RV free wall motion. The ascending aorta was dilated (approximately 4cm) and had a purplish discoloration compatible with acute dissection. There was epicardial-epi-aortic haematoma within the atrioventricular groove near the right coronary artery and between the aorta and pulmonary artery. When visualised, the aortic valve was tricuspid and tri-commissural and morphologically normal. There was an acute dissection arising from a transverse intimal tear 2 cm distal to left coronary ostium. Retrograde dissection into the aortic root and led to a loss of commissural support for right/non- and left/non-coronary commissures. Coronary ostia normally positioned but the ostium of the right coronary artery was dissected without breaching of the intima. Examination of the aortic arch revealed a small (2cm) further transverse tear in proximal aortic arch opposite the innominate artery on the under-surface.

The calibre of the aortic arch was normal. The dissection had generated a large intimal flap involving all epiaortic vessels with dissection of their ostia. The intima within the ostia was not breached.

PROCEDURE: Following institution of bypass and initiation of cooling a soft cross-clamp was applied to the distal ascending aorta during a period of reduced pump flow. Once pump flow was restored, there was marked (>30mmHg) discrepancy between left and right radial artery pressures (right lower) *(suggestive of malperfusion of at least one of the epiaortic arteries)*. The cross-clamp was therefore removed allowing equalisation of right and left radial pressures. Cooling was continued until the temperature nadir was attained. In Trendelenberg position, the

circulation was arrested and the aorta opened obliquely. The opened aortic arch was carefully inspected taking care not to over-aspirate blood within the epiaortic vessels. Antegrade cardioplegia was instilled directly via the coronary artery ostia to achieve complete electro-mechanical arrest. The ascending aorta was then transected just proximal to the innominate artery with line of section excising the arch tear. A wet arterial swab was temporarily placed within the arch to prevent glue spillage and the dissected layers of the aorta were then gently separated to allow instillation of glue between the layers for a distance of approximately 2-3cm within the arch. The dissected layers were held in apposition with soft forceps and clamps until the glue became fixed. The swab was then removed and the any intimal glue residue aspirated. A 0.75cm wide strip of Teflon was then sutured around the open arch using 4'O' prolene, commencing at the most leftward extent of the opened aortic arch and working towards the surgeon. Great care was taken to apply the strip of Teflon to the outside of the aortic wall rather than the transected edge. A 30mm protein-impregnated polyester graft with a single side-arm was then trimmed and bevelled. The graft was trimmed and orientated so that the side-arm would lie on the anterior-inferior aspect of the graft approximately 2cm from this distal anastomosis. This graft was then anastomosed to the buttressed aortic wall applying a further buttress on the graft side.

Quite deep bites were taken into the graft to allow partial telescoping of the graft into the aortic arch. The direction of suturing was again from most distally towards the surgeon suturing the inferior wall first. Sutures were applied loosely and later tightened using a small nerve hook. Great care was taken to ensure that buttressing was on the external aspect of graft and aorta and not within the meeting edges of the suture line. Once this anastomosis was ¾ complete leaving only the right-sided anterior aspect to be completed, the arterial return limb of the bypass circuit was transferred to the graft side-arm using a separate cannula. This side-arm was de-

aired by minimal forward pump flow. An infusion line was passed out to the anaesthetist and inserted into the proximal open end of the graft and an infusion of 4^{o}C cold saline solution gently infused into the graft to allow de-airing during the completion of the anastomosis. Once complete, the saline infusion was stopped and forward bypass flow gently recommenced within an open graft. The graft, aorta and epiaortic vessels were massaged and agitated to complete arch airdrill. The graft was then clamped and bypass flow restored and rewarming commenced 5 minutes after ending the arrest period. With pump flow restored; this distal anastomosis was carefully inspected for haemostasis, moving the graft to allow full inspection under pressure. No suture line bleeding or oozing was accepted and several additional Teflon buttressed sutures were inserted during periods of temporarily reduced flow were placed to achieve absolute haemostasis. Once this was achieved, attention was directed to the proximal aorta. Cardioplegia was re-administered and the aorta transected 1cm distal to the sino-tubular junction taking care to ensure that the adventitial-medial outer layer was not over-excised and compromised. The ascending was then excised completely using cautery ensuring the safety of the main and right pulmonary arteries. The aortic valve was re-suspended with 3 Teflon buttressed 4'O' prolene mattress commissural sutures which were not tied until glue apposition of the dissected root layers was complete. A small wet swab was then inserted into the root to protect the valve and coronary ostia and glue applied between the dissected layers including around right coronary ostium The layers were then held in apposition with soft forceps and clamps until the glue became fixed, intermittently applying a right angled clamp into the right coronary ostium to ensure that its mouth was not compromised by an excess of glue. Following this, an external 0.75cm Teflon collar was applied to outer wall of the sinotubular junction using a running prolene suture working towards the surgeon. A separate length of the 30mm polyester graft was then anastomosed to sino-tubular junction with a further buttress on the graft but not on the aorta. This suture line was initially constructed loosely and

tightened later with a fine nerve hook ensuring the siting of the buttress sutures. A small hole was then made in the graft 2-3cm from the right coronary ostium and a cardioplegia cannula inserted and secured with a buttressed mattress suture used as a purse-string. Antegrade cold blood cardioplegia was then instilled, the graft de-aired and then clamped. The delivery of pressurised antegrade cardioplegia allowed inspection of this proximal anastomosis and insertion of additional haemostatic sutures as necessary. The two grafts were then trimmed to appropriate length and anastomosed together using Teflon buttressed 3'O' prolene. The integrity of this anastomosis was tested using pressurised cardioplegia.

Weaned from bypass in SR but relapsing into AF periodically requiring cardioversion. Good heart action and output. On table trans-oesophageal echocardiogram revealed trivial aortic regurgitation only.

Haemostasis, protamine and decannulation.

CLOSURE: Wires & Vicryl. 2 drains and Redivac drain peri-graft. 2 RV & 2 RA pacing wires.

Further reading

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Anatomy of the mitral valve

Ian Wilson

General Anatomy

The valvar complex comprises the annulus, the leaflets, the tendinous cords, and the papillary muscles. Also important for its functioning is the left atrial musculature inserting to the leaflets and the myocardium to which the papillary muscles are inserted. The valve is obliquely located in the heart and has a close relation to the aortic valve. Unlike the tricuspid valve which is separated by muscle from its counterpart, the pulmonary valve, the mitral valve is immediately adjacent to the aortic valve.

Mitral annulus

The annulus marking the hinge line of the valvar leaflets is more D shaped than the circular shape portrayed by prosthetic valves. The straight border accommodates the aortic valve allowing the latter to be wedged between the ventricular septum and the mitral valve. In this region, the aortic valve is in fibrous continuity with one of the two leaflets of the mitral valve. Expansions of fibrous tissues at either extreme of the area of continuity form the right and left fibrous trigones. The atrioventricular conduction bundle passes through the right fibrous trigone.

Although the term annulus implies a solid ring-like fibrous cord to which the leaflets are attached, this is far from the case. In the area of aortic–mitral fibrous continuity, the distal margin of atrial myocardium over the leaflet defines the hinge line. When viewed from the ventricular aspect, however, the hinge line is indistinct since the fibrous continuity is an extensive sheet. There are prongs of fibrous tissues from each of the fibrous trigones but these were not continuous around the orifice. The annulus opposite the area of valvar fibrous continuity tends to be "weaker" in terms of lacking a well formed fibrous cord. This is the area affected in "annular dilation"

and also most often involved in calcification of the annulus. With severe dilation, the minor axis of the valvar orifice becomes so distended that the leaflets, which are of fixed lengths, become unable to approximate each other.

Leaflets

Distinctly different from the tricuspid valve, the mitral valve has two leaflets. These are notably different in shape and circumferential length. Owing to the oblique location of the valve, strictly speaking, its two leaflets do not occupy anterior/posterior positions nor is one of the leaflets "septal". The septal leaflet is characteristic of the tricuspid valve whereas neither of the mitral leaflets is attached to the septum. The corresponding terms for anterior and posterior are "aortic" and "mural". It is the aortic leaflet that is in fibrous continuity with the aortic valve. The aortic leaflet has a rounded free edge and occupies a third of the annular circumference, whereas the other leaflet is long and narrow, lining the remainder of the circumference. The aortic leaflet hangs like a curtain between the left ventricular inflow and outflow tracts. When the valve is closed, this leaflet appears to form the greater part of the atrial floor but is approximately equal in area to the mural leaflet. It meets the mural leaflet to form an arc shaped closure line, or zone of apposition, that is obliquely situated relative to the orthogonal planes of the body. With the leaflets meeting, the view of the valve from the atrium resembles a smile. Each end of the closure line is referred to as a commissure. These are designated the anterolateral and posteromedial commissures. It is worth noting, however, that the indentations between leaflets do not reach the annulus but end about 5 mm short in the adult heart. Therefore, there are no clear cut divisions between the two leaflets. Furthermore, the free edge of the mural leaflet is often divided into three or more scallops or segments described as lateral, middle, and medial or assigned terms like P1, P2, and P3. Although three scallops are most common, the scallops are not equal in size. The middle scallop

tends to be larger in the majority of hearts. When the mural leaflet is deformed in a floppy valve, the middle scallop is likely to be prolapsed.

Normally, the valvar leaflets are thin, pliable, translucent, and soft. Each leaflet has an atrial and a ventricular surface. When viewed in profile, two zones can be distinguished in the aortic leaflets and three zones in the mural leaflet according to the insertions of the tendinous cords. In both leaflets, there is a clear zone that is devoid of chordal attachments. Nearer the free edge, the atrial surface is irregular with nodular thickenings. This is also the thickest part, corresponding with the line of closure and the free margin. Tendinous cords attach to the underside of this area described as the leaflet's rough zone. The rough zone is broadest at the lowest portions of each leaflet but tapers toward the periphery, or commissure, of the closure line. The basal zone that is found only in the mural leaflet is the proximal area that has insertions of basal cords to its ventricular surface.

Being distant from the ventricular wall, the aortic leaflet does not have attachments to basal cords. In normal valve closure, the two leaflets meet each other snugly with the rough zone and free edge in apposition but at an angle to the smooth zone.

When the closed valve is seen in profile, the major part of the closure line lies below the plane of the atrioventricular junction rising toward the commissures at the peripheral ends so that the atrial surface of the leaflets has a saddle-like configuration. Being tethered by the tensor apparatus, the line of coaptation in a normal valve does not extend above the level of the junction during ventricular systole.

Tendinous cords

The tendinous cords are string-like structures that attach the ventricular surface or the free edge of the leaflets to the papillary muscles. Characteristically, the tricuspid valve has chordal attachments to the ventricular septum allowing it to be

distinguished from the mitral valve on cross sectional echocardiography. The tendinous cords of the mitral valve are attached to two groups of papillary muscles or directly to the postero-inferior ventricular wall to form the tensor apparatus of the valve. Cords that arise from the apices of the papillary muscles attach to both aortic and mural leaflets of the valve. Since cords usually branch distal to their muscular origins, there are five times as many cords attached to the leaflets as to the papillary muscles.

There are numerous classifications of tendinous cords. The predominant surgical classification distinguishes three orders of tendinous cord according to the site of attachment to the leaflets. The first order cords are those inserted on the free edge. They are numerous, delicate, and often form networks near the edge. Second order cords insert on the ventricular surface of the leaflets beyond the free edge, forming the rough zone. These are thicker than first order cords. Third order cords attach only to the mural leaflet since they arise directly from the ventricular wall or from small trabeculations. They insert to the basal portion of the leaflet and run only a short distance toward the free margin. In this area, webs may be seen in place of cords.

Papillary muscles and left ventricular wall

Papillary muscles are the muscular components of the mitral apparatus. As a functional unit, the papillary muscle includes a portion of the adjacent left ventricular wall. Tendinous cords arise from the tips of the papillary muscles. Alterations in the size and shape of the left ventricle can distort the locations of the papillary muscles, resulting in valvar function being disturbed. The papillary muscles normally arise from the apical and middle thirds of the left ventricular wall. Described in most textbooks as two in number, however, there are usually groups of papillary muscles arranged fairly close together. At their bases, the muscles sometimes fuse or have bridges of muscular or fibrous continuity before attaching to the ventricular wall. Extreme fusion results in parachute malformation with potential for valvar stenosis.

Viewed from the atrial aspect, the two groups are located beneath the commissures, occupying anterolateral and posteromedial positions. A single anterolateral papillary muscle occurs in 70% of cases and in 60% of cases that there are two or three papillary muscles, or one muscle with two or three heads, in the posteromedial location. Generally larger than the posteromedial muscle, the anterolateral muscle is supplied by an artery derived from the circumflex or anterior descending branch of the left coronary artery. Since most people have right dominance of the coronary pattern, it is the right coronary artery that most often supplies the posteromedial papillary muscle.

Rupture of a papillary muscle is usually the consequence of infarction of the adjoining ventricular wall. If rupture involves the entire papillary muscle or its group, there will be torrential regurgitation since approximately half the support of each leaflet will be lost. Rupture confined to one head of the papillary muscle complex will be similar to breaking a major cord. During systole, the affected free edge fails to meet with the other leaflet and moves into the left atrial cavity.

SECTIONAL ANATOMY OF THE MITRAL VALVE

Since the mitral valve is a complex with a unique arrangement of its component parts within the left ventricle, cross sectional imaging techniques including four dimensional echocardiography allow it to be visualised in its entirety by building up whole series of planes. The valve can be demonstrated in each of the orthogonal planes of the left ventricle, as well as in the orthogonal planes of the body.

Short axis planes through the ventricle display from apex to the cardiac base the oblique arrangement of the two groups of papillary muscles, the tendinous cords, the fish mouth appearance of the valvar opening, and the aortic outflow tract sandwiched between the ventricular septum and the mitral leaflet. This view allows assessment of the area of the valvar orifice. At right angles to the short axis plane, the long axis plane such as that obtained from the parasternal window produces the so-called two

chamber plane. In this view, the mode of closure of the leaflets and the level of the closure line relative to the atrioventricular junction is seen to best advantage. The aortic and mural leaflets are readily distinguished, allowing detection of hooding, overshoot, or prolapse of each leaflet. The normal valve in closed position shows the aortic leaflet at an angle to the long axis of the ventricle but the mural leaflet is perpendicular. It should be noted that in some normal valves the leaflets may balloon slightly past the plane of the atrioventricular junction during systole, but the zone of coaptation remains below the plane. In valvar opening, the mural leaflet becomes nearly parallel to the inferior wall while the aortic leaflet parallels the ventricular septum.

The second series of long axis sections through the left ventricle, the so-called four and five chamber planes, allow distinction between tricuspid and mitral valves. Being more or less parallel to the zone of apposition between leaflets, it is poor for detecting problems of coaptation. The series of two chamber sections obtainable from the apical window cuts the leaflets obliquely, distorting the true leaflet length and motion. Views of the mitral valve through the transgastric and transoesophageal windows enable more detailed studies of the entire length of the zone of apposition, revealing the arrangement of chordal supports at all segments of the leaflets.

Etiology, Definitions and Assessment of Mitral Regurgitation

Jean-Louis Vanoverschelde

The mitral valve is a complex, bi-leaflet structure that separates the left atrium and the left ventricle. It consists of two leaflets, a fibrous annulus, chordae tendinae, two papillary muscles and their left ventricular attachments. The different elements of the mitral valve apparatus act in concert to open the valve widely during diastole and close its orifice during systole. Mitral valve closure is the result of the dynamic interaction between the annulus, which contracts early in systole, the intraventricular pressure, which acts as the closing force, the chordae tendinae, which prevent the leaflets from prolapsing into the LA, the papillary muscles, whose systolic contraction share the same purpose and the leaflets themselves, whose large apposition along the coaptation line helps reduce the stress on the leaflets and of course serves to provide continence. Disruption in any of these anatomical or physiological structures can cause mitral regurgitation (MR).

The causes of mitral regurgitation are numerous. From a functional point of view, they can be grouped in 3 main categories, as popularized by Alain Carpentier.

Type 1 dysfunction is characterized by a normal leaflet motion. In this category, MR is usually the result of annular dilation. It can also be caused by leaflet perforation or congenital anterior leaflet clefts. By itself, annular dilatation rarely causes significant degrees of MR. However, in the presence of Type 2 or Type 3 dysfunctions; progressive annular dilatation is almost always present and contributes to the progression of MR severity.

Type 2 dysfunction is best described as an increased leaflet motion or leaflet prolapse. There is some controversy about the definition of mitral leaflet prolapses. In the echocardiographic literature, a mitral prolapse is considered to be present whenever any part of the leaflets overrides the plane of the annulus by more than 2

mm. As the mitral annulus is saddle shaped, positive identification of a mitral leaflet prolapse requires seeing the lowest part of the annulus, which is only possible in the long-axis orientation. In the surgical literature, the definition of the mitral prolapse is more restrictive and requires that part of the free edge of one (or more) of the leaflets overrides the annulus. If any other part of the valve overrides the annulus but the free edges remain above the annulus, the term billowing is then used. Type 2 dysfunction can be due to chordae or papillary muscle rupture, as well as chordae or papillary muscle elongation. In Western countries, chordae rupture or elongation are the most frequent causes of chronic MR.

Type 3 dysfunction is characterized by a restrictive leaflet motion. It can be seen either when the mitral valve apparatus is affected by an inflammatory process, such as rheumatic fever (type IIIa), or when the mitral apparatus is tethered, as in functional or ischemic MR (type IIIb).

Echocardiographic assessment.

Echocardiography is essential for establishing the presence of MR, quantitate its severity and hemodynamic consequences, determine its etiology and assess the potential for repairability.

The severity of MR should be evaluated using an integrated approach that includes valve morphology, the size of the regurgitant jet in the LA, the proximal regurgitant jet width or vena contracta, the pulmonary venous flow pattern and the measurement of the effective regurgitant orifice area (EROA) and the regurgitant volume (RV).

The Color jet area is more useful in evaluating the mechanisms of MR than in quantifying its severity.

The pulmonary vein flow pattern can provide information on the hemodynamic impact of MR but is purely qualitative and is affected by many factors (AF, LV dysfunction).

Measurement of the vena contracta width provides quantitative data on the size of the regurgitant orifice and hence on the severity of MR. The vena contracta corresponds to the narrowest segment of the jet on color flow imaging. Values exceeding 7 mm are usually associated with severe MR.

Quantitative measurements of the RV can be obtained using either the pulsed-wave Doppler method or the PISA method (Proximal Isovelocity Surface Area). The pulsed-wave Doppler method requires calculation of the aortic and mitral stroke volumes. In the absence of aortic regurgitation, the difference between these 2 stroke volumes should equal the RV. Values > 60 mL, for the RV, and ≥ 40 mm^2, for the EROA, indicate severe MR.

Assessment of etiology and repairability. Transthoracic echocardiography allows for the accurate determination of the cause of MR. Gray-scale echocardiography frequently permits identification of the underlying cause of the regurgitation, such as rupture of chordae tendinae, mitral valve prolapse with or without a flail leaflet, rheumatic mitral disease, vegetations and LV dilatation. It may also show calcification of the posterior mitral annulus. Color Doppler is also commonly used for the detection and also the characterization of MR. The direction of the MR jet provides valuable information on the etiology of MR. Eccentric jets, which are an easily identifiable using Color Doppler, are indeed frequently associated with an opposing leaflet prolapse. Mitral valve prolapse is associated with a 90% probability of surgical repair, as compared to 63% in case of rheumatic fever. As echocardiography allows for a precise description of the etiology of MR, it also allows for the prediction of valve repairability.

Indications of Surgery in Severe Mitral Regurgitation

Jean-Louis Vanoverschelde

Natural history

The natural history of chronic MR is highly variable, depending of the volume of regurgitation, the LV function, and the underlying cause of MR. Currently, most of the data available on the natural history of MR have been obtained in patients with flail leaflets. These observations may thus not be pertinent to MR of other causes.

Chronic severe MR is associated with a 1% per year risk of major complications which include congestive heart failure, infective endocarditis, cerebrovascular events due to LA enlargement and development of AF, need for mitral valve surgery, and death. Several prognostic factors have been identified in cohort studies.

In general, overall survival is influenced by the presence of symptoms and LV systolic dysfunction. Patients in NYHA functional class III or IV entail a much higher mortality rate (>30% yearly) than those in class I or II (4 % yearly). Similarly, patients with a LVEF < 60% exhibit a poorer 10-year overall survival (around 40%) than patients with a normal LVEF around 60%).

The occurrence of AF is also associated with a poorer survival. The annual incidence of AF is estimated at 5% per year. At 10 years, the incidence is higher in patients with LA enlargement (defined as an LA size > 50 mm), and in patients older than 65 years. Finally, the presence of pulmonary hypertension (defined as a pulmonary artery systolic pressure > 50 mmHg at rest or > 60 mmHg during exercise) is also associated with reduced survival.

The overall survival of MR is also directly and independently related to the severity of MR, as measured by the EROA. Five-year overall survival is indeed better (around 90%) in patients with an EROA < 20 mm², than in those with an EROA ≥ 40 mm² (only around 60%).

The yearly incidence of sudden cardiac death (SCD) approaches 1.8 %. It is higher in patients in NYHA class III/IV (8% / year) than in those in NYHA class II (3% / year) or NYHA class I (1% / year). It is also more frequent in the presence of LV dysfunction (13% / year when LVEF is < 50% vs. < 1.5% / year when LVEF is > 50%). Other risk factors for SCD include a history of syncope or near syncope, a prolonged QT interval, inferolateral repolarization abnormalities, frequent or complex ventricular premature beats, and prolapse of both the anterior and posterior mitral leaflets.

Indications for Mitral Valve Surgery

The only effective treatment of chronic severe MR is surgical repair or replacement of the mitral valve. The indications for surgery are based on natural history data. The medical treatment is limited to the management of heart failure (ACE inhibitors, diuretics) and AF (anticoagulation). Prevention of bacterial endocarditis is no longer recommended.

The optimal timing of corrective surgery is determined by the severity of MR, the presence of symptoms, the LV systolic function, the feasibility of valve repair, the presence of AF, the presence of PHT, and finally the preference and expectations of the patient (see figure 2).20,39

The presence of symptoms is a Class I indication for corrective surgery in patients with severe chronic MR (ESC and ACC/AHA Guidelines: class IB). Delaying surgery until moderate to severe symptoms occur is associated with an increased operative (6.7 % in NYHA IV patients, 2.2 % in NYHA III patients and 1.2 % in NYHA I-II patients) and long-term mortality (48 versus 76%).40 Symptomatic patients with an LVEF below 30% should nonetheless be considered at high operative risk, especially if valve replacement is necessary. In such patients, medical management may be preferable. In patients who do not respond to medical therapy, surgical intervention may be considered if the mitral annular–papillary muscle continuity can be

maintained either by valve repair or replacement with preservation of the chordal apparatus (ESC and ACC/AHA Guidelines : class IIa).

In asymptomatic patients, mitral surgery is indicated when LVEF is < 60 %, LV end-systolic LV diameter is > 40 mm or both (both ESC and ACC/AHA Guidelines: class IB), as in these patients 10-year post-operative survival is at least 20% lower than in patients with a LVEF > 60%.

In asymptomatic patients with a normal LVEF, mitral surgery is also recommended in the presence of AF or PHT.

In asymptomatic patients with severe chronic MR, a normal LV systolic function, no AF or PHT, the ESC guidelines recommend a watchful waiting approach. These patients should be serially followed-up every 6 to 12 months or sooner if symptoms occur, using transthoracic echocardiography. The AHA/ACC guidelines prone a more aggressive approach, recommending to proceed to surgery in the absence of any MR-related complications, if mitral valve repair can be performed with a likelihood of > 90%, and a very low operative mortality. In these patients, mitral repair can be proposed whenever the EROA exceeds 40 mm^2 and the mitral valve is repairable. Recent published propensity score based comparisons between early surgical and watchful waiting strategies support this more aggressive attitude.

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Principles in Restoration of Mitral Valve Incompetence

Patrick PERIER

Mitral valve repair is nowadays a recognized method to surgically treat mitral valve regurgitation. Alain Carpentier has during the past twenty years has worked on the improvement of the surgical techniques, one of the milestones of his work has been the “functional approach”.

Another major breakthrough has been the onset of echocardiography whose role before, during and after the operation has grown to a point where echocardiographers should form tight teams with surgeons. The contributions of echocardiography in mitral valve repair are numerous: for the timing of surgery, for the precise description of the lesions for a preoperative recognition of the etiology, in recognizing intraoperative complications, left ventricular outflow tract obstruction or residual regurgitation, for the follow-up of the patients etc….

Basically, mitral valve regurgitation can be defined as a loss of an efficient surface of coaptation during systole. The aim of mitral valve repair is to restore a good surface of coaptation, thus restoring a competency to the mitral valve.

Mitral valve repair require specific surgical training and skills even in what can be considered the most usual and simple lesion, the prolapse of the posterior leaflet whose standardized treatment is quadrangular resection and plication of the annulus. Nevertheless in a homogeneous cohort of 208 patients, other surgical techniques have been required: a sliding plasty in 98 patients, use of artificial chordae in 5 patients, papillary muscle shortening in 4 patients and removal of posterior annulus calcifications in 5 patients. These techniques resulted in a 100% rate of repair for this lesion for an operative mortality of 2.9% and a 6-year survival of 87%.

There is today little doubt that long time survival after surgery for mitral valve regurgitation is better after mitral valve repair than after mitral valve replacement.

Two groups of patients operated in our institution with mitral valve regurgitation were compared. One group (433 patients) had mitral valve repair and the other (257 patients) mitral valve replacement with Medtronic-Hall prosthesis. The 7-year survival was 74% for the repair group and 58% for the replacement group. This difference was statistically significant. The same differences were observed in subgroups of patients having either isolated mitral valve regurgitation or associated with CABG. It is interesting to note that at 7 years, the reoperation rate was 5% for the repair group and 9% for the replacement group. This underlines the durability and the stability of the repair techniques. The durability over time is dependant of the etiology of the mitral disease as demonstrated by Carpentier's team. A study recently published by this group showed at 25 years a 7% reoperation for the group of degenerative disease and an incidence of 53% of reoperation for the group of rheumatic disease. The trend nowadays is to operate patients with severe mitral valve regurgitation at an early stage when they are not yet symptomatic the goal being to preserve the left ventricular function that might deteriorate unnoticed. Two groups of patients with isolated mitral valve regurgitation have been compared. Seventy-nine patients were non-symptomatic and one hundred eighty four symptomatic. The 7-year survival was 95% for the non-symptomatic group and 75% for the symptomatic group.

In conclusion, improvements in surgical techniques, a better understanding in particular with the help of echocardiography have given mitral valve repair safety, predictability, and durability. Mitral valve repair can be proposed to non-symptomatic to prevent left ventricular dysfunction.

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Ischemic Mitral Regurgitation: Pre-operative Assessment

Jean-Louis Vanoverschelde

Evaluation of patients with ischemic MR requires a multi-disciplinary and often a multi-modality approach.

Echocardiography

Echocardiography is essential for establishing the presence of MR, quantitating its severity and hemodynamic consequences, and ascertain its ischemic origin. As for non-ischaemic forms of MR, grading of the severity of ischemic MR requires an integrated approach that includes assessment of valve morphology, the size of the regurgitant jet in the LA, the proximal regurgitant jet width or vena contracta, the pulmonary venous flow pattern and the measurement of the effective regurgitant orifice area (EROA) and the regurgitant volume (RV).

Quantitative parameters of MR severity can be obtained using either the PISA or the Doppler methods. Caution should nonetheless be exercised when using the PISA method for assessing MR severity. First, recent studies have shown that the shape of the flow convergence zone is rarely hemispherical in patients with ischemic MR, which frequently leads to underestimation of the EROA and the RV by this method. Second, the severity of ischemic MR greatly varies during systole, being the greatest at the beginning and end of systole and least in mid-systole, when the LV pressure is maximal, LV volume is reduced, and the mitral leaflets are pushed back into the annular plane. This phenomenon has important implications with respect to the use of EROA as an index of MR, implying that this should be averaged through systole. The use of volumetric methods for calculating regurgitant volume may thus preferable in patients with ischemic MR.

Echocardiography is also useful to evaluate the characteristics of the LV, particularly its volumes, its sphericity index, its ejection fraction, its diastolic function, and the

distribution of its wall motion abnormalities. It also allows making several anatomic measurements that reflect the pathophysiology of ischemic MR, including the tenting area, the leaflet angles, the coaptation depth, the bending distance and the leaflet length. These measurements should be made in the parasternal long-axis view in mid-systole. In normal individuals, the coaptation depth and the tenting area do not exceed 0.6 cm and 1 cm^2, respectively. Greater degrees of morphologic disturbance have been shown to be predictive of the likelihood of MR persistence following mitral annuloplasty, with the optimal cut-offs for distinguishing patients with persistent MR being a coaptation depth > 0.6 cm, a tenting area of > 2.5 cm^2, or a posterior leaflet angle >45°. Recently, the tenting volume derived from 3D-echocardiography has been shown to be a better index of mitral valve remodeling than the 2D tenting area.

Stress echocardiography

Exercise echocardiography can be used to evaluate the dynamic nature of ischemic MR. Changes in EROA exceeding 13 mm^2 have been shown to carry adverse prognostic implications and to predict the occurrence of both heart failure and death. Dobutamine echocardiography can also be used to evaluate myocardial viability. The absence of myocardial viability or contractile reserve seems to be an independent predictor of a poor outcome in patients undergoing mitral annuloplasty. On the other hand, reliable improvement in moderate ischemic MR by isolated CABG surgery can be observed in patients with concomitant presence of viable myocardium and absence of dyssynchrony between papillary muscles.

Magnetic resonance imaging

Cardiac magnetic resonance imaging (cMRI) is best suited to evaluate LV function and remodeling. It also permits quantification of both the spatial and transmural extent of infarction. In patients with severe ischemic mitral regurgitation, the severity of posterior papillary muscle region scarring correlates with decreased segmental wall motion and mitral regurgitation early after coronary revascularization and

annuloplasty. Routinely assessing scar burden may identify patients for whom annuloplasty alone is insufficient to eliminate mitral regurgitation.

Coronary angiography

The role of coronary angiography is mainly to assess the extent and severity of coronary artery disease and to evaluate the appropriateness of vessels for percutaneous or surgical revascularization.

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Surgical Options for Ischaemic Mitral Regurgitation

Chris Munsch

The pathophysiology of IMR is complex, and it therefore is no surprise that the strategy for surgical management is not always straightforward.

Conventionally the surgical options have been revascularisation either alone, or in combination with either repair or replacement.

Revascularisation alone

Whilst there are some reports that revascularisation alone can be effective in reducing IMR, this would only seem to apply to mild IMR in patients with otherwise good LV function. The advantage of this approach is that the operative risk should be reduced; although the data supporting this premise do not necessarily reflect current surgical outcomes. Preoperative dobutamine stress echo can be used to help define this group of patients. Intra-operative testing is less reliable and we have largely abandoned its use.

Revascularisation with repair

Given the poor long term prognosis of uncorrected IMR, most authorities would recommend that even mild to moderate IMR should be corrected at the time of CABG. The usual, straightforward, strategy is to perform an undersized restrictive annuloplasty by implanting a ring two sizes smaller than the measured intertrigonal distance. This approach was popularised by Bolling, although his original series was a mixture of cardiomyopathies and not entirely IMR patients. Retrospective studies have demonstrated 5 year survival after annuloplasty of generally around 50-60%, whilst others have shown residual or recurrent regurgitation in up to 30% of patients. In addition there is a significant incidence of functional mitral stenosis. Closer analysis of the mechanics and the pathophysiology of IMR have revealed that subvalvar tethering and asymmetrical deformation of the mitral valve contribute to

poorer long term outcomes. To address these shortcomings a more considered approach to mitral reconstruction is required, including the use of 3D echo and measurement of tenting areas, patch enlargement of the posterior leaflet, division of tethering cords and the use of geometrically tailored annuloplasty rings. Just banging in a small ring, it seems, will no longer do!

Revascularisation with replacement

Given the potential pitfalls with reconstructive surgery, a strong argument can be made for bioprosthetic valve replacement with subvalvar preservation, and would be considered a reasonable approach for the surgeon who is not familiar with the concepts and techniques of mitral reconstruction. There have been no published randomised studies comparing repair with replacement, and there is no convincing evidence that repair is superior to replacement. There has been at least one meta-analysis of retrospective comparisons which showed that repair is associated with significantly better short and long term outcomes, although there is the possibility of publication bias in favour of repair.

Other strategies

Other operative strategies have been described or are being developed to treat IMR. Papillary muscle repositioning, usually in association with ventricular reverse remodelling procedures has shown early promise. The edge to edge technique is generally unsuccessful because it fails to address the primary lesions of leaflet tethering and annular dilatation. And of course the cardiologists are exploring percutaneous solutions with coronary sinus annular constraint devices, which gives them something to do, and something to talk about but actually allow partially addresses the problem.

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Tricuspid Valve Regurgitation – Indications for Surgery and Operative Options

Patrick PERIER

The tricuspid valve remains an enigma and tricuspid valve dysfunctions are too often ignored and untreated.

Most of the time, tricuspid regurgitation is a finding in patients with advanced mitral valve disease. The tricuspid regurgitation is functional secondary to pulmonary hypertension and right ventricular dilatation leading to a dilatation of the tricuspid annulus. Patients that require a simultaneous tricuspid valve surgery have to be clearly identified, which remains a difficult task. There are no clear parameters to help for the decision making. It seems that nowadays the most reliable criteria is the dilatation of the annulus as measured by echocardiography or during the surgical exploration. Many surgical techniques have been described to narrow the dilated tricuspid annulus, sutures, commissuroplasty etc… the basic principle of the Carpentier annuloplasty ring is to reshape and to restore normal proportions to the various components of the tricuspid valve. All the technical refinements of valve reconstructive surgery may be needed to reconstruct a good surface of coaptation, and the new developments, artificial chordae; pericardial substitutes have considerably reduced the indications for tricuspid valve replacement.

More rarely tricuspid valve disease is isolated as can be seen in congenital malformation, after bacterial endocarditis or traumatism.

Valve repair is the technique of choice to surgically treat dysfunctions of the tricuspid valve. The prognosis is totally different if the tricuspid valve disease is isolated or associated with left-sided valve dysfunctions being then the consequence of right ventricular dysfunction. Echocardiography is the method of choice to study the tricuspid valve and to select the patients who require tricuspid surgery.

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Surgery for the tricuspid valve

The tricuspid valve lies at the entrance of the heart between the right atrium and ventricle. Symptoms of TV disease are primarily extra cardiac and insidious, pathology of the TV rarely occurs in isolation.

Anatomy

The TV is a trileaflet valve composed of anterior, posterior and septal leaflets. Both the anterior and posterior leaflets are attached to the RV free wall and the septal leaflet into the base of the interventricular septum. As with the mitral valve, the TV is supported by a subvalvular apparatus consisting of papillary muscles and cords. There is no true fibrous TV annulus which in part explains the large change in size of the TV throughout the cardiac cycle. It forms the shape of a hyperbolic parabolic (as does the mitral valve) and this needs to be borne in mind when choosing how best to repair the valve.

Classification of TV disease

TV disease is commonly classified as either organic or functional.

Organic disease – implies that there is abnormality of the subvalvular apparatus such as following rheumatic fever, infective endocarditis, carcinoid etc

Functional disease – is more common and regurgitation occurs in the presence of an apparently normal TV apparatus. This occurs due to annulus dilatation and dysfunction. Due to the lack of a fibrous annulus, dilatation of the RV leads to functional regurgitation.

Quantification of TR by echocardiography is made difficult by the non-circular characteristics of the annulus, therefore slight changes in orientation of the echo probe may lead to inaccurate measurements. TR is considered to be severe and warrant surgical intervention with an annulus diameter >40mm, vena contracta > 7mm or PISA > 9mm.

Tricuspid valve repair

Dreyfus in 2005 reported on the criteria for surgical repair in 311 patients undergoing concomitant MV surgery. The TV was measured between the antero-septal and antero-posterior commissure (as this is the area of greatest dilatation). If this annular measure was greater than 70mm, patients underwent remodelling annuloplasty. There was no difference in in-hospital mortality between the two groups or mid-term survival. However, in those patients not receiving an annuloplasty, the degree of TR assessed by echocardiography at follow-up had progressed and more patients were in NYHA classes II-IV (majority of patients following TV repair NYHA I, none in III/IV).

Van de Vaire, compared two cohorts, from 2002 and 2004 undergoing MV surgery, in the 2002 group, only patients with severe TR underwent TV repair and in 2004, patients with both severe TR and/or TV annular dimension > 40mm underwent repair.

In the 2002 cohort analysis of the group of patients without echocardiographically severe TR but TV dilatation demonstrated progressive annular dilatation, worsening TR and RV dilatation. IN the 2004 cohort, those patients with annular dilatation without severe TR exhibited reverse remodelling of the RV and reduced TR.

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Surgical treatment for AF

AF is the most common rhythm disturbance and is present in ~2% of the population with an age related increase in incidence.

Previously AF was considered to be relatively benign, however, there is a significant morbidity and mortality related to AF this is mainly due to

- Loss of co-ordination of AV contraction
- Stasis of blood flow within the LA leading to thromboembolic complications such as stroke. In ~20-30% of acute strokes, patients are in AF.

AF is also an independent predictor of mortality.

Mechanisms of AF

Mechanisms responsible for the initiation and propagation of AF include focal mechanisms and multiple wavelet hypotheses. Mechanisms of focal activity may involve both triggered activity and re-entry. Due to shorter refractory periods and abrupt changes in myocyte fibre orientation, the pulmonary veins have a strong potential to initiate AF. In Paroxysmal AF high frequency sites are mostly located at the LA/PV junction and ablation leads to prolongation of the AF cycle length and conversion to sinus rhythm. In persistent AF, sites are located throughout the atrium making ablation and return to sinus rhythm more difficult. Multiple wavelets perpetuate AF by continuous conduction of several independent wavefronts through the atrial muscle in a chaotic manner. As long as the number of wavefronts does not fall below a critical level, AF is sustained.

Classification of AF

ESC guidelines identify 5 types of AF

1. First diagnosed AF – every patient presenting with AF regardless of history

2. Paroxysmal AF – self-terminating usually within 48 hours but up to 7 days (after 48h risk of spontaneous conversion is low)
3. Persistent AF – Episode lasting longer than 7 days or requiring pharmacotherapy or DCCV to revert to sinus rhythm
4. Long-standing persistent – AF > 1 year when a rhythm control strategy is adopted
5. Permanent AF – Presence of AF is accepted and rhythm control not pursued

Medical management

Rate versus rhythm control with medical therapy has not been demonstrated to reduce all cause mortality, stroke rate or quality of life (AFFIRM).

Surgical management

Surgical therapy for AF is directed at altering the geometrical and anatomical changes that occur and act as substrates for AF. The Cox-Maze procedure was designed to interrupt macro-re-entrant circuits thought to be responsible for AF. Incisions were placed across the atria allowing the SA node to direct a sinus impulse through both atria, allowing all of the atrial myocardium to be activated and therefore preserving atrial transport.

This procedure has gone through a number of iterations and refinements to reduce the incidence of pacemaker dependence and in line with new technologies.

The traditional cut-and-sew Cox-Maze procedure has been replaced by performing linear lines of ablation using technologies such as radiofrequency, high intensity focused ultrasound and cryoablation.

Ablation technology needs to fulfil a number of criteria

1. The ability to make a transmural lesion from either the epicardial/endocardial surface

2. Safe to use in the vicinity of other anatomical structures e.g. coronary sinus, valves, oesophagus
3. Should make the surgery simpler than the old cut-and-sew technique
4. Ability to be used in a non-invasive procedure

Cryoablation – unlike the other energy sources destroys tissue by freezing rather than heating. Achieves transmurality on the arrested heart and is safe around valvular tissue. Difficult on beating heart, may not be transmural in this setting, also coagulates blood and potential for embolisation in the beating heart.

High intensity focused ultrasound – ablates tissue by mechanical hyperthermia. Targeted thermal coagulation, affected less by heat sink.

Radiofrequency ablation – one of the first energy sources to be used. Can be either uni- or bipolar. Problems with achieving transmurality using unipolar devices. Bipolar devices well documented to produce transmurality, however, tissue must be clamped in the jaws of the device, potentially limiting the lesion set and requiring additional technology to perform a complete Cox-Maze lesion set.

Surgery for AF

The ESC/EACTS guidelines have 3 recommendations for surgical ablation of AF

1. Considered in symptomatic patients undergoing cardiac surgery (IIa/A)
2. May be performed in asymptomatic patients undergoing surgery if feasible with minimal risk (IIb/C)
3. Minimally invasive lone AF surgery is feasible in patients with symptomatic AF after failure of catheter ablation (IIb/C)

Cox-Maze procedure

The first two iterations of the Cox-Maze (CM) procedure were abandoned due to the high incidence of requirement for PPM implantation. The final version CM III had symptomatic freedom from AF of 97% at a mean 5.5 year follow-up, however,

documentation of rhythm was not stringent in all cases and involved in some telephone/questionnaire follow-up with only single ECG not more rigorous Holter monitoring. The latest iteration of the procedure the CM IV was introduced in 2002 and replaced cut-and-sew with linear ablation lines.

CM IV lesion set

Beating heart

1. Bipolar ablation to cuffs of atrial tissue surrounding right and left pulmonary veins
2. Right atrial free wall lesion via a small atriotomy with a purse string at the base of RA appendage.
3. A 2cm gap is left between this and a vertical atriotomy from the crista terminalis towards the AV groove. The superior aspect of this incision is connected to the tricuspid valve annulus at the 2 o'clock position (unipolar).
4. From the purse string at the base of the RA appendage a lesion is created across the RA endocardium to the 10 o'clock position of the tricuspid valve annulus.
5. Using the bipolar device a line is made superiorly to the SVC and down to the IVC from the vertical atriotomy.

Arrested heart

6. The rest of the left sided lesions are performed though the left atrium on an arrested heart
7. The atriotomy is extended around the right inferior pulmonary vein and superiorly to the dome of the LA, connecting to the right PV isolation lesion set.
8. Lesion then created from inferior aspect of the left atriotomy to the left PV isolation lesion set.

9. With the next lesion extending from the superior aspect of the left atriotomy into the left superior pulmonary vein
10. A lesion is then created from the inferior aspect of the left atriotomy to the MV annulus.
11. The left atrial appendage is amputated and a final lesion is created into the left superior pulmonary vein.

Reporting results

Reporting of results for AF surgery needs to be rigorous. Current consensus statements propose that success for such therapy is defined as freedom from AF, atrial tachyarrhythmia or atrial flutter off of anti-arrhythmic drugs.

Results of the CM IV

Prospective data on 282 patients who underwent the CM IV procedure were collected. 42% had paroxysmal and 58% persistent AF, with a median duration of AF of 3.7 years. Of this group, 66% were undergoing concomitant procedures. Operative mortality was 2% in the concomitant and 1% in the stand-alone group. Early post-operative arrhythmias occurred in 53% of patients and PPM implantation was required in 9%.

At 3, 6 and 12 months, freedom form atrial tachyarrhythmia (ATA) was 89%, 93% and 89%.

Freedom from both ATAs and ant-arrhythmic drugs was 63%, 79% and 78%.

On multivariable analysis, increasing LA size, failure to anatomically isolate the posterior left atrium (non-box lesion set) and incidence of early post-op ATA were independent predictors of failure. Duration of post-operative AF was not an indicator of failure in this analysis.

Further reading

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