NOTES

Supplementary notes for wet lab demonstrations - 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^{o}C. Bypass was discontinued after a naso-pharyngeal temperature of 36^{o}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.

Bonus questions:

Which British monarch died of acute type A dissection ? ***What is the Euroscore for this patient ?***

HISTORY OF CORONARY REVASCULARISATION

(the old trials and Acute Coronary Syndrome)

Mr. M. Dalrymple-Hay – Derriford, Plymouth.

CHOICE OF CONDUIT INCLUDING ENDOSCOPIC HARVESTING

Mr. J. Zacharias – Lancashire Cardiac Centre, Blackpool.

THE IMPACT OF CO-MORBIDITIES ON OUTCOME

Mr. C. Lloyd – Derriford Hospital, Plymouth.

Mortality after cardiac surgery has been extensively studied in an attempt to identify patients at risk for cardiac surgery and to provide a platform for comparing data between centres. Variables are typically defined as (1) preoperative – either predetermined such as age and gender, or acquired such as diabetes, left ventricular function, renal disease or (2) Operative – such as urgency, previous cardiac operations or type of operation.

Morbidity after cardiac surgery can also be modelled with preoperative and operative variables. The most significant outcomes are for end organ damage such as stroke, renal impairment, long term ventilator support or gastrointestinal complications.

The first widely used model for predicting the impact of comorbidities on survival outcome was the Parsonnet Score in 1989 derived from North American data. While initially the model accurately reflected mortality, as results improved the model began to overpredict mortality. In the United States this has now been largely replaced by the STS (Society of Thoracic Surgeons) score.
In Europe the EuroSCORE was developed in the late 1990's from European data and initially introduced in addative format which allowed quick, bedside calculation. While the model reasonably predicted mortality for low risk groups, it was unreliable for high risk groups. A further refinement using regression co-efficients was published in 2003 and while initially this appeared more accurate, over time this too has shown to overpredict and prove unreliable in certain operative groups.

Analysis performed by the SCTS has shown that the weight of co-morbidities differs for different cardiac procedures and therefore there may not be a single model for all cardiac surgery.

A natural extension of developing a model to predict outcome has been the assessment of surgical performance, and in the UK the publishing of surgeon specific mortality

References:

Parsonnet V, Dean D, Bernstein AD. A method of uniform stratification of risk for evaluating the results of surgery in acquired adult heart disease. *Circulation* 1989;79:I3-I12.

Nashef SA, Roques F, Michel P, Gauducheau E, Lemeshow S, Salamon R. European system for cardiac operative risk evaluation (EuroSCORE). Eur J Cardiothorac Surg. 1999 Jul;16(1):9-13.

Wendt D, Osswald BR, Kayser K, Thielmann M, Tossios P, Massoudy P, Kamler M, Jakob H. Society of Thoracic Surgeons score is superior to the EuroSCORE determining mortality in high risk patients undergoing isolated aortic valve replacement. Ann Thorac Surg. 2009 Aug;88(2):468-74

Dewey TM, Brown D, Ryan WH, Herbert MA, Prince SL, Mack MJ. Reliability of risk algorithms in predicting early and late operative outcomes in high-risk patients undergoing aortic valve replacement. J Thorac Cardiovasc Surg. 2008 Jan;135(1):180-7

Michel P, Roques F, Nashef SA; EuroSCORE Project Group. Logistic or additive EuroSCORE for high-risk patients? Eur J Cardiothorac Surg. 2003 May;23(5):684-7

Bhatti F, Grayson AD, Grotte G, Fabri BM, Au J, Jones M, Bridgewater B; North West Quality Improvement Programme in Cardiac Interventions. The logistic EuroSCORE in cardiac surgery: how well does it predict operative risk? Heart 2006 Dec;92(12):1817-20

BLOOD CONSERVATION IN CARDIAC SURGERY

Mr. G. Murphy – Bristol Royal Infirmary.

Rationale

- Blood transfusion and resternotomy for bleeding are both independently associated with increased morbidity (MI, stroke, acute kidney injury, infection), mortality (30 days, 1 year and 5 years) and hospitalisation costs (>10% increase in the average cost of a procedure per unit transfused, or 100% increase if reopened for bleeding).
- Why these factors should increase morbidity and mortality is poorly understood. They may increase the severity of the inflammatory response or directly cause organ injury.
- Health economists predict blood shortages (and therefore escalating costs) over the next 20 years
- There is considerable variability in the use of blood conservation techniques and in rates of bleeding and transfusion.

Objective

Does reducing exposure to allogenic blood products or excessive bleeding improve patient outcomes?

Evidence

The evidence to support blood conservation has been summarised in a recent set of guidelines (1). These really serve to highlight how poor the evidence base is however there is reasonable evidence to support the following blood conservation paradigm.

Preoperative

- Identification of patients at risk
- Treatment of preoperative anaemia and coagulopathy
- Appropriately timed cessation of preoperative antiplatelet agents

Intraoperative

- Good surgical technique/ zero tolerance for re-sternotomy for bleeding
- Cardiopulmonary bypass; low prime volume, retrograde priming, MECC, closed vs. open circuits, circuit coating
- Optimisation of physiological anaemia tolerance; NIRS based algorithms
- Pharmacological strategies; Tranexamic Acid, Aprotinin, platelet anaesthesia
- Mechanical cell salvage and autotransfusion
- Normovolaemic haemodilution and autotransfusion
- Point of care tests for early diagnosis and directed treatment of coagulopathy with Platelets, FFP, Cryoprecipitate, Factor VII etc.

Postoperative

- Restrictive transfusion thresholds
- Postoperative cell salvage
- Appropriate use of blood volume replacement solutions

Summary and Conclusion

Current thinking refers to adoption of multiple interventions as part of ‘Patient Blood Management’. What the optimum cost effective combination may be, in terms of reducing patient morbidity and mortality is unclear however and is the subject of much ongoing research.

Reference

Ferraris VA, Ferraris SP, Saha SP, Hessel EA, 2nd, Haan CK, Royston BD, Bridges CR, Higgins RS, Despotis G, Brown JR et al: Perioperative blood transfusion and blood conservation in cardiac surgery: the Society of Thoracic Surgeons and The Society of Cardiovascular Anesthesiologists clinical practice guideline. Ann Thorac Surg 2007, 83(5 Suppl):S27-86.

FUNCTIONAL ANATOMY OF THE AORTIC VALVE

Mr. I. Wilson – Queen Elizabeth Hospital, Birmingham.

- **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 form 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.

- **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.

NOTES

AETIOLOGY AND ASSESSMENT OF AORTIC VALVE DISEASE INDICATION INCLUDING THE ASYMPTOMATIC PATIENT THE LOW GRADIENT POOR LV SCENARIO

Professor J-L Vanovershelde – Louvain University, Brussels.

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 measured using 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.

WHEN TO REPLACE THE AORTIC VALVE IN PATIENTS UNDERGOING CABG

Mr. M. Dalrymple-Hay - Derriford, Plymouth.

THE CHOICE OF PROSTHESIS / NEW ANTICOAGULANTS

Professor M. Redmond - Our Lady's Hospital, Dublin.

The advantageous durability of mechanical valves is offset by the risk of thromboembolism and need for life-long anticoagulation with its associated risk of bleeding. Bioprosthetic valves do not require long-term anticoagulation, but carry the risk of structural failure and reoperation.

Two historic prospective randomised clinical trials (Edinburgh Heart Valve Trial 1975-79 and VA Cooperative Study on Valvular Heart Disease 1979-82) compared outcomes of valve replacement with bioprosthetic (first generation porcine heterograft) versus mechanical (Bjork-Shiley tilting disc) valves; they both confirmed the increased risk of bleeding with mechanical valves and increased risk of structural failure and reoperation with tissue valves. The Edinburgh trial demonstrated a small survival advantage with mechanical AVR. The ability to extrapolate these data to decisions made in modern practice is limited as these valves are now obsolete.

Based on these trials, the ACC/AHA earlier guidelines heavily weighted patient age in deciding between mechanical and tissue valves. Without randomised trials comparing present generation valves, the revised 2006 ACC/AHA and the 2007 ESC guidelines rely on level C evidence for recommendations; in ACC/AHA guidelines, class I recommendations for valve selection are limited to the use of mechanical prosthesis in the setting of an existing well-functioning mechanical valve, and the use of a bioprosthetic valve in the setting of a patients unwillingness to take warfarin or of a major contraindication to its use. However, both sets of guidelines recommend allowance for preference of the informed patient in decision for valve choice, (class IIa in 2006 ACC/AHA, class I in 2007 ESC).

A meta-analysis of 32 studies evaluating mortality from 15 mechanical and 23 tissue valves series found no difference in risk-corrected mortality between mechanical and bioprosthetic valves regardless of age, while another retrospective series reported that age and not valve type was predictive of valve-related mortality, that reoperation was higher after tissue AVR only for patients >60yrs, but combined valve-related morbidity was higher after mechanical AVR for all patients >40yrs.

Advances in tissue fixation and anticalcification treatments in second and third generation bioprostheses have supported the current trend towards the increased use of tissue rather than mechanical valves and the use of tissue valves in progressively younger patients. The Carpentier-Edwards pericardial aortic valve has a 94% freedom from structural deterioration at 10yrs and 77% at 15yrs in patients with a mean age of 65yrs, with a 10% chance that a 65yo patient would require reoperation before 80yrs. Third generation valves appear to be even more durable. Furthermore, improved surgical techniques have rendered redo valve operations safer.

The main determinants of valve selection are individual patient life expectancy, patient's tolerance to the need for repeat valve replacement and the use of oral anticoagulants with its associated changes in life-style. (see figure 1).

Because mortality risk is similar after mechanical and bioprosthetic AVR, a 50 yr old patient should expect at least 1 reoperation after tissue AVR, but overall valve-related morbidity is far higher after mechanical AVR; lifestyle adjustments are more likely after mechanical AVR because of long-term anticoagulation with warfarin. Thus some patients will choose to avoid reoperation whereas others will opt to minimise lifestyle changes and limit risk of valve-related morbidity while accepting the likelihood of at least 1 reoperation.

Choice of AVR can be influenced by need for growth in children and adolescence, in which case the Pulmonary Autograft is a more appropriate choice. Aortic homografts are a valuable alternative in patients with intractable endocarditis with or without abscess formation.

Recently, potential alternatives to warfarin as long-term oral anticoagulants have emerged. Dabigatran, a direct thrombin inhibitor and rivaroxaba, a direct Factor Xa inhibitor have been evaluated in clinical trials (RE-VOLUTION and REDORDS respectively). These drugs have rapid onset of action, predictable pharmacokinetics, fixed dosing and no requirement for routine blood monitoring. Approval for the limited use of these drugs by NICE and the FDA is pending. Should these drugs prove effective for valve-related anticoagulation, they will have a profound influence on the choice for mechanical prosthesis

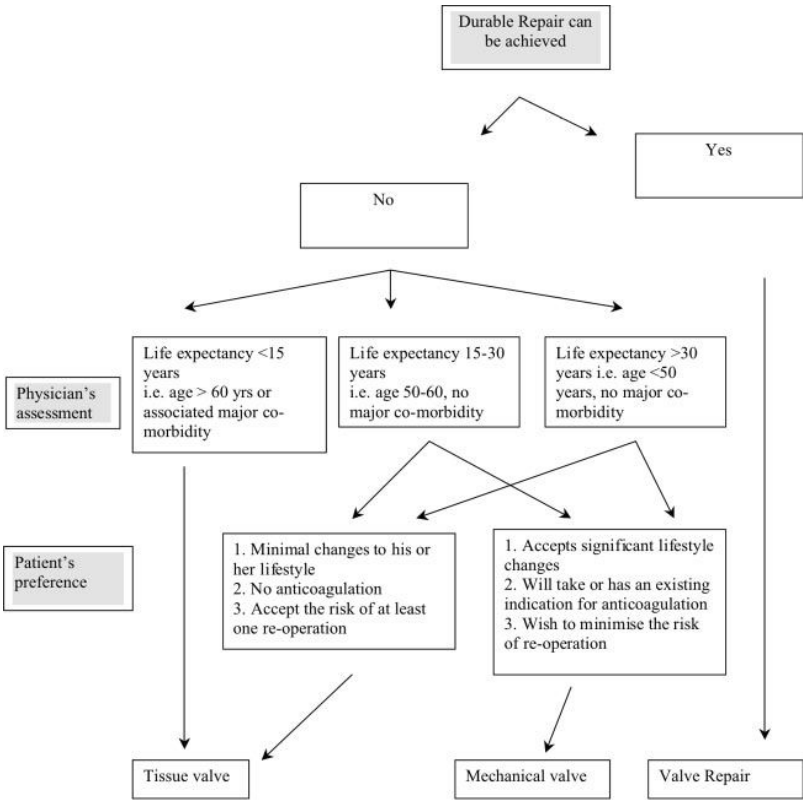


Figure 1

Key References

1. Oxenham H, et al. Twenty year comparison of a Bjork-Shiley mechanical heart valve prosthesis with porcine bioprostheses. *Heart*. 2003;89:715-721.

2. Hammermeister K, et al. Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the Veterans Affairs randomized trial. *J Am Coll Cardiol*. 1998;32:1486-1582.

3. American College of Cardiology, American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2006; 48:e1-e148.

4. Vahanian A, et al. For the Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and ESC Committee for Practice Guidelines. Eur Heart J. 2007;28:230-268.

5. Lund O, et al. Risk-corrected impact of mechanical versus bioprosthetic valves on long-term mortality after aortic valve replacement. J Thorac Cardiovasc Surg. 2006;132:20-26.

6. Chan V, et al. Performance of bioprostheses and mechanical prostheses assessed by composite of valve-related complications to 15 years after aortic valve replacement. J Thorac Cardiovasc Surg. 2006;131:1267-127.

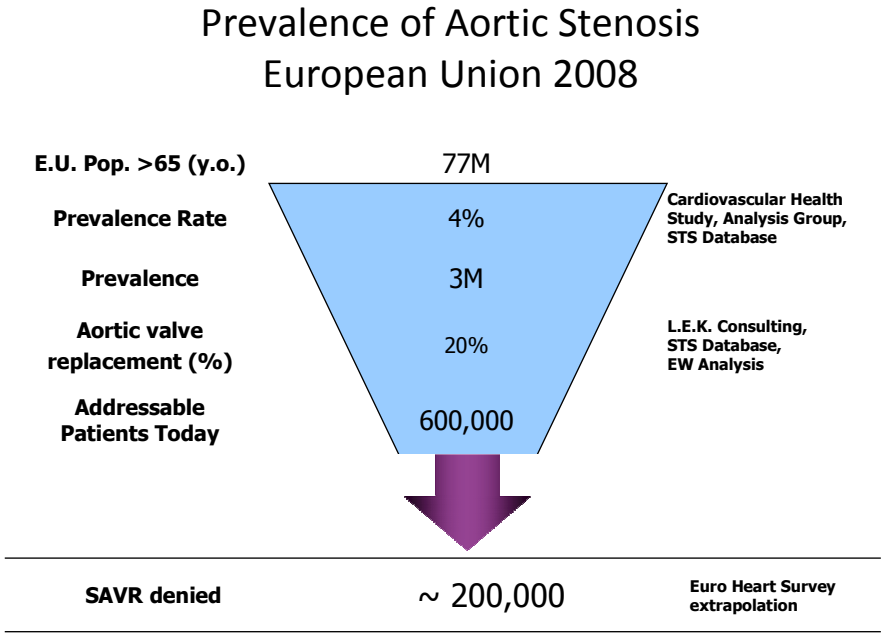
TAVI: TECHNOLOGY AND DIRECTION

DO WE NEED A TRIAL TO COMPARE SURGERY WITH TRANSCATHETER VALVES?

Professor A. P. Kappetein - Erasmus MC, Rotterdam.

Introduction

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 [1, 2] . 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[3]. Furthermore mortality is already substantial in the months following the first symptoms [4]. Both, the ESC and ACC/AHA cardiology societies have endorsed guidelines on valvular heart disease emphasizing the need for surgical aortic valve replacement (SAVR) once symptoms develop or in case of impaired LV function (Level of evidence grade 1)[5, 6]. 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[7]. 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 SAVR and there are 600000 patients with symptomatic AS in Europe alone, this means that hypothetically about 200000 patients would not be considered for intervention[8]. 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.

Alain Cribier pioneered the transcatheter aortic valve implantation (TAVI) technology and reported the first in man experience of TAVI in a patient with symptomatic AS who was deemed inoperable in 2002[9]. Subsequent feasibility studies validated the proof of concept [10, 11] . The Edwards-SAPIEN

valve (Edwards Lifesciences, Irvine, CA, USA) and the Medtronic-Corevalve system (Medtronic Corporation, Minneapolis, MN, USA) are the only two TAVI platforms with CE mark approval since 2007. Numerous single-center and multi-center observational registries followed with dazzling speed suggesting the safety and efficacy of the TAVI technology [12-16] . Especially the 30-day mortality of around 8% in patients with high or prohibitive operative risk appears promising and resembles short-term outcome in high-risk cohorts in the surgical literature[17-22]. The TAVI technology comes with its own specific hurdles and complications [23], not necessarily overlapping with those of SAVR: vascular injury; stroke, cardiac injury such as heart block, coronary obstruction, and cardiac perforation; paravalvular leak; and valve misplacement. The non-uniformity in presenting respective data makes comparison of results from different centers hazardous and impractical[24, 25]. The Valvular Academic Research Consortium, a FDA approved collaboration between academic research organizations and professional societies in the United States and Europe is an initiative to generate a consensus statement on TAVI related definitions aiming to create order and uniformity making data more prone to analysis and comparison.

Technical refinements and commercial entrepreneurship have made the technology accessible to many centers worldwide. This might pose future implications especially in the current era where randomized trials with TAVI are strikingly lacking.

Grossly, there are three types of medical practices: the first is the institution with on-site interventional cardiologic and cardiothoracic activity and with close inter-disciplinary collaboration where interventional cardiologists and cardiothoracic surgeons try to reach a consensus on which patients to select for a specific surgical or interventional treatment strategy [8]. These centers would reasonably respect and adhere to the so-called CE mark labeling indications. Secondly there are centers where interventional cardiologists and cardiothoracic surgeons don't really convene and work as two separate departments. Finally there are practices running an interventional cardiology program without on-site cardiothoracic surgery, estimated to make up 37% of all PCI centers in the European Union. Expectedly, these kinds of organizations without intimate collaboration between cardiothoracic surgeons and interventional cardiologists will look to broaden their interventional activities with an attractive innovation like TAVI. If this kind of explosion and widespread distribution of a new technology is appropriate and well-timed is highly debatable. The flipside would be a worldwide practice being less controllable, potentially clouding the safety and efficacy profile of the procedure. Needless to say that provoked criticism by the medical community and health authorities could jeopardize future reimbursement policies [26, 27].

Regardless, with the compelling data presented by multiple registries and leading centers, randomized clinical trials comparing TAVI with SAVR should be the logical next step. This next step in establishing a new treatment strategy should not be taken for granted as governmental authorities entitled to grant premarket approval to cardiovascular devices are under increased scrutiny and quality control [28].

The PARTNER trial (Placement of AoRTic TraNscathetER Valve Trial) is the first to randomize patients with high or prohibitive operative risk to SAVR, 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 SAVR or TAVI. The trial completed its randomization early 2009. One-year outcome results will be reported in the forthcoming months. By study design, findings will only apply to this highly selected patient cohort representing only a fraction

of the global AS burden.

While anticipating the results of this first randomized trial, over 18000 patients worldwide have been treated with TAVI by May 2010. Inevitably, with increased operator experience and access to the device, physicians will shift their attention to younger patients with a less pronounced operative risk. Similar to what happened in the coronary revascularization arena [29], the blending of surgical and interventional expertise has created unique interdisciplinary dynamics reinforcing these new endeavors and paving the way for a randomized trial comparing TAVI with SAVR in a surgical moderate to high-risk patient population.
In this spirit the SURTAVI trial (SURgical and Transcatheter Aortic Valve Implantation) is conceived. The interdisciplinary approach and consensus of the so-called Heart Team (the cardiothoracic surgeon, interventional cardiologist and other treating physicians if necessary) is crucial. This aspect of decision making cannot be over-emphasized and is essential for the quality of current medical practice in general and any planned randomized trial of TAVI versus SAVR in particular. The upcoming VARC document on TAVI definitions and the accumulating TAVI expertise in Europe has created a unique momentum for such a European randomized initiative.

To summarize, for a new technology to be accepted as a new asset in the armamentarium for treating symptomatic AS several essential questions need to be answered: does the technology work? Which patients are likely to benefit (patient selection)? How does this new strategy compare with the alternatives? And what's the cost of the intervention? The proof of concept has been validated. The innovative less invasive transcatheter strategy should be at least as effective but safer than traditional SAVR or have proof of superiority for both safety and efficacy compared to medical therapy.
Theoretical benefits of these transcatheter instrumentations in a beating heart avoiding the need of musculoskeletal incisions, cardioplegic arrest, aortic cross clamping, full cardiopulmonary bypass (including subsequent LV septal motion abnormality) seem evident. Ultimately the cost-effectiveness will determine whether the new treatment strategy is a valid option to be considered for reimbursement by governmental health institutions. The price–tag of the device is essential and will ideally cover the company's capital investment made during research and development. The cost-effectiveness relationship will only become favourable once competitive companies enter the market and introduce alternative devices at lower prices.

1.1 Surtavi criteria - Patient characterization

Risk assessment is essential in the complex strategy to approach patients with valvular heart disease in general and AS in particular. Various risk models primarily focusing on short-term mortality have been validated for AS patients[30-38]. Some were initially derived from a broader patient population undergoing any type of cardiac surgery, others were more explicitly tailored to patients with AS. Most notably, contemporary scoring models tend to be consistent in lower risk patients but diverge with increasing risk profile. This can be partly explained by the fact that these models were extracted from large databases where the average patient risk is fairly low. Therefore such models are less well validated for higher risk patients and expectedly perform less well in the "outlier" population currently considered for TAVI[39].
An in-depth reappraisal of existing scoring models reveals some concordant risk factors (e.g. age, gender…) but also established risk factors that are clearly missing (e.g. mediastinal radiation, porcelain aorta and frailty)[20, 40, 41]. Furthermore definitions of individual components are not uniform and do not correspond to current suggested guidelines/definitions by respective professional societies.

The goal of the SURTAVI criteria is to establish a new comprehensive yet transparent and updated scoring model to identify and characterize the contemporary patient population considered for aortic valve therapy. Subsequently it can be used for benchmark testing to compare institutional, operator or device performance and eventually be validated for its discriminating performance to determine which patients would fare better with TAVI or SAVR and vice versa, becoming a tool for patient counseling on procedural risk.

The SURTAVI criteria consist of baseline clinical characteristics and predictive features from the standard preoperative work up, notably ECG, biochemistry, echocardiography and Multislice Computed Tomography (MSCT). The rationale to select an item in this new scoring model is based on a critical reappraisal of prior risk scoring models and clinical judgment/expert opinion.

We first identified those components recurring in the previously published scoring models and looked for updated definitions by international professional societies. We then added missing risk factors, identified in the literature, which we felt, were essential. We grouped the components in 5 headings (demographic, cardiac, non cardiac, imaging, biochemistry). The weighting process followed a pragmatic and simplified approach based on data from established scoring models (e.g. Odds Ratios from STS, EuroSCORE…) and subjective appreciation of impact. Each item was granted a numerical score from 0-4. Initially discrimination was deemed more important than calibration as the proposed model could be recalibrated (adjusting the weights to the studied population) in the future.

Patients at intermediate risk (EuroScore of ≥6 (e.g. 75 yrs+ and at least 1 co-morbidity; 80 yrs+) and ≤30) will be screened for Aortic will be screened by the local Heart Team.

Surgical risk algorithms (Logistic EuroScore and Society of Thoracic Surgeons – Predicted Risk of Mortality (STS-PROM)), and frailty scores (Lee score and Charlson scores) must be recorded to guide but will not dictate patient allocation. The final decision of the local Heart Team will be documented and signed on a Heart Team Decision Form.

The primary objective of the SURTAVI trial is to assess whether in patients with symptomatic severe aortic stenosis and at intermediate risk, Transcatheter Aortic Valve Implantation (TAVI) is non-inferior to Surgical Aortic Valve Replacement (SAVR) with respect to the event free survival time of the combined endpoint of all-cause mortality and stroke at a median follow-up duration of 2 years.
Secondary Objectives is to compare patients with symptomatic severe aortic stenosis and at intermediate risk treated with Transcatheter Aortic Valve Implantation (TAVI) to Surgical Aortic Valve Replacement (SAVR) with respect to quality of life, clinical benefit, and health economics.

REFERENCES

1. Iung, B., et al., *A prospective survey of patients with valvular heart disease in Europe: The Euro Heart Survey on Valvular Heart Disease.* Eur Heart J, 2003. **24**(13): p. 1231-43.
2. Supino, P.G., et al., *The epidemiology of valvular heart disease: a growing public health problem.* Heart Fail Clin, 2006. **2**(4): p. 379-93.
3. Ross, J., Jr. and E. Braunwald, *Aortic stenosis.* Circulation, 1968. **38**(1 Suppl): p. 61-7.
4. Rosenhek, R., et al., *Natural history of very severe aortic stenosis.* Circulation. **121**(1): p. 151-6.

5. Bonow, R.O., et al., *2008 Focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons.* Circulation, 2008. **118**(15): p. e523-661.
6. Vahanian, A., et al., *Guidelines on the management of valvular heart disease: The Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology.* Eur Heart J, 2007. **28**(2): p. 230-68.
7. Iung, B., et al., *Decision-making in elderly patients with severe aortic stenosis: why are so many denied surgery?* Eur Heart J, 2005. **26**(24): p. 2714-20.
8. Serruys, P.W., *Keynote address--EuroPCR 2008, Barcelona, May 14th, 2008. Transcatheter aortic valve implantation: state of the art.* EuroIntervention, 2009. **4**(5): p. 558-65.
9. Cribier, A., et al., *Percutaneous transcatheter implantation of an aortic valve prosthesis for calcific aortic stenosis: first human case description.* Circulation, 2002. **106**(24): p. 3006-8.
10. Webb, J.G., et al., *Percutaneous transarterial aortic valve replacement in selected high-risk patients with aortic stenosis.* Circulation, 2007. **116**(7): p. 755-63.
11. Grube, E., et al., *Percutaneous implantation of the CoreValve self-expanding valve prosthesis in high-risk patients with aortic valve disease: the Siegburg first-in-man study.* Circulation, 2006. **114**(15): p. 1616-24.
12. Piazza, N., et al., *Procedural and 30-day outcomes following transcatheter aortic valve implantation using the third generation (18 Fr) corevalve revalving system: results from the multicentre, expanded evaluation registry 1-year following CE mark approval.* EuroIntervention, 2008. **4**(2): p. 242-9.
13. Bleiziffer, S., et al., *Survival after transapical and transfemoral aortic valve implantation: talking about two different patient populations.* J Thorac Cardiovasc Surg, 2009. **138**(5): p. 1073-80.
14. Webb, J.G., et al., *Transcatheter aortic valve implantation: impact on clinical and valve-related outcomes.* Circulation, 2009. **119**(23): p. 3009-16.
15. Thomas, M., et al., *Thirty-Day Results of the SAPIEN Aortic Bioprosthesis European Outcome (SOURCE) Registry. A European Registry of Transcatheter Aortic Valve Implantation Using the Edwards SAPIEN Valve.* Circulation.
16. Gerckens, U., et al., *Procedural and mid-term results in patients with aortic stenosis treated with implantation of 2 (in-series) CoreValve prostheses in 1 procedure.* JACC Cardiovasc Interv. **3**(2): p. 244-50.
17. Brown, J.M., et al., *Isolated aortic valve replacement in North America comprising 108,687 patients in 10 years: changes in risks, valve types, and outcomes in the Society of Thoracic Surgeons National Database.* J Thorac Cardiovasc Surg, 2009. **137**(1): p. 82-90.
18. Halkos, M.E., et al., *Aortic valve replacement for aortic stenosis in patients with left ventricular dysfunction.* Ann Thorac Surg, 2009. **88**(3): p. 746-51.
19. Maslow, A., et al., *Aortic valve replacement with or without coronary artery bypass graft surgery: the risk of surgery in patients > or =80 years old.* J Cardiothorac Vasc Anesth. **24**(1): p. 18-24.
20. Chang, A.S., et al., *Cardiac surgery after mediastinal radiation: extent of exposure influences outcome.* J Thorac Cardiovasc Surg, 2007. **133**(2): p. 404-13.
21. Walther, T., et al., *Transapical aortic valve implantation in 100 consecutive patients: comparison to propensity-matched conventional aortic valve replacement.* Eur Heart J, 2010. **31**(11): p. 1398-403.

22. Piazza, N., et al., *A comparison of patient characteristics and 30-day mortality outcomes after transcatheter aortic valve implantation and surgical aortic valve replacement for the treatment of aortic stenosis: a two-centre study.* EuroIntervention, 2009. **5**(5): p. 580-8.
23. Masson, J.B., et al., *Transcatheter aortic valve implantation: review of the nature, management, and avoidance of procedural complications.* JACC Cardiovasc Interv, 2009. **2**(9): p. 811-20.
24. Piazza, N., et al., *Clinical endpoints in transcatheter aortic valve implantation: a call to ARC for standardised definitions.* EuroIntervention, 2009. **5**(1): p. 29-31.
25. Thomas, M. and O. Wendler, *Transcatheter aortic valve implantation (TAVI): how to interpret the data and what data is required?* EuroIntervention, 2009. **5**(1): p. 25-7.
26. Van Brabandt, H. and M. Neyt, *Safety of percutaneous aortic valve insertion. A systematic review.* BMC Cardiovasc Disord, 2009. **9**: p. 45.
27. Van Brabandt, H. and M. Neyt, *What is the evidence in favor of percutaneous aortic valve insertion?* Am J Cardiol, 2009. **103**(4): p. 575.
28. Dhruva, S.S., L.A. Bero, and R.F. Redberg, *Strength of study evidence examined by the FDA in premarket approval of cardiovascular devices.* JAMA, 2009. **302**(24): p. 2679-85.
29. Serruys, P.W., et al., *Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease.* N Engl J Med, 2009. **360**(10): p. 961-72.
30. Roques, F., et al., *Risk factors and outcome in European cardiac surgery: analysis of the EuroSCORE multinational database of 19030 patients.* Eur J Cardiothorac Surg, 1999. **15**(6): p. 816-22; discussion 822-3.
31. Shahian, D.M., et al., *The Society of Thoracic Surgeons 2008 cardiac surgery risk models: part 3--valve plus coronary artery bypass grafting surgery.* Ann Thorac Surg, 2009. **88**(1 Suppl): p. S43-62.
32. Jin, R., G.L. Grunkemeier, and A. Starr, *Validation and refinement of mortality risk models for heart valve surgery.* Ann Thorac Surg, 2005. **80**(2): p. 471-9.
33. Edwards, F.H., et al., *Prediction of operative mortality after valve replacement surgery.* J Am Coll Cardiol, 2001. **37**(3): p. 885-92.
34. Hannan, E.L., et al., *Risk index for predicting in-hospital mortality for cardiac valve surgery.* Ann Thorac Surg, 2007. **83**(3): p. 921-9.
35. Gardner, S.C., et al., *Comparison of short-term mortality risk factors for valve replacement versus coronary artery bypass graft surgery.* Ann Thorac Surg, 2004. **77**(2): p. 549-56.
36. Dewey, T.M., et al., *Reliability of risk algorithms in predicting early and late operative outcomes in high-risk patients undergoing aortic valve replacement.* J Thorac Cardiovasc Surg, 2008. **135**(1): p. 180-7.
37. Nowicki, E.R., et al., *Multivariable prediction of in-hospital mortality associated with aortic and mitral valve surgery in Northern New England.* Ann Thorac Surg, 2004. **77**(6): p. 1966-77.
38. van Gameren, M., et al., *Do we need separate risk stratification models for hospital mortality after heart valve surgery?* Ann Thorac Surg, 2008. **85**(3): p. 921-30.
39. van Gameren, M., et al., *How to assess risks of valve surgery: quality, implementation and future of risk models.* Heart, 2009. **95**(23): p. 1958-63.
40. Lee, D.H., et al., *Frail patients are at increased risk for mortality and prolonged institutional care after cardiac surgery.* Circulation. **121**(8): p. 973-8.
41. Girardi, L.N., et al., *No-clamp technique for valve repair or replacement in patients with a porcelain aorta.* Ann Thorac Surg, 2005. **80**(5): p. 1688-92.
42. Baumgartner, H., et al., *Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice.* J Am Soc Echocardiogr, 2009. **22**(1): p. 1-23; quiz 101-2.

SYNTAX TRIAL UPDATE AND IMPLICATIONS OF FUTURE RESEARCH

Professor A. P. Kappetein - Erasmus MC, Rotterdam.

Recent years have seen an ongoing debate as to whether coronary artery bypass graft (CABG) surgery or percutaneous coronary intervention (PCI) is the most appropriate revascularisation strategy for patients with coronary heart disease (CAD). The Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery (SYNTAX) study was conducted with the intention of defining the specific roles of each therapy in the management of de novo three-vessel disease or left main CAD. Interim results after 12 months show that PCI leads to significantly higher rates of major adverse cardiac or cerebrovascular events compared with CABG (17.8 versus 12.4; p=0.002), largely owing to increased rates of repeat revascularisation. However, CABG was much more likely to lead to stroke. Interestingly, categorisation of patients by severity of CAD complexity according to the SYNTAX score has shown that there are certain patients in whom PCI can yield results that are comparable to, if not better than, those achieved with CABG. Careful clinical evaluation and comprehensive assessment of CAD severity, alongside application of the SYNTAX score, can aid practitioners in selecting the most suitable therapy for each individual CAD patient.

Since their inception, both PCI and CABG have undergone significant developments that have reduced rates of morbidity and mortality despite the increasing age and prevalence of co-morbidities in the patient population receiving revascularisation. Advances in cardiac surgery include off-pump CABG, enhanced myocardial preservation, improvements in anaesthesia, pre-operative risk assessment and post-operative care, and an increased use of arterial conduits, which have reduced the rate of graft occlusion. In patients treated with PCI, improvements in technology and antiplatelet therapy coupled with landmark studies have effectively led to the replacement of balloon angioplasty with coronary artery stenting, which is the current preferred method of PCI.

The selection of appropriate therapy for CAD has been the subject of continuing debate for many years. Several studies comparing the use of bypass surgery and coronary bare-metal stents (BMS) in patients with multivessel disease have revealed higher rates of repeat revascularisation at five years in patients treated with BMS, while those patients treated with CABG have higher rates of stroke. Nevertheless, overall survival has been comparable between both groups. However, seminal improvements in treatment options have now rendered these studies historical in their applicability to contemporary practice. The introduction of drug-eluting stents (DES) has greatly enhanced the PCI approach to managing CAD, with demonstrated superiority in reducing restenosis over their bare-metal predecessors while maintaining similar rates of death and myocardial infarction (MI). These reductions in restenosis and re-intervention have also been reproduced in patients with multivessel disease and left main disease, such that the use of PCI has expanded to the treatment of patients with severe CAD. Nevertheless, to date the use of PCI in this patient population has not been supported by adequate data from evidence-based medicine or sufficiently powered randomised clinical trials. Indeed, current guidelines state that CABG remains the gold standard and treatment of choice for patients with severe CAD, including three-vessel disease and left main CAD.

The SYNTAX Study

The Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery (SYNTAX) study was designed to assess the optimum revascularisation strategy for patients with de novo three-vessel or left main CAD. To avoid criticism that patients enrolled would be non-representative of real-world patient cohorts, the SYNTAX trial adopted an all-comers design in which all eligible patients with de novo three-vessel or left main CAD were included.

Two year results

Two-year results from the on-going SYNTAX trial show significantly higher rates of MI with PCI when compared to CABG.

At two years the long term durability of CABG is starting to show. The early, higher rate of some complications associated with CABG in the first year, including stroke, have now reversed. In the first

year MI occurred in 3.3% of CABG patients compared to 4.8% of PCI patients. In the second year only 0.1% of CABG patients had a MI, compared to 1.2% of PCI patients.

Differences between the two groups were small in patients with low SYNTAX scores and much bigger in patients with high SYNTAX scores. Patients with a SYNTAX score in the top tercile had event rates of 28.2% in the PCI group compared to 15.4% in the CABG group ($p<0.001$).

Almost 96% of the original trial numbers were included in the two-year analysis: 836 in the CABG arm and 885 in the PCI arm. At two years, MACCE rates were significantly different between the two groups, driven by a repeat revascularization rate in PCI-treated patients that was more than double that of the CABG-treated group. The significantly higher rate of strokes seen in CABG-treated patients at one year was also seen by two years, but the difference appeared to be a carryover from the first 12 months, since very few strokes occurred between the one- and two-year mark in either group. For the hard end point of death/stroke/MI, there were no significant differences between the two groups.

Two-year outcomes for SYNTAX

End point	CABG (%)	PCI (%)	p
All-cause death	4.9	6.2	0.24
All stroke	2.8	1.4	0.03
Stroke before 1 y	2.2	0.6	0.003
Stroke after 1 y	0.6	0.7	0.82
MI	3.3	5.9	0.01
MI before 1 y	3.3	4.8	0.11
MI after 1 y	0.1	1.2	0.008
All-cause death, stroke, MI	9.6	10.8	0.44
Repeat PCI	8.6	17.4	<0.001
MACCE	16.3	23.4	<0.001

The rates of MACCE were no different between the two revascularization strategies for patients who were low risk by SYNTAX score at baseline (17.4% for CABG, 19.4% for PCI; $p=0.63$). But as that risk rose, so too did the curves begin to separate: in patients with intermediate risk by SYNTAX, MACCE rates were 16.4% for CABG-treated patents and 22.8% for PCI-treated patients, just missing statistical significance ($p=0.06$). In high-risk patients, CABG was clearly the winner, with MACCE rates of 15.4% vs 28.2% in the PCI-treated group ($p<0.001$).

As with the one-year results, the two-year outcomes differed according to whether the patients were enrolled in the study for treatment of three-vessel disease or for left main stenting. The subset analysis included low numbers and was not appropriately powered, so it had to be considered only hypothesis-generating. But at least for the primary MACCE end point, event rates were significantly lower in CABG patients with three-vessel disease—14.4% vs 23.8% ($p<0.001$)—but were no different, statistically, between the groups for patients with left main disease—19.3% for CABG, 22.9% for PCI ($p=0.27$)

Longer follow-up is needed and the patients will be followed up until 5 years. At the time of the meeting the 3 years results will be available and presented.

SURGERY OF THE AORTIC

UPDATES FOR THE 3rd MILLENIUM

Professor D. Cameron – John Hopkins Hospital, U.S.A.

Operations to replace or repair the aortic root have been one of the major success stories of modern cardiac surgery. Once feared and avoided except in dire emergency, root procedures now carry low operative risk in the elective setting and are highly reproducible. Many of the critical advances in understanding the complexity of the anatomic and functional behavior of the aortic root have occurred in the United Kingdom, which can rightly call itself the home of aortic root replacement, both its valve sparing and valve replacement forms. Surgeons will likely be called upon to intervene upon the root with increasing frequency, in part due to better detection of root pathology by imaging surveillance, better recognition of clinical syndromes associated with aortic aneurysms, and the still-limited applicability of endovascular approaches; indeed, the root is likely to be the last sanctuary of traditional aortic surgical prowess for some years to come.

The Bentall procedure has enjoyed more than 40 years of clinical use and longterm results have been excellent. Refinements in myocardial protection, surgical technique (abandonment of the inclusion technique), and valve prostheses have kept it the gold standard, by which all other root procedures should be measured. Valve sparing procedures offer freedom from anticoagulation and perhaps lower rates of thromboembolism and endocarditis and better quality of life, but at present are not as durable as the classic Bentall. Continuing evolution of valve sparing surgical techniques and root prostheses will likely yield further improvements in late outcomes.

References

Bentall HH, De Bono A. A technique for complete replacement of the ascending aorta. Thorax 1968;23:338-9.

Yacoub MH, Gehle P, Chandrasekaran V, et al. Late results of a valve-preserving operation in patients with aneurysms of the ascending aorta and root. J Thorac Cardiovasc Surg 1998;115:1080–90.

David TE, Feindel CM, Webb GD, et al. Long-term results of aortic valve-sparing operations for aortic root aneurysm. J Thorac Cardiovasc Surg 2006;132:347–54.

Cameron DE, Alejo DE, Patel ND, et al. Aortic root replacement in 372 Marfan patients: evolution of operative repair over 30 years. Ann Thorac Surg 2009;87:1344–9

Gott VL, Gillinov AM, Pyeritz RE, Cameron DE, Reitz BA, Greene PS, Stone CD, Ferris RL, Alejo DE, McKusick VA. Aortic root replacement. Risk factor analysis of a seventeen-year experience with 270 patients. J Thorac Cardiovasc Surg. 1995 Mar;109(3):536-44

ANATOMY OF THE MITRAL VALVE

Mr. I. Wilson – Queen Elizabeth Hospital, Birmingham.

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 commisure. These are designated the anterolateral and posteromedial commisures. 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 commisure, 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 commisures 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 commisures, 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.

AETIOLOGY, DEFINITION AND ASSESSMENT OF MITRAL VALVE DISEASE

Professor J-L Vanovershelde – Louvain University, Brussels.

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.20,21

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.

PRINCIPLES IN RESTORATION OF MITRAL VALVE COMPETENCE

Dr. P Perier - Herz und Gefäß Klinik, Bad Neustadt/ Saale, Germany.

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 "[i].

Another major breakthrough has been the onset of echocardiography whose role before, during and after the operation has grown to a point where echocardiographists 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 [ii], 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 [iii]. 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 [iv]. 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[v] 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.

[1] Carpentier , A. Cardiac Valve Surgery – « The french Correction ».
J. Thorac. Cardiovasc. Surg. 1983;86:323-337

[1] Perier P., Stumpf J., Götz C., Lakew F., Schneider A., Clausnizer B., Hacker R.
Valve repair for mitral regurgitation caused by isolated prolapse of the posterior leaflet.
Ann Thorac Surg 1997;64:445-50

[1] Perier P., Stumpf J., Clausnizer B., Hacker R.
Klinsiche Erfahrungen mit der Mitrlaklappenrekonstruktion
Herz 1996; 21:166-171

[1] Chauvaud S.
Mitral valve reconstruction – The third Decade
AATS, May 2001

[1] Tribouilloy C., Enriquez-Sarano M., Schaff H. et al
Impact of preoperative symptoms on survival after correction of organic mitral regurgitation
Circulation 1999;99:400-405

MITRAL VALVE REGURGITATION:

EVIDENCE FOR INTERVENTION (INCLUDING ASYMPTOMATIC)

Professor J-L Vanovershelde – Louvain University, Brussels.

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 %.34 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.

TRICUSPID VALVE: THE EVIDENCE FOR INTERVENTION AND OPERATIVE TECHNIQUES

Dr. P. Perier - Herz und Gefäß Klinik, Bad Neustadt/ Saale, Germany.

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.

1- Tager R., et al
Long term follow-up of rheumatic patients undergoing left-sided valve replacement with tricuspid annuloplasty – Validity of preoperative echocardiographic criteria in the decision to perform tricuspid annuloplasty
Am J Cardiolo 1998;81:1013-1016

2- Schapira et al
Evaluation of tricupsid regurgitation severity: echocardiographic and clinical correlation
J Am Soc Echocardiogr 1998;11:652

3- Porter A., et al
Tricuspid regurgitation late after mitral valve replacement: clinical and echocardiographic evaluation
J Heart Valve Dis 1999;8:57

4- Tribouilloy CM, Enriquez-Sarano M, Bailey KR, Tajik AJ, Seward JB
Quantification of tricuspid regurgitation by measuring the width of the vena contracta with Doppler color flow imaging: a clinical study.
J Am Coll Cardiol 2000; 36:472-8

5- Sugimoto T, Okada M, Ozaki N, Hatakeyama T, Kawahira T
Long-term evaluation of treatment for functional tricuspid regurgitation with regurgitant volume: characteristic differences based on primary cardiac lesion.
J Thorac Cardiovasc Surg 1999;117: 463-71

6- McCarthy PM, Bhudia SK, Rajeswaran J, Hoercher KJ, Lytle BW, Cosgrove DM, Blackstone EH
Tricuspid valve repair: durability and risk factors for failure.
J Thorac Cardiovasc Surg 2004 Mar 127:674-85

7- Nath J, Foster E, Heidenreich PA
Impact of tricuspid regurgitation on long-term survival.
J Am Coll Cardiol 2004; 43: 405-9

8- Ohata T, Kigawa I, Yamashita Y, Wanibuchi Y
Surgical strategy for severe tricuspid valve regurgitation complicated by advanced mitral valve disease: long-term outcome of tricuspid valve supra-annular implantation in eighty-eight cases.
J Thorac Cardiovasc Surg 2000;120:280-3

9- Dreyfus G, Corbi P, Chan K, Bahrami T
Secondary Tricuspid Regurgitation or Dilatation: Which Should Be the Criteria for Surgical Repair?
Ann Thorac Surg 2005 ;79 :127

ATRIAL FIBRILLATION – THE RATIONALE FOR TREATMENT

Mr. M. Lewis - Royal Sussex County Hospital, Brighton.

Patients present to cardiac surgeons with either lone or concomitant (AF and something else, such as MVR) AF.

The complex changes in electrophysiology/ atrial structure that lead to AF are not yet completely understood.

In general, patients seek treatment for their AF for three reasons:

1. Symptom relief
2. Avoidance of the sequelae of AF including stroke
3. Possibly, an improvement in their survival

There is some data to support the fact that surgical treatment can keep patients out of AF and therefore, hopefully, help with symptomatic relief.

There is a paucity of published randomised surgical trials to help with our discussion of the other points. There are a number of ongoing surgical studies looking at concomitant and stand alone AF therapy. Until these are available, it is necessary to look at the catheter derived AF ablation data and synthesise this with what we do know about surgical AF treatment.

The current evidence base will be presented along with a look at some of the holes in our knowledge.

Selected reading list:

HRS/EHRA/ECAS Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation: Recommendations for Personnel, Policy, Procedures and Follow-Up
Heart Rhythm, Vol 4, No 6, June 2007

Big document but covers the current position to a large extent.

Mortality, Morbidity, and Quality of Life After Circumferential Pulmonary Vein Ablation for Atrial Fibrillation
Pappone et al. JACC Vol. 42, No. 2, 2003:185–97

Best data available for comparing medical Rx with catheter based ablation- not randomised!

Minimally Invasive Surgical Treatment of Atrial Fibrillation
Randall K. Wolf, MD, FACS
Semin Thorac Cardiovasc Surg 19:311.e1-311.e9 © 2007

Nice review of the "Wolf technique"

NOTES

COMPLETE CLOSED CHEST SURGICAL TREATMENT OF LONE AF

Dr. A. Yilmaz – Nieuwegein, Netherlands.

The classical Maze III procedure has been the gold standard for treatment of symptomatic, drug-refractory atrial fibrillation (AF). Despite high success rates, it has not been widely adopted due to its technical challenge, need for median sternotomy and use of extra-corporal circulation (1). Less invasive procedures have been developed due to increasing knowledge about the pathophysiology of AF and the development of ablation devices, which replace the original 'cut-and-sew"technique for scarification. Ectopic foci from the pulmonary veins (PVs) play an important role in the pathophysiology, especially in lone paroxysmal AF (2). Therefore, both cardiologists and surgeons focus on these vessels for curing AF. Recent clinical and experimental studies also suggest an important role of the autonomic nervous system (3-5). Wolf et al. developed an off-pump procedure in which the PVs are isolated, ganglionic plexus (GP) are ablated and the left atrial appendage are amputated through bilateral thoracotomy(6).

We developed an operation technique for a complete thoracoscopic approach isolating the PVs, ablating the GP and amputating the LAA for the treatment of lone AF (7).

Patients with drug-refractory AF with at least a moderate heart function were eligible for surgery. Previous cardiac or lung surgery and left atrium larger than 60mm were relative contraindications and previous unsuccesfull percutaneous catheter ablation is not.

The patient is placed in a supine position under general anaesthesia with double lumen tube intubation. The procedure is performed in one scrub through alternating three thoracoports on both sides. On the right side, the pericardium is opened anterior to the phrenic nerve, followed by exploration of Waterstone's groove for optimal positioning of the ablation device later on. Blunt dissection around the PVs is performed and isolation of the PVs ia achieved by multiple bipolar radiofrequency ablations. A conduction block is confirmed by the absence of PV potentials in case of AF and exit block (no pacing possibility) in case of sinus rhythm. Plexus locations are identified by high-frequency pacing inducing a vagal response. In case of response, ablation of GP can be performed and repeated if necessary. Connecting lesions to the left side creating finally a extended box lesion set are performed by connecting the superior PVs by an ablation line on the left atrial roof and connecting both inferior PVs; ending finally by performing the mitral (M)-line to the fibrous trigonum of the anterior mitral leaflet. The procedure of isolating the PVs is repeated on the left side, except that the pericardium is opened posterior to the phrenic nerve. In addition the ligament of Marshall is devided and the left atrial appendage is amputated by using an endostapler. Patients are extubated in the operating theatre and sent to the ward after 6 hours postoperative. Discharge changes between postoperative day 1 and 4. All patients receive oral anticoagulation for at least 3 months after surgery. The preoperative antiarrhythmic drugs are usually continued. Sotalol was started if a patient was only on rate-control medication. Electrical cardioversion was performed in the OR at least once and later if a patient had symptomatic AF. In case of asymptomatic AF, the referring physician was advised to perform one later on.

The results of success are in concordance with those of others (8,9). Additional lines in the left atrium could lead to better results and extensions of the indication for surgery (10)This procedure appears to be less succesfull than the more elaborate classical Maze III operation; however, the advantages are clear. It is an off-pump, minimal invasive procedure and has a shorter operation time, intensive care and hospital stay. The advantage of this surgical procedure in comparison to a percutaneous catheter procedure is its epicardial approach under direct vision, possibility of GP-ablation and amputation of the left atrial appendage. This procedure could therefore offer an alternative for percutaneous catheter ablation and is even effective after unsuccessful ones. Further development in hybrid procedure techniques for longstanding persistent AF, creating both epicardial and endocardial ablation lines with adequate mapping will be an important contribution in expanding the field of lone AF therapy in the near future.

References:

(1) Ballaux PK, Geuzebroek GS, van Hemel NM, Kelder JC, Dossche KM, Ernst JM, Boersma LV, Wever EF, Brutel de la Riviere A, Defauw JJ. Freedom from atrial arrhythmias after classic maze III surgery: a 10-year experience. J Thorac Cardiovasc Surg 2006;132(6):1433-40.

(2) Häissaguerre M, Jaïs P, Shah DC, Takahashi A, Hocini M, Quiniou G, Garrigue S, Le Mouroux A, Le Métayer P, Clémenty J. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med 1998;339:659-66.

(3) Pappone C, Santinelli V, Manguso F, Vicedomini G, Gugliotta F, Augello G, Mazzone P, Tortoriello V, Landoni G, Zangrillo A, Lang C, Tomita T, Mesas C, Mastella E, Alfieri O. Pulmonary vein denervation enhances long-term benefit after circumferential ablation for paroxysmal atrial fibrillation. Circulation 2004;109:327-34.

(4) Zhou J, Sherlag BJ, Edwards J, Jackman WM, Lazzara R, Po SS. Gradients of atrial refractoriness and inducibility of atrial fibrillation due to stimulation of ganglionated plexi. J Cardiovasc Electrophysiol 2007;18:83-90.

(5) Scherlag BJ, Yamanashi W, Patel U, Lazzara R, Jackman WM. Autonomically induced conversion of pulmonary vein focal firing into atrial fibrillation. J Am Coll Cardiol 2005;45:1878-86.

(6) Wolf RK, Schneeberger EW, Osterday R, Miller D, Merrill W, Flege Jr JB, Gillinov AM. Video-assisted bilateral pulmary vein isolation and left atrial appendage exclusion for atrial fibrillation. J Thorac Cardiovasc Surg 2005;130:797-802.

(7) Yilmaz A, Van Putte BP, Van Boven WJ. Completely thoracoscopic bilateral pulmonary vein isolation and left atrial appendage exclusion for atrial fibrillation. J Thorac Cardiovasc Surg 2008;136:521-2.

(8) Edgerton JR, Edgerton ZJ, Weaver T, Reed K, Prince S, Herbert MA, Mack MJ. Minimally invasive pulmonary vein isolation an partial autonomic denervation for surgical treatment of atrial fibrillation. Ann Thorac Surg 2008;85:34-8.

(9) Wudel JH, Chaudhuri P, Hiller JJ. Video-assisted epicardial ablation and left atrial appendage exclusion for atrial fibrillation: extended follow-up. Ann Thorac Surg 2008;85:34-8.

(10) Gillonov Am, Bhavani S, Blackstone EH, Rajeswaran J, Svensson LG, Navia JL, Petersson BG, Sabik 3rd JF, Smedira NG, Mihaljevic T, Mc Carthy PM, Shewchik J, Natale A. Surgery for permanent atrial fibrillation: impact of patient factors and lesion set. Ann Thorac Surg 2006;82:502-13.

CONCOMITANT AF LESION SET ETC

Mr. M. Dalrymple-Hay – Derriford Hospital, Plymouth.

OPTIMAL POST-OPERATIVE MANAGEMENT AFTER SURGERY FOR ATRIAL FIBRILLATION

Mr. P. Braidley – Northern General Hospital, Sheffield.

This session will cover management of the following issues :

•Underlying cardiac pathology
•Pacing
•Electrolyte balance
•Diuretics / Fluids
•Anti-arrhythmia drugs
•Anticoagulation
•Follow up and monitoring
•Reporting

In this area there is little trial evidence to guide practice but we will aim to present a broad consensus approach to these issues.

Suggested reading:

HRS/EHRA/ECAS Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation: Recommendations for Personnel, Policy, Procedures and Follow-Up.
Calkins et al Europace (2007) 9, 335–379

Benefits of prophylactic continuous infusion of frusemide after the Maze procedure for AF.
Ad et al. JTCVS (2002) 123, 323-6

Mitral valve surgery plus concomitant atrial fibrillation ablation is superior to mitral valve surgery alone with an intensive rhythm control strategy.

von Oppell UO, et al Eur J Cardiothorac Surg. 2009 Apr;35(4):641-50

Predictive factors of sustained sinus rhythm and recurrent atrial fibrillation after a radiofrequency modified Maze procedure.

Beukema WP, et al Eur J Cardiothorac Surg. 2008 Oct;34(4):771-5.

PRE-OPERATIVE ASSESSMENT

Professor J-L Vanovershelde – Louvain University, Brussels.

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 %.34 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.

ISCHEMIC MITRAL VALVE REGURGITATION:
SURGICAL TECHNIQUES AND THE LIMITS OF OUR KNOWLEDGE

Professor O. Alfieri – Ospedale San Raffaele Hospital, Italy.

Although the presence of mitral regurgitation (MR) is associated with an unfavorable prognosis in patients with ischemic cardiomyopathy, the opportunity to correct MR in such a clinical context is not well established.

Many observational studies show improvement in symptoms and quality of life following surgery and reverse left ventricular remodeling has been consistently documented in selected patients, presumably positively affecting survival.

A careful evaluation of the surgical risk versus the benefit of the operation is mandatory in the individual patient and definite surgical contraindications have to be identified (signs and symptoms of right heart failure, absence of contractile reserve, heavy comorbidities).

Annuloplasty (preferably with an undersized, complete, rigid and shaped ring) is the most common operation used to correct ischemic MR.

Recurrence of MR, however, is non uncommon, particularly when the operation is not carried out in the early phase of the disease, before the occurrence of advanced left ventricular remodeling.

The well defined echocardiographic variables predicting unfavorable outcome with annuloplasty should be taken into account.

Under certain circumstances, surgery should include additional procedures apart from annuloplasty to enhance the effectiveness and the durability of the mitral repair (left ventricular restoration, repositioning of the papillary muscles, resection of the secondary chordae, edge-to-edge suture, external cardiac support, etc…).

Finally mitral valve replacement should be considered when unsatisfactory results are expected with mitral valve repair due to advanced left ventricular remodeling and long-lasting heart failure.

References

1- Grigioni F, Enriquez-Sarano M, Zehr KJ, Bailey KR, Tajik AJ. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. Circulation. 2001;103(13):1759-64.

2- Braun J, van de Veire NR, Klautz RJ, Versteegh MI, Holman ER, Westenberg JJ, Boersma E, van der Wall EE, Bax JJ, Dion RA. Restrictive mitral annuloplasty cures ischemic mitral regurgitation and heart failure. Ann Thorac Surg. 2008;85(2):430-6; discussion 436-7.

3- Hung J, Papakostas L, Tahta SA, Hardy BG, Bollen BA, Duran CM, Levine RA. Mechanism of recurrent ischemic mitral regurgitation after annuloplasty: continued LV remodeling as a moving target. Circulation. 2004;110(11 Suppl 1):II85-9.

4- De Bonis M, Lapenna E, Verzini A, La Canna G, Grimaldi A, Torracca L, Maisano F, Alfieri O. Recurrence of mitral regurgitation parallels the absence of left ventricular reverse remodeling after mitral repair in advanced dilated cardiomyopathy. Ann Thorac Surg. 2008;85(3):932-9.

5- Magne J, Pibarot P, Dagenais F, Hachicha Z, Dumesnil JG, Sénéchal M. Preoperative posterior leaflet angle accurately predicts outcome after restrictive mitral valve annuloplasty for ischemic mitral regurgitation. Circulation. 2007;115(6):782-91.

6- Magne J, Girerd N, Sénéchal M, Mathieu P, Dagenais F, Dumesnil JG, Charbonneau E, Voisine P, Pibarot P. Mitral repair versus replacement for ischemic mitral regurgitation: comparison of short-term and long-term survival. Circulation. 2009;120(11 Suppl):S104-11.

THE ASSESSMENT OF ISCHAEMIC HEART DISEASE

Mr. S. Rooney, Mr. G. Cooper and Mr. M. Lewis

Objective:

At the end of this session, participants should be able to demonstrate knowledge and understanding of the preoperative evaluation of patients undergoing surgery for ischaemic heart disease.

The following aspects of patient assessment will be considered.

History
Examination
Troponin
ECG
Exercise Stress Test
Positron Emission Tomography
Dobutamine Stress Echo/MRI
Coronary angiography.

The format of the session will be that of case based discussion. Scenarios will be considered and investigations reviewed. Indications and contraindications for surgery will be examined to allow treatment plans to be formulated.

The following textbooks/websites may prove useful reading:

The ECG Made Easy. Hampton J.R., Churchill Livingstone
A concise introduction to the ECG.

Measurements in Cardiology. Edited by Peter Sutton, Parthenon Publishing.
Good chapters on ECG (including X-tests), Echo, Cardiac Catheterisation and Nuclear Cardiology.

www.cardiologysite.com
A useful website that introduces the concepts of angiography including a helpful description of the views used to image the coronaries.

http://info.med.yale.edu/intmed/cardio/imaging/contents.html
Clear site, with many images available. Very good 3D viewer demonstrating relation of structures from different viewing angles. (Look in the “Heart in Radiographs” section).

http://heart.bmj.com/collections/index.dtl
Excellent collection of review articles on a wide selection of topics that may come up in the exam.

CARDIOPULMONARY BYPASS

Mr. I. Wilson, Mr. R. Gohil, and Dr. D. Green, Dr. P. Townsend -

These seminars will explore some of the following scenarios. These sessions will be of a practical/interactive nature. The aim is to develop recognition/management of potentially life threatening problems which may occur during cardiopulmonary bypass including:

WEANING FROM CARDIOPULMONARY BYPASS
AIR EMBOLISM
CANNULATION PROBLEMS
EXCESSIVE ARTERIAL LINE PRESSURES
LOW PO_2 LEVELS ON CPBP
USE OF APROTININ/CONTROL OF ACT
POOR VENOUS RETURN/AIR LOCK
PROTAMINE ANAPHYLAXIS
USE OF CENTRIFUGAL PUMPS
MANAGEMENT OF IVC TEAR DURING CPBP
OXYGENATOR FAILURE

Suggested Reading

Taylor K M. Cardiopulmonary Bypass in Seminars in Thoracic and Cardiovascular Surgery 1990.2:291-415
Taylor K M. Cardiopulmonary Bypass. Principles and Management. Williams and Wilkins 1986
Utley J R. Pathophysiology and Techniques of Cardiopulmonary Bypass Vol I and Vol II Williams and Wilkins 1982/1983

NOTES