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.

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AORTIC STENOSIS - THE EVIDENCE BASE FOR SURGERY

WHO, WHY, WHEN. The Influence of Coronary Artery Disease

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

Aims:

Discuss AHA/ACC guidelines for AVR

Differentiate indications for AVR in asymptomatic patients.

Discuss factors associated with worse prognosis in asymptomatic AS

Understand rationale AVR in patients undergoing CABG

Understand issues low aortic valve gradient and poor LV function

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NOTES

AORTIC VALVE REPLACEMENT

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Two randomized prospective trials of biological vs mechanical prosthesis established the concept of balancing the structural deterioration of biological valves over time with the risks of associated anticoagulation with mechanical valves when choosing the most appropriate prosthesis for an individual patient. The results of multiple cohort investigations have re-enforced this concept and conventional interpretation of the overall data suggests mechanical valves are most suited to patients under the age of 65 years and biological valves otherwise. However, a rational decision must be individualized for each patient and take into account their life expectancy, associated co-morbidity, contra-indications to anticoagulation and personal preference. For example, the 10 year survival of a patient with aortic valve disease maybe reduced from 70% to 40% if there is associated coronary artery disease. Proponents of biological valves point to evidence of greater durability of the 'modern' day prosthesis and the reducing risks of any subsequent re-intervention when making a case to expand their use. Proponents of mechanical valves point to their well established durability and the reducing risks of associated anti-coagulation problems with modern regimes and alternative therapies. For stentless bioprosthesis, randomized clinical studies comparing them to stented valves have produced differing outcomes and disagreement as regards improved outcomes. There is little definitive evidence of superiority, particularly in patients with small aortic root diameters. Likewise, when considering the risks of patient-prosthetic mismatch there is conflicting data but little compelling evidence that it exists as an important clinical problem in contemporary cardiac surgical practice.

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NOTES

PATIENT PROSTHESIS MISMATCH IN AORTIC VALVE REPLACEMENT: POSSIBLE BUT PERTINENT?

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Aortic valve replacement (AVR) is now the second most commonly performed cardiac operation and with a rising elderly population the number of such procedures is increasing. More than a quarter of a century ago Rahimtoola coined the term Patient Prosthesis Mismatch (PPM) to describe the situation in which the effective orifice area of a prosthetic valve is smaller than that of the native valve [1]. Initially PPM did not attract much attention because at that time operative mortality and more overt morbidity were much more immediate and relevant issues. Furthermore, over 90% of AVR performed today still use a prosthesis with a sewing ring and by definition must have at least some degree of PPM. Finally as many patients can tolerate at least moderate aortic stenosis with excellent functional status and well preserved ventricular function over long periods without the need for surgery, the clinical relevance of PPM is uncertain.
In the last decade, however, Pibarot and colleagues more precisely sub-classified PPM according to the effective orifice index area of a prosthetic valve as mild (>0.85cm^2/m^2), moderate (0.65 – 0.85cm^2/m^2) or severe (<0.65cm^2/m^2) [2,3]. Since then, the cardiac surgical literature has become increasingly concerned with the possible adverse effects of PPM on short and long term survival as well as functional status. However, surgical procedures designed to enlarge the aortic root to avoid PPM increase the complexity of the operation and its operative mortality even in the best centres. So is there compelling evidence that the potential detrimental effects of PPM merit the performance of more complex and higher risk operations ?

1. Two important considerations regarding PPM and outcome
The question of whether PPM impacts adversely on clinical outcome may initially appear straightforward but in reality is bedevilled by two facts. First, and most importantly, patients at the highest risk of PPM are those who are already also at the highest risk from surgery. PPM is most likely to occur in patients with small aortic roots who accordingly receive smaller prosthetic valves. This scenario is most probable in the elderly (especially females) who are also more likely to have severe coronary artery disease and poorer cardiac function and are therefore already at a higher risk from surgery. Even multivariate analysis can only partially, at best, discriminate between these confounding factors.
A second complicating factor is the now well recognized, albeit counter-intuitive, fact that there can be marked discrepancies between the manufacturer labelled and actual diameter of the valve prosthesis [4-6]. Failure to appreciate this has almost certainly contributed to a frequent inability in the literature to relate labelled size of valves to haemodynamic performance.

2. PPM and short term survival from AVR
The strongest evidence implying that that PPM has an important adverse influence on operative mortality following AVR is by Pibarot's group. In 1266 consecutive AVR patients the overall 30 day mortality was 4.6%. However in 38% of patients with moderate or severe PPM the relative risk of mortality was increased two fold in patients with moderate PPM and eleven fold in those with severe PPM. Although the authors reported that co morbid factors such as older age, female gender, coronary artery disease, hypertension, diabetes, and emergent/salvage operation were more prevalent in patients with moderate and severe PPM and might have contributed to the higher mortality in these patients they still went on to conclude that PPM is a strong and independent predictor of short-term mortality among patients undergoing AVR.
Few other studies have identified such an adverse effect of PPM on operative outcome. Indeed Blackstone and colleagues study of the effect of PPM on operative outcome in over 13000 patients undergoing AVR found that PPM as defined in their paper as <1.2cm2/m2, was rare. There was a small increase in in-hospital mortality 1-2%, although intermediate and long-term mortality were unchanged when compared to the PPM group.

3.PPM and long term survival from AVR

The most definitive study on the potential effects of PPM on long term survival was by Blackstone and colleagues in a study of over 13 000 patients undergoing aortic valve replacement and forward for up to 15 years. After adjustment for other preoperative risk factors the authors could identify no effect of PPM on survival. This study reinforces the findings of several other studies. Similarly, Hanayama and colleagues, in a study of almost 700 patients undergoing aortic valve replacement and followed for 10 years, could find no effect of even severe PPM on survival. Indeed 3 other studies could find no effect on long term outcome.

4. PPM and cardiac failure after AVR.

Ruel and colleagues reported PPM (defined as an effective orifice area of less than 0.80cm^2/m^2) as an independent predictor of post operative congestive heart failure in over 1500 patients undergoing AVR, but interestingly had no effect on survival. Interestingly enough, when a slightly different cut-off value was used, less than 0.85cm^2/m^2, the association between mismatch and heart failure disappeared.

5. PPM and functional recovery after AVR

This question was recently addressed in a study from the Cleveland Clinic in 1108 patents undergoing aortic valve replacement and whose functional post-operative recovery was assessed by the Duke Activity Status Index (DASI) at 8 months after surgery. Overall there was a significant improvement in post operative functional recovery in all patients, but no measure of valve orifice area could be correlated with functional recovery. On the other hand, female sex, increasing age and pre-operative renal impairment are associated with a poor functional recovery.

6. Surgical options and risks for avoiding PPM

The potential risk of PPM has led several authors to recommend manoeuvres to enlarge the aortic annulus or root. While these procedures may be technically successful in permitting implantation of a larger sized prosthesis they also increase the complexity of the procedure and, more importantly, the operative risk, particularly as those who are most likely to have PPM are those who have the smallest roots, in other words elderly females. Even in the best hands enlargement of the aortic root often can transform a straightforward operation into a more complex procedure as witnessed by a significant increase in mortality.

7. Summary and conclusions.

Most patients undergoing prosthetic AVR will have some degree of PPM because the presence of a sewing ring makes most aortic valve prostheses by definition inherently stenotic. While intuitively PPM might be felt to adversely effect operative outcome, and functional activity or survival the best evidence shows that in the vast majority it is of little clinical relevance. This however is consistent with the observation that asymptomatic patients can have severe aortic stenosis (defined as an area less than 0.6cm^2/m^2) and remain asymptomatic with good long term outcome as long as they are.

Even in the small cohort of patients with potentially severe PPM there is still little substantial evidence that this has an important adverse effect on short or long term functional recovery or survival. However, the increased risks of enlarging the aortic roots in such patients, who are usually elderly and with small aortic roots and who are already at the highest risk from surgery, should therefore be carefully considered – especially as most will do well with less than a perfect result because activities are already limited.

AORTIC VALVE REGURGITATION

INDICATIONS FOR SURGERY AND TECHNIQUES OF REPAIR

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Aortic valve regurgitation may be due to dilatation of the aortic root, cusp prolapse, cusp retraction, or a combination of pathologic components. Surgery is indicated for severe AR if symptomatic, or LV dysfunction is present, or in conjunction with other primary cardiac procedures. Surgery is also reasonable for asymptomatic patients in the presence of LV dilatation (LVEDD > 70 mm, LVESD > 50 mm), also for moderate AR in conjunction with other cardiac procedures. Surgery is indicated also irrespective of the degree of AR if aortic diameters exceed 45 mm in Marfan's syndrome, 50 mm in the presence of a bicuspid valve, and 55 mm in others.

Surgery for aortic valve regurgitation must take the valve and – if present – aortic pathology into consideration. While valve replacement is still a standard therapy for the aortic valve, in the past years improved understanding of the mechanisms of aortic regurgitation has made reconstructive surgery possible.

Repair must correct the underlying pathology, i.e. root dilatation, cusp prolapse, or cusp retraction. Different pathologies may coexist, and upon careful inspection, a combination is found in the majority of patients. Similar to mitral repair, surgical correction should probably address all components that are present.
Root dilatation can be corrected by valve reimplantation within a vascular graft, root remodeling, or subcommissural plication. Cusp prolapse can be corrected by triangular correction or simple plication. Deficiency or retraction of cusp tissue requires cusp augmentation, i.e. using autologous pericardium.

We have explored the possibilities and limits of aortic valve repair over 10 years. With systematic repair approaches repair is possible in more than 80% of patients with aortic regurgitation, 10-year freedom from reoperation is better than 90%. Operative mortality is not increased over valve replacement, the incidence of valve-related complications, such as endocarditis or thromboembolic problems has been low. Aortic valve function is essentially physiologic. Most importantly, endocarditis – if it occurs - has been amenable to conservative therapy.

Aortic valve repair is a new and promising treatment option for patients with aortic regurgitation. Mid-term stability is similar to the more established results of mitral repair procedures.

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NOTES

CORNONARY ARTERY BYPASS GRAFT VERSUS PERCUTANEOUS CORONARY ANGIOPLASTY: CABG ON THE REBOUND?

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Purpose of Review To examine the benefits of coronary artery bypass grafting (CABG) against percutaneous coronary intervention (PCI) and particularly the use of drug eluting stents (DES) in situations where CABG has traditionally been considered the most effective therapy on clinical and economic grounds.

Recent Findings Current studies reconfirm that CABG is still the best therapy in terms of improved survival and freedom from reintervention for most patients with proximal left anterior descending, multivessel and left main coronary artery disease (CAD) and that these benefits are even greater in diabetic patients. Health economic analyses also confirm the cost effectiveness of medical therapy and CABG but not PCI. Furthermore, several metaanalyses have shown that DES do not improve survival or freedom from myocardial infarction over bare metal stents but increase the risk of stent thrombosis with its associated medical and financial implications for prolonged dual antiplatelet medication.

Implications of the findings for clinical practice In view of the evidence in favour of CABG this article questions the justifiability of some trials of PCI vs CABG, especially in diabetic patients and those with left main stem CAD, and exhorts the need for a multidisciplinary team approach to the management of CAD as a 'minimum standard of care'.

KEY WORDS: coronary artery bypass grafting (CABG); percutaneous coronary intervention (PCI); multivessel and left main coronary artery disease; cost effectiveness; drug eluting stents; diabetes; multidisciplinary team.

Introduction

The last decade has witnessed the relentless growth of percutaneous coronary intervention (PCI) in patients with stable coronary artery disease (CAD). In many industrialised countries the ratio of PCI to CABG now exceeds 4 to 1 [1]. The initial promise, however, that drug eluting stents (DES) would eliminate the problem of restenosis has proved not to be the case, particularly with more complex lesions, and is

compounded by an increased incidence of early and late stent thrombosis particularly when used in off label settings [2*]. In view of continuing mortality and high repeat rates of revascularization with PCI best evidence suggests that coronary artery bypass grafting (CABG) is still the best therapy for most patients with multi vessel and left main CAD both on clinical and economic grounds.

Isolated left anterior descending coronary artery

The left anterior descending coronary supplies a greater amount of myocardium than the circumflex or right coronary arteries and, consequently, disease in its proximal portion has more important adverse prognostic significance even in asymptomatic patients when there is objective evidence of ischaemia [3]. The most durable and proven treatment is revascularization with an internal mammary artery (IMA) graft which significantly reduces the subsequent risk of death, myocardial, recurrent angina and any need for further intervention [4,5]. The conventional requirement for a median sternotomy incision and cardiopulmonary bypass has, nevertheless, resulted in the less invasive option of PCI with stents increasingly replacing CABG, unless contraindicated by anatomical (eg ostial lesion) or pathological complexities.

However, a recent meta-analysis has demonstrated that an IMA graft to the left anterior descending coronary artery by a minimally invasive approach on the beating heart is both clinically [6**] and economically [7**] more effective than stenting with bare metal stents (BMS) over a four year follow-up period. PCI resulted in a three fold increase (13%) in recurrent angina and the need for reintervention in comparison to surgery (4%) [6**]. Although there was no difference in survival between the interventions it is possible that a potential survival benefit of surgery was underestimated both because of the relatively limited duration of follow up and because more severe and complex lesions, unsuitable for stenting, would have been excluded from the trials but still have benefited from surgery. Consequently, while some patients with proximal left anterior descending CAD which is amenable to both interventions may favour the less invasive approach of PCI it is important that they understand that that there is a substantially higher risk of reintervention, at least with BMS, and that they may be foregoing a survival advantage from an IMA graft.

Multi Vessel Coronary Artery Disease

The use of PCI in multivessel CAD has been based on 15 randomised trials of PCI versus CABG which individually reported similar survival (but at least a three fold increase in repeat interventions) with PCI.

However the results of these trials may be applicable to only small groups of patients with multivessel CAD as the trials only included around 5% of all potentially eligible patients, predominantly those with single or double vessel CAD and normal left ventricular function [8**] a group who are known to have little prognostic benefit from CABG [4]. For example, in the 1829 patients in the BARI trial only 41% had three vessel CAD, only 36% had a significant LAD stenosis and around 80% had normal left ventricular function [9**]. And the same type of patients largely dominated the more recent ARTS [10] and SoS [11**] trials which involved the widespread use of stents. By largely excluding patients with more severe disease who have a proven survival benefit from surgery (ie three vessel CAD, chronic total occlusions and impaired left ventricular function) the trials were, in effect, not only biased against the prognostic benefit of CABG [8**] but by being used to justify PCI in such patients have denied them the prognostic benefit of surgery.

Even so a meta-analysis of these trials, involving almost eight thousand patients followed for up to eight years, did show a small but prognostically significant benefit of CABG translating into an NNT of 53 and accompanied by a three fold reduction in need for repeat reintervention at five years [12]. While the ARTS trial found a similar five year survival of 92% between CABG and PCI in its 1205 patients [10] the SoS trial reported significantly better five year survival for CABG (93.4%) than PCI (89.1%) in its 988 patients [11**].

However, it is important to emphasise again that these trials almost certainly underestimate the true survival advantage from CABG for two reasons: (i) analysis on an intention to treat basis discounts the benefits accrued by those PCI patients who cross to CABG eg 58% of BARI PCI patients had actually undergone CABG by 10 years [9**]; (ii) as explained above these trials contained relatively low risk patients in whom the prognostic benefit of CABG is less definite.

As expected the greatest benefit from CABG was seen in those patients with more extensive CAD [12] and is consistent with the results from several large observational data bases which have consistently shown a survival benefit for CABG in patients with multivessel CAD [13-16]. For example in the New York Registry of almost 60,000 patients, propensity matched for cardiac and non-cardiac comorbidity, within 3 years of follow up there was an absolute 5% decrease in survival and a 7 fold increase in the need for reintervention in patients undergoing PCI in comparison to CABG [14]. Similar results were reported in

the Cleveland Clinic [13] Northern New England [15] and Duke [16] databases.

Left Main Stem Stenosis

LMS stenosis is reported to be present in 4%-6% of patients undergoing coronary angiography [17**,18] and in up to 30% of CABG patients [19]. Because of its proven survival benefit CABG has generally been regarded as the "gold standard" therapy for significant left main stem (LMS) stenosis for the last decade in ACC/AHA guidelines for CABG [20] and in both ESC [21] and ACC/AHA [22,23] guidelines for PCI. However in a recent survey of interventions in patients with LMS stenosis, 29% of European patients and 18% of North American patients underwent PCI rather than CABG [24**].

While the proximal anatomic position of the left main coronary artery and its relatively large diameter make it an attractive target for PCI two important factors mitigate against long-term success with PCI. First, up to 90% of stenoses are distal and/or bifurcating lesions [25-32**] which are notoriously prone to restenosis [33-36] and, second, up to 80% of patients have concomitant multi-vessel CAD [25-32**] where CABG already offers a survival advantage [13-16]. Serruy's group emphasized the importance of distal LMS stenosis in predicting adverse outcomes reporting that after a median follow up of 20 months, in 130 patients with LMS stenosis, the cumulative incidence of major adverse cardiac events was 30% in patients with distal versus 11% in those without distal disease ($p= 0.007$) mainly driven by the different rate of target vessel revascularization (13% vs 3%; $p = 0.02$) [36**].

The relevance of PCI studies in LMS stenosis to real clinical practice is also frequently compromised by absence of precise data on selection criteria for PCI rather than CABG, the proportion of all LMS stenosis patients undergoing PCI rather than CABG, the proportion of PCI patients ineligible for CABG because of other co-morbidity (who were therefore also high risk for PCI) allied to incompleteness of angiographic follow-up and limited duration of clinical follow-up (rarely exceeding two years) which discounts the survival benefit of CABG which accrues with time.

Eight studies of BMS, conducted between 1999-2003, and involving over 1100 patients had an overall in-hospital mortality of 6% with a need for further immediate revascularisation averaging 4% (range from 0% to 20%) [8]. Most importantly however by two years of follow-up overall mortality averaged 17% (range 3% to 31%) and the need for repeat revascularisation rate averaged 29% (range 15% to 34%) [8].

Better results with BMS have been reported in younger patients with good left ventricular function, predominantly ostial or mid shaft LMS lesions and a lower incidence of concomitant CAD with mortality rates of 3.4% [37] to 7% [38] at one year and 7.4% [39] at three years but with respective repeat revascularization rate of 32%, 28% and 28%. It should, however, be borne in mind that the risks of CABG would also be low in such low-risk populations eg the one year mortality in 504 CABG patients in the SoS trial was 0.8% [11**].

The ability of DES to reduce restenosis has encouraged their use in LMS stenosis [25-32**] although, again, relevance to real clinical practice is also hampered by lack of detail regarding eligibility criteria for PCI vs CABG, small individual patient numbers (50 to 130 per study), incomplete angiographic assessment of restenosis and limited clinical follow–up (usually less than a year). However, as a higher proportion of these patients had distal or bifurcation LMS stenosis (up to 90%) and also significant CAD (up to 100%) early results appear encouraging with an average in-hospital mortality of 2% and an average immediate repeat revascularisation rate of 2%. However at a mean follow-up of less than a year (range 6-18 months) mortality had increased to 7% and repeat revascularisation to 13% (range from 2%-38%). In the only study with complete angiographic follow-up, at both three and then nine months, Price and colleagues reported that the restenosis rate increased from 34% to 44% [28**]. Ominously, two of these studies cautioned that as restenosis is frequently asymptomatic surveillance angiography is mandatory [28**,32**]. It is uncertain, however, how often or for how long repeat angiography is necessary in this critical location and its associated financial implications.

Although several trials of PCI vs DES in LMS are ongoing, including the SYNTAX, FREEDOM and LEMANS no results have yet been formally reported Three registries have compared CABG and DES in patients with LMS stenosis [29,31,32**]. In the Bologna Registry of 154 CABG and 157 PCI patients [32**], at a median follow up of 14 months the overall respective mortality was 12% and 13% (but 3% respectively in low risk patients), with an eight fold increase in repeat revascularization with DES (26% vs 3%). In an Italian Registry [29] of 107 PCI and 142 CABG patients one year mortality was similar after adjustment for baseline characteristics but the need for repeat revascularization was 20% for PCI and 4% for CABG patients. Lee and colleagues reported six month outcome in 50 PCI and 123 CABG patients with

LMS stenosis but the small numbers and short follow up make data difficult to interpret [31].

The UK Society of Cardiothoracic Surgery database reported 3% mortality in all five thousand patients undergoing CABG for LMS stenosis in 2003 in contrast to a mortality of 1.8% in all seventeen thousand patients without LMS stenosis [19]. Whether surgical results can be improved further with the use of off-pump CABG and composite arterial grafts based on bilateral IMA grafts [40], to simultaneously avoid the use of cardiopulmonary bypass and to permit a no-touch aortic technique (thereby reducing the risk of stroke), is the subject of ongoing trials [41*].

Diabetes

The BARI trial has recently confirmed that even in relatively low risk diabetic patients (only 50% had three vessel CAD, only 40% had true proximal LAD disease and most had normal ventricular function) there is a survival advantage at 10 years for CABG in comparison to PCI (58% vs 46% p=0.025) [9**]. This is also consistent with five year survival data for 208 diabetic patients in the ARTS trial (CABG 92% vs PCI 87%) [10] and 147 diabetic patients in the SoS Trial (CABG 94.6% vs PCI 82.4%) [11**]. In addition to the survival advantage for CABG there was a striking difference in the need for overall reintervention in the BARI trial (18% of CABG and 80% of PCI patients [9**]) and in the 208 diabetic patients in the ARTS trial (10% of CABG and 43% of PCI patients [10**]).

As indicated earlier the survival benefit for CABG may be greater in real life in diabetic patients with more severe CAD who were excluded from the trials. In a regional database of 7,159 diabetic patients who underwent coronary revascularization during 1992 to 1996, 2,766 (39%) were similar to those in the BARI trial. Of these, the 736 who underwent PCI were younger, had higher ejection fractions and less extensive coronary disease than the 2030 who underwent CABG but when adjusted for differences in baseline clinical characteristics, had a significantly higher overall mortality (HR = 1.49; p = 0.04) and particularly so in the 1,251 patients with 3VD (HR = 2.02; p = 0.04) [42].

Two reasons why CABG offers a survival advantage for multi vessel and left main CAD

There are two reasons which probably explain the consistent survival benefit for CABG reported in several large observational studies [13-16]. First, because bypass grafts are placed to the mid coronary vessel, CABG protects whole zones of vulnerable proximal myocardium not only against the 'culprit' lesion (of

any complexity) but also offers prophylaxis against new lesions in diffusely diseased endothelium. In contrast, PCI only treats immediate culprit lesion, assuming that it is technically feasible, but has no protective effect against the development of new proximal disease. Second, the failure of stenting to achieve complete revascularization in most patients with multivessel disease reduces survival proportional to the degree of incomplete revascularization [43**]. Over 22000 patients from New York State's Percutaneous Coronary Interventions Reporting System were subdivided by complete (69%) or incomplete (30%) revascularization. After adjustment for baseline differences, patients with incomplete revascularization were significantly more likely to die at any time (adjusted hazard ratio=1.15) and especially those with total occlusions and a total of at least two incompletely revascularized vessels (hazard ratio=1.36) [43**].

Will these conclusions be altered by drug eluting stents?

For the two reasons, explained above, it is unlikely that DES or indeed any other type of stent will match the results of CABG for most patients with multivessel or left main CAD. And, indeed, these same reasons are also the most likely explanation of several meta-analyses which report that PCI with stents has no survival advantage over optimal medical therapy in stable CAD [44,45**] and that although, in comparison to BMS, DES reduce the risk of restenosis in low-risk coronary lesions, they do not reduce the risk of mortality or subsequent myocardial infarction [46-49**]. Nevertheless this reduction in restenosis has resulted in DES reaching 90% penetration in some centres and being used in up to 60% of 'off label' indications.

A further consideration is that stent thrombosis appears to be a potentially important limitation of DES associated with an increased risk of myocardial infarction of 65-70% and of mortality of 25% to 45% [50,51**,52]. Although multifactorial in aetiology, the single most important mechanism is impaired endothelialization leaving a potentially prothrombotic substrate within the vessel [53**]. While the precise incidence of stent thrombosis with DES is unknown, the annual risk is estimated at between 1% and 5%, depending on the complexity of the lesion, other patient co-morbidities, and use in 'off label' situations. However the FDA have cautioned that the use of DES is 'associated with increased risks of both early and late stent thrombosis, as well as death and myocardial infarction' [2].

These clinical concerns are compounded by cost implications; not only are DES significantly more

expensive than BMS but new recommendations that patients remain on clopidogrel for at least a year [54**] and possibly indefinitely, despite the increased bleeding risks, and at a minimum cost of around a thousand dollars per year will add significantly to costs.

Health Economics for Multi Vessel Coronary Disease

A recent study in the British Medical Journal looked at cost effectiveness of interventions in 1720 patients who were allocated to PCI, CABG or either therapy according to the recommendations of a panel of nine experts and followed for 7 years. It was concluded that while medical therapy and CABG were cost effective at a conventional QUALY of £30,000 ($60,000) PCI was not cost effective and that the additional benefit of stenting over best medical therapy was 'too small to justify the additional cost' [55**]. These findings are consistent with a previous report by the Health Technology Assessment Group in the United Kingdom who also questioned whether the additional costs of DES were justifiable warning that that the widespread us of DES might 'reduce the gain in quality and possibly the duration of life arising from CABG in the long term' [56].

Need for a multi disciplinary teams approach to informed consent.

The adverse clinical and economic implications of the phenomenal growth in PCI, without an appropriate supportive evidence base, are self evident. Most importantly this strategy has, in effect, denied many patients with multivessel and left main CAD, and particularly diabetic patients, the survival and freedom from reintervention benefits offered by CABG. This reinforces the dangers when a recommendation for stenting is made by an individual interventional cardiologist rather than by a multidisciplinary team (MDT) which should also include a non-interventional cardiologist and surgeon. The MDT should be the minimum mandatory 'standard of care', to ensure that the most balanced and appropriate advice is consistently offered [57,58] and should be enforced by appropriate regulatory bodies and those who pay for coronary interventions.

Cautions about future trials of PCI and CABG

The justification for randomized trials of PCI vs CABG in patients in whom there has been shown to be a strong and consistent survival benefit for CABG (eg left main stem CAD, diabetes, some patterns of multivessel CAD) requires careful consideration because it can be argued that, in the absence of real clinical

equipoise between the interventions (ie substantial uncertainty over the risks and benefits of each therapy) such trials may withhold a proven and efficacious treatment [59]. Both Ethics Committees who approve such trials and participating patients must be aware that apparent satisfactory short-term outcomes of PCI are less favourable even within a year and that significant uncertainties about its reliability and durability over the longer term should be weighed against the proven survival benefits

of surgery. It is vital, however, that where such trials are conducted that they are powered sufficiently to evaluate mortality as well as other clinically important differences (to avoid an erroneous conclusion that the two interventions are equally effective), that they include at least medium term follow-up of at least five years (as the benefits of surgery accrue with time) and that they maintain a registry of all potentially eligible patients not entered into the trials (to reflect real clinical practice).

Conclusions

Current studies reconfirm that CABG remains the best therapy in terms of superior survival and freedom from reintervention for most patients with proximal left anterior descending, multivessel and left main coronary artery disease (CAD) and that these benefits are magnified in diabetic patients. Furthermore in economic terms PCI is not a cost effective intervention in comparison to medical therapy or CABG. These conclusions are unlikely to be changed by DES which have not been shown to improve survival or freedom from myocardial infarction in any situation and with which uncertainties persist over the precise risk of stent thrombosis. A multidisciplinary team approach to the management of CAD should be enforced as the 'minimum standard of care' to ensure that patients receive the most balanced advice and can make the most informed choices.

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UNSTABLE ANGINA: EARLY SURGICAL REVASCULARISATION FOR ALL?

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The role of revascularisation for patients with acute coronary syndrome without Q-wave myocardial infarction remains controversial. Acute coronary syndrome encompasses a wide range of patients, from those presenting with non-ST elevation myocardial infarction to those who have recent onset angina without evidence of myocardial necrosis. The "old" surgical trials of unstable angina [1, 2] had not identified a clear survival benefit of coronary revascularisation over medical therapy, except for patients with moderate LV dysfunction (ejection fraction between 30% and 50%). However, the limitations of these trials in which there was a 30% cross over from medical to surgical treatment and with the "best"medical treatment of the time pre-dating the introduction of statins and angiotensin converting enzyme inhibitors are well known. It has been common practice to recommend surgical revascularisation for patients with unstable angina on the basis of the same indications advocated by the trials of coronary artery bypass grafting in patients with chronic coronary artery disease.

The recent understanding of the pathophysiology of acute coronary syndrome and the advent of percutaneous revascularisation techniques led to the design of new trials of treatment of unstable angina. In these studies patients were randomised to an "early invasive" and an "initial conservative" strategy [3-6] Coronary revascularisation was achieved largely by percutaneous techniques and coronary artery bypass grafting was used in approximately 20% of patients. The main end-points of these studies were a composite of mortality, incidence of myocardial infarction, re-hospitalisation or refractory angina.

The results have been variable. The VANQWISH study demonstrated an adverse effect of the early revascularisation strategy compared with the more conservative approach, while the three more recent trials (FRISC II, TACTICS and RITA III) suggest a benefit associated with an early revascularisation strategy. These benefits consist mainly of a reduced incidence of non-fatal myocardial infarction [4, 5], recurrent or refractory angina [6] and/or re-hospitalisation [5]. No study has demonstrated a clear survival benefit for either approach. On the basis of these findings, there is a tendency to recommend early revascularisation for patients with acute coronary syndromes who have not suffered a Q-wave myocardial infarction. This policy is supported by the fact that percutaneous revascularisation techniques have become safer and more effective, particularly with the advent of stenting and the introduction of drugs that effectively downgrade platelet activity. Can we extend these recommendations to patients who need surgical revascularisation?

The studies did not specifically analyse the results of patients who required CABG. However, in the VAMQWISH trial a high in hospital mortality (11%) was detected among patients undergoing CABG at a median of 8 days from admission (FIG). This compares unfavorably with the mortality of other trials and that of patients undergoing CABG at a median of 24 days in the same trial. These studies appear

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comparable for most known patient related risk factors associated with increased operative mortality and the only significant difference can be found in the degree of myocardial necrosis at time of enrollment. In VANQWISH all patients had a CK/CK-MB rise greater than 1.5 times the upper limit of the reference level of the enrolling hospital. In the FRISC II trial the prevalence of raised CK/CKMB in the patients enrolled is not reported, however in this study less than 60% of the patients had a troponin-T rise ≥ 0.1 □g/L. In TACTICS only 37% of the patients had a CK-MB rise, but this was small (< 3 times the upper reference limit for each hospital), and over 40% of patients in the study had no troponin rise [7]. In RITA III CK/CK-MB rise was an exclusion criteria for entry. All the patients in the VAMQWISH study had myocardial infraction, albeit non-Q, and the influence of timing of surgery on mortality in this group is well known. In the study by Curtis et al [8]the mortality of patients undergoing CABG within a week from MI ranged between 18.6% and 7.4% and decreased significantly to 2.7% if surgery was performed after 3 weeks. A recent study of 5517 patients undergoing CABG showed that in-hospital mortality was highest in the group undergoing surgery within 1 week from MI (13%) [9]. Finally, the mortality of patients undergoing CABG within 30-days from MI in UK in 2000 and 2001 was 5.9% and 6.3 % respectively [10]. This was significantly higher than for patients without MI (2% and 1.9% respectively) and remarkably similar to the overall mortality of the patients in VANQWISH undergoing surgical revascularisation (7.7%).

It is not possible to differentiate between non-Q and Q-wave MI in these reports, and there are no studies to address the relationship between level of CK rise and perioperative mortality.

How do we screen patients presenting with acute coronary syndrome?

By comparison with the other trials that used Troponin (T or I) VANQWISH used 'conventional' cardiac enzymes as markers of myocardial damage. In that study myocardial damage was biochemically defined as: "*one or more cardiac enzymes (Creatine kinase; CK, Aspartate Amino Transferase; AST, or Lactate dehydrogenase; LDH) reaching 1.5 times above the laboratory upper limit of normal and/or two consecutive CK and Creatine kinase MB fractions (CK-MB) separated by 4 hours to exceed the upper limit of the laboratory reference interval*" [11].

These criteria for myocardial injury are not consistent with current views (ref). Troponin (I or T) measurements alone are often used for the detection of myocardial necrosis. This does not reliably differentiate between "small" and "significant" injury. Myocardial damage sufficient to release a detectable increase in conventional cardiac enzymes is usually considered MI. To detect patients who have significant myocardial damage and are at high risk of peri-operative mortality assessment of conventional myocardial enzymes in addition to Troponins is therefore essential. The standards recommended by the American College of Cardiology and by the European Society of Cardiology advise that most sensitive and specific non-Troponin biochemical marker of myocardial damage is CK-MB (mass measurment). A biochemically significant amount of myocardial injury consistent with an MI occurs when:

- CK-MB exceeds the 99th percentile of a reference control value on two successive samples.
- Or a maximal value 2 times the upperlimit of normal for that laboratory on one occasion after the index clinical event.

For those laboratories that might not use CK-MB, a pragmatic approach would be the use of total CK. However, the sensitivity and specificity of this marker is lower than CK-MB and this could lead to a small but significant proportion of patients at greater operative risk.

In summary, review of the recent unstable angina trials suggests therefore a pragmatic strategy for the patients in need of surgical revascularisation. The key factor seems to be whether there has been a CK/CK-MB rise and this information should be gathered in addition to the troponins. In patients without CK rise but with abnormal troponins, these studies suggest that the potential benefits of an early revascularisation approach are not offset by elevated operative mortality. In the patients with more pronounced myocardial necrosis waiting for at least 3 weeks, if clinically acceptable, may come with a significantly reduced operative mortality. The risk of operative death in patients with CK rise needing early surgical revascularisation remains high.

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NOTES

THE IMPACT OF MEDICAL MANAGEMENT ON SURGICAL PRACTICE IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE AND ACUTE CORONARY SYNDROMES

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

The aims of treatment are to

- Prevent MI and death
- Reduce incidence of acute thrombotic events
- Prevent ventricular dysfunction
- Minimise or abolish symptoms

Accompanying the lecture will be flow charts with recommended pharmacological therapies based on some of the following references

CAPRIE Lancet 1996;348:1329-1339
Grundy et al. JACC 2004;44:720-732
4S Lancet 1994;344:1383-1900
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Heidenreich et al. JAMA 1999;281:1927-1936

THE ROLE OF CARDIOPULMONARY BYPASS

Mr. M. Pullan – The Cardiothoracic Centre, Liverpool.

What is required for successful Coronary artery grafting?

What is the evidence and who fares best with what?

What am I trained to do?

Hannan EL, Wu C, Smith CR, Higgins RSD, Carlson RE, Culliford AT, Gold JP, Jones RH. Off-pump versus on-pump coronary artery bypass graft surgery: differences in short-term outcomes and in long-term mortality and need for subsequent revascularization. *Circulation*. 2007;116:1145–1152

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Takagi, Hisato MD, PhD; Tanabashi, Toshiyuki MD; Kawai, Norikazu MD; Umemoto, Takuya MD, PhD **Cognitive decline after off-pump versus on-pump coronary artery bypass graft surgery: Meta-analysis of randomized controlled trials.** *Journal of Thoracic & Cardiovascular Surgery. 134(2):512-513e1, August 2007.*

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Lim, Eric FRCS (C-Th); Drain, Andrew MRCS ; Davies, William MRCS ; Edmonds, Lyn MCLIP ; Rosengard, Bruce R. FRCS A systematic review of randomized trials comparing revascularization rate and graft patency of off-pump and conventional coronary surgery. *Journal of Thoracic & Cardiovascular **Surgery**. 132(6):1409-1413, December 2006*

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NOTES

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.

NOTES

SECONDARY MITRAL REGURGITATION: ROLE OF ECHOCARDIOGRAPHY

Secondary mitral regurgitation (MR) is, by definition, a mitral valve dysfunction with apparently "normal" mitral valve, caused by a ventricular disease: ischemic ("ischemic" IMR) or non ischemic disease as dilated idiopathic cardiomyopathy or aortic valve disease cardiomyopathy ("functional" FMR). It is opposed to organic MR where the LV dysfunction is a consequence of a primary mitral valve disease.
Ischemic/functional MR is a ventricular disease and not a valvular one. It explains why this frequent disease (10 to 20%) is also a life threatening disease associated with a significant excess of mortality, it represents furthermore an independant predictive factor of mortality (vicious circle). The evaluation of these MR has to be complete: rest echocardiography plays a major and pivotal role: it permits the diagnosis (MR often silent) and their quantification: severe MR in ischemic/functional context has specific cut-off values (in comparison with organic MR) : ERO > 20 mm2 (vs 40 mm2), Regurgitant volume > 30 ml (vs 60 ml) and Vena Contracta Width > 4 mm (vs 7 mm), criteria classically considered as "moderate" MR. For the quantification of ischemic MR exercise echocardiography is critical and may increase the severity of there intermittent MR: it represents a diagnostic but also a prognostic value (when ERO difference is superior to 13 mm2). The Echo Dobutamine is more dedicated to assess myocardial viability and contractile reserve in ischemic/functional MR. MRI and/or MRI Dobutamine is also a critical tool to determine myocardial viability and almost precise the LV geometry (local or global deformation) and papillary muscle (PM) displacement. (post and/or ant). Despite the complexity and diversity of underlying mechanisms, Echo and/or MRI permits to classify ischemic MR in 2 main groups: asymetric (A) where the jet is eccentric, origin localised on P3 scallop with a local LV deformation (inferior) and PPM displacement with a moderate LV dysfunction and a symetric one (S) where the jet is central with a global deformation of an enlarged LV and both PM displacement with a seagull sign of the anterior leaflet (A2), with a severe LV dysfunction (EF < 35%). This last group of IMR (symetric) is very similar to functional MR by echocardiography. The tricuspid and RV assessment has not to be forgotten (annular dilatation ++) BNP may be also very useful in this group of patients to evauate more objectively their functional status.
Treatement of Isch/Funct MR is logical because it interrupts the vicious circle induced by MR on LV dilatation and dysfunction. The impact on mortality is still controversial. Because IMF/FMR is ventricular disease, the treatment has to be integrated in the scope of heart failure therapy. It is now very well demonstrated that beta blockers and/or resynchronization therapy may improve some FMR. Percutaneous approaches are being developed and might be useful in some group of patients, but it is still controversial and in evaluation. The surgical treatment is the most appropriate treatment to correct completely these MR but it requires a very precise and complete evaluation by the cardiologist: symetric or asymetric group, precise location of the jet (P3 or all post scallops), LV dimensions (particularly if ESD is above 55 mm), EF, results of stress testing, precise LV and subvalvular geometry by MRI, myocardial viability and contractile reserve, tricuspid annulus dilatation (> 40 mm).... In these conditions surgeons are able to provide the best surgical approach for an individual patient: in the asymetric goup of IMR undersized (2 sizes) or ischemic ring annuloplasty is logical. In symetric IMR and Functional MR undersized annuloplasty (2 sizes) or specific ring is also logical. The most important criteria to obtain in all groups of secondary MR is a coaptation distance > 8 mm measured intra-operatively by TEE. In some cases when LV enlargement is critical, LV procedures may be associated (CorCap, PM sling, Ventriculoplasty....), when there is a seagull sign of anterior leaflet (A2) chordal cutting has been proposed. Cellular cardiomyoplasty was also recently proposed.
Secondary MR is a severe disease which has to be precisely evaluated by cardiologists in order to provide critical informations for the best surgical approach. Percutaneous options and impact on survival are still controversial and need further prospective evaluation.

MITRAL REGURGITATION: THE EVIDENCE FOR INTERVENTION

Mr. B. Bridgewater – Wythenshawe Hospital, Manchester.

It is quite clear that symptomatic severe mitral regurgitation carries a poor prognosis if left untreated. Successful mitral valve repair can both eliminate symptoms and improve prognosis. Mitral repair, where possible, is superior to mitral valve replacement in terms of both operative mortality and long term outcome. All these issues are dealt with comprehensively in the ACC/AHA guidelines[1].

Decision- making in asymptomatic mitral regurgitation is less clear. Over the last couple of years there have been 2 important publications. Enriquez-Sarano et al[2] analysed a large series of patients with asymptomatic mitral regurgitation and found that the severity of MR as measured by effective regurgitant orifice area was an important predictor of outcome. They suggest that patients with an EROA> 40mm2 should undergo surgery irrespective of symptoms, LV size or function. Rosenhek et al[3] looked at a group of asymptomatic patients with severe MR and followed them by 'watchful waiting' and operated only at the onset of symptoms, atrial fibrillation, pulmonary hypertension, LV enlargement (LVESD>4.5) or LV dysfunction. The whole group had an outcome as expected and less than 1/3 of patient needed surgery over the period of follow up.

The updated ACC/AHA guidelines[1] give an LV size threshold for recommending intervention has decreased from 4.5 to 4.0 cm and they suggest that surgery is reasonable for asymptomatic patients with normal LV size and function in experienced centres in which the likelihood of successful repair with residual regurgitation is greater than 90%.

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NOTES

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.

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NOTES

MITRAL STENOSIS:

THE EVIDENCE BASE FOR SURGERY AND CHOICE OF PROSTHESIS

Professor M. Underwood – Prince of Wales Hospital, Hong Kong.

The normal mitral valve area is 4 – 5 cm and symptoms during exercise can occur when the area becomes <2.5 cm. Symptoms occur at rest when the area is <1.5 cm. The transmitral gradient is related to the square of the flow and the diastolic filling period, so that symptoms are worse during exercise, emotional stress, infection, pregnancy and fast atrial fibrillation. When symptoms are minimal the 10 year survival maybe as high as 80%, (60% may have no progression), but when symptoms occur at rest , survival becomes dismal at 0-15% over 10 years. If MS is untreated death is usually caused by heart failure (60-70%), systemic embolus (20-30%) and pulmonary embolus (10%). MS rarely causes sudden death.

Asymptomatic Patients.

1) Mild MS. (Area >1.5 cm^2 and mean gradient <5mmHg): observe with routine follow up and echo assessment.

2)Moderate to severe stenosis. (Moderate: area <1.5 cm^2 and mean gradient >5mmHg, severe <1.0 cm^2 and mean gradient >15mmHg). Consider valvuloplasty (if suitable morphology) particularly if PASP > 50mmHg or positive exercise test.

Symptomatic Patients.

All symptomatic patients considered for intervention. Group with symptoms but mild MS, stratify with exercise-testing first (rare group).

Mitral Valve Prosthesis :(risks of warfarin versus re-operation). Reports are varied in complication rates ie in one study the annual risk of embolism and / or complications of anti-coagulation in elderly patients (>65 years) was 0.36% for tissue valves (n=326) and 1.08% for mechanical valves (n=250), with 7 re-operations in the tissue group and 2 in the mechanical group. The risk of redo MVR is 4.7% compared to 4.1% first time, and pericardial valves now have <15% risk of failure by 15years. A meta analysis of the INR in patients with mechanical MVR shows that an INR >3 decreases the risk of thromboembolism significantly more than the increased risk of bleeding. Failure rates of bioprosthesis in the mitral position is higher than in the aortic position which may influence choice of mechanical prosthesis. Many patients with MS will be anticoagulated for indications and patient factors other than potential prosthestic replacement (around 80%).

NOTES

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.

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SURGICAL MANAGEMENT OF HEART FAILURE

STRUCTURE/FUNCTION RELATIONSHIPS BETWEEN FIBER ORIENTATION AND VENTRICULAR EFFICIENCY

Structural Considerations

The conical pattern of normal heart size and shape is well known since Hippocrates and Galen, and was described by the forefathers of anatomy More importantly, a helical spiral at the cardiac apex was demonstrated by Lower in the 1600's, an internal helix was postulated by Senec in the 1700's, and described as the Treibwerk by Krehl in the 1891 **(1).** Until recently, this helical intrinsic form was not proven, and inability to unfold the heart's basic structure was considered to reflect the "Gordian Knot" by cardiac anatomists. Studies by Francisco Torrent-Guasp began in 1950 **(2)** and have finally unraveled the cardiac structural pattern. Torrent-Guasp spatially unfolded the intact heart to demonstrate a rope like model, that contains a myocardial fold that separates heart structure into two simple loops named the basal and apical loops . The muscular myocardial band begins at the pulmonary artery and ends at the aorta. The structural components include a horizontal or transverse fiber orientation for the *basal loop* that surrounds the right and left ventricles, and a change in fiber direction through a spiral fold in the ventricular band to cause a ventricular helix that now contains obliquely oriented fibers that form a descending and ascending segment of the *apical loop* with an apical vortex. These components and the implications of this model were discussed in a recent NIH workshop (3)**.**

Surgical correction of the dilated heart requires changing the spherical configuration architecture into a more normal elliptical form **(4;5)** The architectural patterns of the normal and dilated hearts define a) the *transversely* oriented fiber arrangement of the basal loop that surrounds the normal helix that is comprised of an apical loop with *obliquely* oriented fibers reciprocally arranged in an approximate 60 degree angle and b) the spherical geometry of a failing heart, whereby the basal loop is intact but stretched, and continues to surround a more spherical fiber arrangement of apical loop fibers that develop a more transverse fiber arrangement , so that the fiber orientation of the apical loop becomes less oblique to now more closely resemble the horizontal basal loop. Examples of this rearrangement of apical loop fibers to a more transverse ,or circumferential pattern will be shown from the corrosion cast studies of Gorodkov in Moscow. A form related background for rebuilding ventricles that stems from the spatial orientation of cardiac fibers that suggest the failing ventricle becomes functionally impaired when the normal obliquely orientated helical heart fibers become more horizontal when muscle stretches into the spherical form. **(6;7)**

Structure /Function

The bioengineering infrastructure for this mechanical change in size and shape is rebuilding more oblique fiber orientation. This anisotropic configuration requires a conical form, whereby the increased deformation responsible for contractile strain improves from the widened base to the helical apex vortex (**(8)**). The pattern of ejection and filling are related to a sequential twisting of the LV to eject and rapid untwisting to suction venous return to rapidly fill **(9).** These normal twisting patterns were originally described by Borelli in 1660 (10)**,** and are visible at operation or by MRI recordings.

Basic scientific studies set the precedent for this geometric component, by establishing how the normal 15% fiber shortening of isolated muscle strands is changed when an integrated fiber orientation exists within the intact heart **(11;12)**. Ejection fraction is 30% if fiber orientation is transverse, and increases to 60% with oblique fiber direction, as deformation increases during transition from midwall to apex. **(11)**. This functional pattern is evident on DENSE MRI studies that displays the transverse and oblique fiber orientation of the basal and apical loops. More importantly, the stretched dilated heart loses the normal twisting pattern, and a constriction and dilation pattern define functions to correspond to the stages that were was deduced by William Harvey from pivotal anatomic but not functional dissections that showed the pulmonary circulation (15)**.**

These global considerations apply for normal fibers, whereas damaged stretched hearts contain sites of fibrosis or excess collagen formation that can limit the expected improvement after changing ventricular form. A clear contrast between ischemic and non ischemic dilated failing ventricles is provided by precise infarct location that identifies the abnormal regional site after coronary occlusion. In this ischemic cohort, the remote, non infarcted muscle stretches to allow compensatory function. Conversely, a more global process exists in non ischemic patients, whose architecture has variable site location (16-18). This inhomogeneity in a global process must direct efforts at uncovering the predominant region responsible for chamber stretching, and simultaneously uncovering how remote regions with less intrinsic disease compensate to support cardiac output. Localizing major segments of intrinsic disease is essential, since efforts at rebuilding must concurrently provide a) an elliptical shape to the abnormal spherical structure, and b) exclude predominant regions of damaged muscle fibers responsible for causing this global widening.

From a sports perspective the normal heart is like a football, where a spiral motion is generated from passing the elliptical shaped contour. Conversely, the basketball, with a spherical configuration defines the shape of the dilated heart, whose efficiency is diminished by abnormal form, and this ineffective action is independent from the person passing the ball ; structure, not player is at fault .

A biologic example of the sports paradigm defines the elliptical pattern of the normal heart with a football like shape, the spherical architecture of an ischemic heart were a basketball like shape results from infarction of the apico-septal regions, and this form leads to restoration to surgically rebuild the elliptical form to return architecture back towards the normal football like configuration.

Implications with Helical Fiber Orientation

The helical architecture of the normal heart has been confirmed by strain relationships using MRI (19), corrosion casts showing spiral architecture,(20) and by sonomicrometry crystals (21); each pattern reflects the normal oblique fiber orientation that conveys maximum force during ejection and suction. These observations coincide with the helical heart configuration, and change when dilation alters this architecture because of flattening of the double helical arms of the apical loop.

Prior sonomicrometer crystal recordings document the importance of obliquity to achieve the maximum extent of directional shortening (21). This functional framework nicely coincides with conceptual determinants of efficiency of ejection fraction (EF) , 60% EF is expected with oblique fiber direction, a value that falls 30% EF with transverse or horizontal fiber direction (11). Although fiber orientation is oblique, recordings from crystal tracings seem to reflect spiral coils within the fibers (*or coils within coils*) to obtain maximal efficiency (22). Conversely, reduced shortening develops with a more horizontal position for crystals placement (21).

These shortening patterns closely link with changes in ventricular shape following reconstruction methods that address "disease versus form". The patch position is flatter in ischemic disease when the scar becomes the only marker during ventricular shape rebuilding and may result in a more spherical chamber. Conversely, a more conical chamber is created when "form" becomes the guideline for patch placement; obliquity now becomes the marker for patch insertion and conceptual goal of the insertion policy. Future comparisons of ventricular function by MRI tagging are needed to define the extent of deformation, and evaluate the validity of this form reconstruction objective.

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PATHOPHYSIOLOGIC IMPLICATIONS OF THE VENTRICULAR MYOCARDIAL BAND (Helical Heart)

The conical pattern of normal heart size and shape is well known since Hippocrates and Galen. More importantly, a helical spiral at the cardiac apex was demonstrated by Lower in the 1600's, an internal helix was postulated by Senec in the 1700's, and described as the Treibwerk by Khrell in the 1800's(1). Until recently, this helical intrinsic form was not proven, and inability to unfold the heart's basic structure was considered to reflect the "Gordian Knot" by cardiac anatomists. Torrent-Guasp spatially unfolded the intact heart to demonstrate the presence of a myocardial fold that separates heart structure into two simple loops named the basal and apical loops that start at the pulmonary artery and end at the aorta.(2) The structural components include a horizontal or transverse fiber orientation for the *basal loop* that surrounds the right and left ventricles, and a change in fiber direction through a spiral fold in the ventricular band to cause a ventricular helix that now contains obliquely oriented fibers that form a descending and ascending segment of the *apical loop* with an apical vortex.

Structure / Function

Understanding of myocardial function must incorporate knowledge of underlying structure to allow a clear structure / function relationship to emerge, with a key ingredient that relates into how fiber orientation of the septum impacts right and left ventricular performance. The presentation will to 1) define the components of septal architecture and demonstrate that they are explained by the model of the helical ventricular myocardial band **(3)** 2) demonstrate the structure function relationship that results from preservation of the normal anatomic framework, 3) describe how distortion of this anatomic framework by lesions of the left or right side of the heart can impair biventricular function, 4) define how stretch of the septum with bowing of its thick structure into either left or right ventricle can create an "architectural disadvantage" that impairs septal contractile function, 5) indicate how restoration of normal anatomy will return septal function toward normal and thus improve biventricular performance, providing excitation contraction coupling is not impaired, 6) identify the importance of a sequential activation of the septum and it's effect on cardiac dynamics, especially in regard to resynchronization, and 7) demonstrate why the septum is the "motor of biventricular function", and "the lion of right ventricular function.

Sallin **(4)** and Ingels **(5)** furnished physiological studies that confirmed the importance of fiber orientation, by relating the effects of different angulations to expected ejection fraction. Basic science studies of myocyte function done on isolated fibers from muscle strips in a Petrie dish show that the maximal shortening of these fibers is 15%. The intact heart, however, has a continuum of muscle wrapped in a pattern that alters fiber orientation in different myocardial regions. Ejection fraction is 30% if the fibers are transverse, as occurs in the free wall of the right ventricle and increases to 60% if there is predominant oblique fiber orientation, a pattern that comprises the septum and free wall of the left ventricle beneath the

external wrapping of the left segment of the basal loop **.** The characteristic motion of twisting of the septum is linked to the anisotropic form created by this oblique fiber orientation **.** *Fiber orientation therefore accounts for both aspects of RV function that includes a) bellows like action resulting in compression, caused by the transverse basal loop and b) twisting due to oblique sequential septal contraction.*

The twisting action disappears when the cardiac chamber dilates and the septum gets bowed into the right ventricle by left sided lesions such as aortic or mitral insufficiency, dilation from ischemic or non ischemic congestive heart failure, or rhythm interruption from wide QRS or left bundle branch block. Similarly, right sided lesions like pulmonary insufficiency, atrial septal defect, or pulmonary outflow tract obstruction bow the septum toward the left side. The central theme **(6;7)** is that each event disrupts normal architecture by stretching the septum so that a more transverse, rather than oblique fiber orientation comprises it's spatial configuration and subsequently disrupts it's sequential twisting function needed for maintaining cardiac output into an increased resistance vascular bed. Of great importance to the cardiac surgeon is the introduction of septal dysfunction due to "septal stunning " that arises from impaired protection strategies during correction of underlying mechanical defects.

Clinical Examples

Two examples in the category of congenital heart disease include a) early or late development of right heart failure following a procedure that reduced pulmonary hypertension by correcting a defect, and b) septal dysfunction by stretching from a volume overload, as with pulmonary insufficiency.

In the first instance, the septum had normal function preoperatively , despite pulmonary hypertension. Loss of the septal twist from stunning will either 1) limit right heart performance intra operatively if pulmonary hypertension persists, or 2) evolve postoperatively if pulmonary pressure is lowered, as the RV constrictive property of the basal loop is preserved to maintain efficient cardiac output. . However, RV failure supervenes in the ICU if delayed pulmonary vasoconstriction, because twisting capacity is impaired by septal stunning. The solution is to study septal motion intra operatively, and determine if the method of protection allows normal post operative function. If it does not, an alternate protective method should be sought.

In the second instance, septal stretch by RV volume overload will alter fiber orientation by bowing, and impair septal twist. This is frequent finding in patients with pulmonary insufficiency and right heart failure after repair of Tetralogy of Fallot, and clear from preoperative absence of septal shortening or late thickening by echo study. The solution to this "architectural septal stretch disadvantage" is supplementation of procedures that are used to restore valvular competence , with new approaches that

include restoration of the septum into a mid line position , while avoiding it's injury by safe protective techniques. . A " ventricle-valve" approach is needed, and an example will be shown(8).

Implications

Understanding of the central role of the septum in RV function provides the theoretical basis for treatment of entities such as right ventricular infarction and post cardiac surgery RV dysfunction. Knowledge of the anatomy and physiology of the septum also allows the rational design of operations to treat various cardiac conditions such as RV dysplasia, RV failure from pulmonary insufficiency, transplantation RV dysfunction, right sided congenital defects that affect LV function, techniques of myocardial protection, planning physiologic treatment of RV dysfunction by use of a structure / function relationship, and for left ventricular restoration. The septum is the "lion of RV function" and further understanding of its role as a component of the helical ventricular myocardial band will alter thinking about surgical management.

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THE ROLE OF RESTORATION, REVASCULARISATION AND REPAIR

"SURGERY FOR HEART FAILURE" OR

"CAN YOU MAKE A SILK PURSE OF A SOW'S EAR?"

Professor J. Dark – Freeman Hospital, Newcastle.

Heart failure is common, an increasing burden on health care, and with an ageing population, will become a greater problem. The number of patients in the USA at the severe end of the spectrum (estimated at 200000) is 100 times greater than the annual number of heart transplants (approx 2000). This ratio will be even worse in the UK, where we perform only a quarter as many transplants. In both countries, this procedure is already restricted to the sickest patients, usually in hospital and on inotropes. Mechanical devices do not yet have the requisite durability and biocompatibility to be more than temporary solutions.

The effectiveness of "medical therapy" with a range of proven drugs, but also including resynchronisation and implantable defibrillators, continues to improve, certainly with regard to survival. But there is clearly room for additional measures, and surgical options are repeatedly proposed for some of these patients.

There is no doubt that for modest degrees of cardiac dysfunction, correction of obvious lesions – critical coronary stenosis or significant mitral regurgitation - is of benefit both in terms of relieving symptoms and improving life expectancy. Correction of severe MR with valve repair, even in asymptomatic patients, restores normal life expectancy; uncorrected patients have more heart failure and a poorer prognosis. There are many who claim that this improvement, particularly in LV shape and size – described as "remodelling" – can be extended to more severely affected patients. The rationale is sound – reduce preload, allow the ventricle to be smaller, and the survival will be better. But does it work for the most severely affected? Is it a substitute for transplant, and can it delay or avoid the future need?

These are two separate questions, but answers to both are lacking. Dion's group have shown that reduction mitral annuloplasty is effective for both ischaemic and non-ischaemic cardiomyopathy with significant MR, but only up to a certain size of ventricle. The worst patients neither re-modelled their ventricles or had a survival benefit. We do not know whether the better patients had a survival benefit, but some, admittedly retrospective data from the US suggests they did not. For those with the biggest ventricles, one option is to add a restraining device. In his description of the Acorn trial, Acker states that "…CSD (cardiac support device) reverses the natural history of heart failure..and represents a new and effective approach for patients with enlarged hearts" But the improvements were modest. Of those with concomitant mitral repair, only 5% were in NYHA. This trial showed that these patients can be operated on with very low risk (1.3% mortality) but gives us very little data on the sickest patients.

The same arguments extend to "LV restoration" the collective term for operations which re-shape the ventricle, of which the Dor procedure is now the best known. This is principally applied to the sequelae of large anterior infarcts, and improvements in LV size and NYHA grade are claimed. But the studies are single centre, with an undeniable selection bias, and retrospective. A recent Brazilian paper suggests that restoration with CABG for viable myocardium is better than either procedure if isolated. But entry criteria were restricted only to EF<50%!
Thus at the earlier stages of heart failure, surgical results are good, with low mortality, and *some* evidence of geometric advantage. For many patients there may be symptomatic advantages. But the suggestion that prognosis is improved and later failure prevented is entirely unproven, and will remain so until prospective trials against the *best* of medical therapy can be performed.

For the most severely affected, the evidence is either lacking, or suggests little advantage to a high-risk procedure. The history of the Battista operation is a lesson for all in this field.

If the myocardial reserve is too depleted to achieve a good result, can we add to it. Autologous stem cells,

principally injected at the time of myocardial infarction, but also used in conjunction with conventional surgery, clearly have some effects. But the benefits have been very limited, and no one approach has a proven role.

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Improved clinical outcome after intracoronary administration of bone-marrow-derived progenitor cells in acute myocardial infarction: final 1-year results of the REPAIR-AMI trial

Schächinger V, Erbs S, Elsässer A et al, for the REPAIR-AMI Investigators

European Heart Journal 2006 27(23):2775-2783;

TRANSPLANTATIONF AND VENTRICULAR ASSIST DEVICES

Mr. D. Jenkins – Papworth Hospital, Cambridge.

Heart transplantation is the best treatment for patients with end stage heart failure in whom the predicted survival without transplantation is less than 2 years. As the number of donor hearts is limited, recipient selection is important to allow the best utilisation of this scarce resource. The majority of patients have dilated or ischaemic cardiomyopathy. Advanced age, malignancy, raised PVR, active infection and other end organ failure are contraindications to heart transplantation. In the UK adult heart transplantation is performed at six designated centres each covering a local region for donor organs to limit ischaemic time. The donor heart should be blood group compatible and the donor should match the recipient in size. Care is needed in assessing the donor heart function after brain stem death. The donor heart is protected by cardioplegic arrest and hypothermia during transport and there is an increasing failure rate with prolonged ischaemic times. The standard implant operation has changed little since the description by Lower and Shumway, with a left and right atrial cuff anastomosis, anastomosis of the main pulmonary artery and the ascending aorta. Advances in immunosuppression and perioperative care have transformed outcome with up to 80-90% 1 year survival in the best centres. Early complications include primary graft dysfunction, acute rejection and infection. Longer term survival is limited by allograft coronary artery disease.

Ventricular Assist Devices (VADS) offer mechanical support to the failing left (or right/or both) ventricle to restore 'cardiac' output and organ perfusion. There are now many devices available (current third generation) and they can be classified into implantable/extracorporeal and pulsatile/continuous flow. In the UK three centres are designated to provide VAD therapy. In most cases VAD support is used as a 'bridge' to transplantation in situations when a heart is not immediately available, or when raised PVR or other temporary end organ dysfunction precludes heart transplantation. There is some evidence for recovery of cardiac function in a minority of patients supported with VADS. As VADS become more reliable and donor organ availability continues to decline the prospect of VAD support as permanent chronic therapy becomes real, although at present there is not enough evidence to justify the resources necessary. The outcome depends on the patient population supported and complications include bleeding, infection, embolism and device failure. Short term simple devices can be used in post cardiotomy patients and occasionally those in multiorgan failure.

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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.bmjjournals.com/cgi/collection/heart_education
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

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NOTES