NOTES

CARDIAC TRANSPLANTATION: YESTERDAY'S THERAPY

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Since the introduction of Cyclosporin for immunosuppression in the early 1980's, cardiac transplantation has been the gold standard for the treatment of end-stage heart failure. No other therapy could rival the early success rate, physiological normality and long term survival stretching out over two decades. Xeno-transplants, cardiomyoplasty and the early LVADs either never appeared or were inferior alternatives.

But with the epidemic of heart failure, particularly in the elderly, transplantation was never going to do more than scratch the surface of the problem – an expensive and complex luxury for the lucky few. Activity is falling in all the Western countries; in the UK, from a peak of more than 300 transplants in the late 80s, we now do little more than 100 per year in adults. The same pattern, though not to the same extreme, is seen in Europe and even the USA.

Medical treatment advances – ACEI's, ARB's, B blockers, Spironolactone, CRT – have resulted in a progressive improvement in survival and quality of life. Perhaps because of this, with sicker patients eventually coming to transplant, results have not improved for 20 years. Expensive innovations – new drugs in particular- have had no real impact

Most importantly, mechanical devices are catching up. That the first generation were primitive was demonstrated in the REMATCH trial; a two year survival of 25% was regarded as "success" and resulted in FDA approval for the original Heartmate. But the next generation of continuous flow pumps was much better. Miller's paper describing 130 patients with the Heartmate 2 device showed a 75% six month and 68% one year survival in patients being bridged to transplant. After transplant, 85% survive one year. And the third generation, with examples such as Ventrassist, or Heartware, are better still. Publications are now coming out of the Intermacs Registry, a real attempt to escape from the inevitable bias of institutional publications. Bridge to transplant survival is approaching 80% at six months. More notably, the "destination therapy" patients, usually older, with no transplant intention, have a very similar outcome. Most of the deaths are within the first month, with curves then going flat, a convincing demonstration that many of the biocompatibility problems have been largely solved. Physiological performance can be excellent.

Finally, there is the tantalising prospect of myocardial recovery, realised in one series, but yet to be emulated.

Not all is perfect. The current generation of devices support only the left ventricle, and can't be applied to severe biventricular failure. Many of the adults with complex congenital disease will still need a transplant. So transplantation, for the lucky ones, is still the best in 2009, but not for much longer!

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THE TREATMENT OF CHOICE FOR AORTIC ARCH REPAIR IS OPEN

REPLACEMENT

TAVI: PATIENT CHOICE, TECHNIQUES AND RESULTS

RECERTIFICATION – AN INTERNATIONAL PERSPECTIVE

THE STICH TRIAL: IS THIS THE DEATH-KNELL FOR LEFT VENTRICULAR REMODELLING SURGERY

RADIO FREQUENCY ABLATION

A sample operation note for aortic dissection repair

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

DIAGNOSIS: Type A aortic dissection. Aortic regurgitation. Acute renal dysfunction.
Thrombolysis 48 hours pre-operation
OPERATION: Aortic valve resuspension, ascending aortic replacement

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

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

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

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

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

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

MYOCARDIAL AND CEREBRAL PROTECTION: Intermittent antegrade cold blood cardioplegia applied approximately at 20 minute intervals. Cooled to 15^{o}C. Pre-treatment with dexamethasone 100mg and mannitol 1g/kg prior to circulatory arrest. Prior to circulatory arrest, the patient was placed in a 15^{o} Trendelenberg position to allow an open distal anastomosis construction during a 27 minute period of hypothermic circulatory arrest. During cooling, the head was packed in ice. At circulatory arrest, anaesthetic infusions were discontinued and restarted following the arrest period. Rewarming was undertaken with similar maximal temperature gradients and α-stat pH management strategy. The maximal blood outflow temperature was not allowed to exceed 37^{o}C. Bypass was discontinued after a naso-pharyngeal temperature of 36^{o}C had been reached and maintained for 10 minutes.

FINDINGS: There was a haemopericardium with approximately 300ml of blood and clot evacuated on opening the pericardium. Heart action appeared satisfactory apart from some minor depression of RV free wall motion. The ascending aorta was dilated (approximately 4cm) and had a purplish discoloration compatible with acute dissection. There was epicardial-epi-aortic haematoma within the atrioventricular groove near the right coronary artery and between the aorta and pulmonary artery. When visualised, the aortic valve was tricuspid and tri-commissural and morphologically normal. There was an acute dissection arising from a transverse intimal tear 2 cm distal to left coronary ostium. Retrograde dissection into the aortic root and led to a loss of commissural support for right/non- and left/non-coronary commissures. Coronary ostia normally positioned but the ostium of the right coronary artery was dissected without breaching of the intima. Examination of the aortic arch revealed a small (2cm) further transverse tear in proximal aortic arch opposite the innominate artery on the under-surface. The calibre of the aortic arch was normal. The dissection had generated a large intimal flap involving all epiaortic vessels with dissection of their ostia. The intima within the ostia was not breached.

PROCEDURE: Following institution of bypass and initiation of cooling a soft cross-clamp was applied to the distal ascending aorta during a period of reduced pump flow. Once pump flow was restored, there was marked (>30mmHg) discrepancy between left and right radial artery pressures (right lower) *(suggestive of malperfusion of at least one of the epiaortic arteries)*. The cross-clamp was therefore removed allowing equalisation of right and left radial pressures. Cooling was continued until the temperature nadir was attained. In Trendelenberg position, the circulation was arrested and the aorta opened obliquely. The opened aortic arch was carefully inspected taking care not to over-aspirate blood within the epiaortic vessels. Antegrade cardioplegia was instilled directly via the coronary artery ostia to achieve complete electro-mechanical arrest. The ascending aorta was then transected just proximal to the innominate artery with line of section excising the arch tear. A wet arterial swab was temporarily placed within the arch to prevent glue spillage and the dissected layers of the aorta were then gently separated to allow instillation of glue between the layers for a distance of approximately 2-3cm within the arch. The dissected layers were held in apposition with soft forceps and clamps until the glue became fixed. The swab was then removed and the any intimal glue residue aspirated. A 0.75cm wide strip of Teflon was then sutured around the open arch using 4'O' prolene, commencing at the most leftward extent of the opened aortic arch and working towards the

surgeon. Great care was taken to apply the strip of Teflon to the outside of the aortic wall rather than the transected edge. A 30mm protein-impregnated polyester graft with a single side-arm was then trimmed and bevelled. The graft was trimmed and orientated so that the side-arm would lie on the anterior-inferior aspect of the graft approximately 2cm from this distal anastomosis. This graft was then anastomosed to the buttressed aortic wall applying a further buttress on the graft side.
Quite deep bites were taken into the graft to allow partial telescoping of the graft into the aortic arch. The direction of suturing was again from most distally towards the surgeon suturing the inferior wall first. Sutures were applied loosely and later tightened using a small nerve hook. Great care was taken to ensure that buttressing was on the external aspect of graft and aorta and not within the meeting edges of the suture line. Once this anastomosis was ¾ complete leaving only the right-sided anterior aspect to be completed, the arterial return limb of the bypass circuit was transferred to the graft side-arm using a separate cannula. This side-arm was de-aired by minimal forward pump flow. An infusion line was passed out to the anaesthetist and inserted into the proximal open end of the graft and an infusion of 4^{o}C cold saline solution gently infused into the graft to allow de-airing during the completion of the anastomosis. Once complete, the saline infusion was stopped and forward bypass flow gently recommenced within an open graft. The graft, aorta and epiaortic vessels were massaged and agitated to complete arch airdrill. The graft was then clamped and bypass flow restored and rewarming commenced 5 minutes after ending the arrest period. With pump flow restored; this distal anastomosis was carefully inspected for haemostasis, moving the graft to allow full inspection under pressure. No suture line bleeding or oozing was accepted and several additional Teflon buttressed sutures were inserted during periods of temporarily reduced flow were placed to achieve absolute haemostasis. Once this was achieved, attention was directed to the proximal aorta. Cardioplegia was re-administered and the aorta transected 1cm distal to the sino-tubular junction taking care to ensure that the adventitial-medial outer layer was not over-excised and compromised. The ascending was then excised completely using cautery ensuring the safety of the main and right pulmonary arteries. The aortic valve was re-suspended with 3 Teflon buttressed 4'O' prolene mattress commissural sutures which were not tied until glue apposition of the dissected root layers was complete. A small wet swab was then inserted into the root to protect the valve and coronary ostia and glue applied between the dissected layers including around right coronary ostium The layers were then held in apposition with soft forceps and clamps until the glue became fixed, intermittently applying a right angled clamp into the right coronary ostium to ensure that its mouth was not compromised by an excess of glue. Following this, an external 0.75cm Teflon collar was applied to outer wall of the sinotubular junction using a running prolene suture working towards the surgeon. A separate length of the 30mm polyester graft was then anastomosed to sino-tubular junction with a further buttress on the graft but not on the aorta. This suture line was initially constructed loosely and tightened later with a fine nerve hook ensuring the siting of the buttress sutures. A small hole was then made in the graft 2-3cm from the right coronary ostium and a cardioplegia cannula inserted and secured with a buttressed mattress suture used as a purse-string. Antegrade cold blood cardioplegia was then instilled, the graft de-aired and then clamped. The delivery of pressurised antegrade cardioplegia allowed inspection of this proximal anastomosis and insertion of additional haemostatic sutures as necessary. The two grafts were then trimmed to appropriate length and anastomosed together using Teflon buttressed 3'O' prolene. The integrity of this anastomosis was tested using pressurised cardioplegia.
Weaned from bypass in SR but relapsing into AF periodically requiring cardioversion. Good heart action and output. On table trans-oesophageal echocardiogram revealed trivial aortic regurgitation only.
Haemostasis, protamine and decannulation.
CLOSURE: Wires & Vicryl. 2 drains and Redivac drain peri-graft. 2 RV & 2 RA pacing wires.

Bonus questions:
Which British monarch died of acute type A dissection ?

What is the Euroscore for this patient ?

Inserted PPT entitled CABGaug09book

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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IMPACT OF CO-MORBIDITIES ON OUTCOMES FOLLOWING CARDIAC SURGERY

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1. Carotid Disease & the Role for Carotid Surgery

Stroke occurs in ~2% of patients undergoing CABG. Analysing 13 series containing 484 CVA / 36,797 CABG procedures 38% occur early (<24hrs) and 62% late (within 7 days) and carotid disease (CD) may be responsible for 40% of all post-operative strokes.

Symptomatic CD (i.e. with history of TIAs etc) is associated with the highest risk of post-operative CEA. In patients without IHD, The North American Symptomatic Carotid Endarterectomy trial (NASCET) demonstrated a significant reduction in event free survival and subsequently the Asymptomatic Carotid Surgery trial (ASCT) concluded that for patients <75years with a asymptomatic stenosis >70% benefited from CEA.

There is no prospective randomised control to guide treatment of the patient with asymptomatic significant carotid disease awaiting CABG, and interpretation of data comes from historical controls or from known patients with CD undergoing CABG, and there has been little publishes data since the 2004 AHA guidelines. These advocated pre-operative CEA on the basis of a >11% risk of stroke with >80% carotid stenosis, and a >20% risk of stroke with bilateral disease compared to pre-emptive CEA stroke rate of 4% and a mortality of <3%, but again this is based upon data from papers of 10-20 years ago.

A number of meta-analyses have been carried out on this topic by Prof Naylor from Leicester. He concluded that although 40% of peri-operative strokes occur due to CD, the risk of MACE from pre-operative CEA (mortality / stroke/ MI) of 11%, there is 'no systematic evidence that staged or synchronous operations confer any benefit over CABG alone. Although the evidence base for performing CEA in the asymptomatic patient prior to CABG is lacking, in departments where staged CEA performed under LA can be carried out with low risk of MACE then this approach may be considered reasonable in an attempt to reduce the incidence of peri-operative stroke. A randomised trial is clearly needed.

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2. Renal Impairment

Patients with mild renal impairment have a 10-20 fold risk of cardiac death compared to matched controls and renal impairment affects over 10% of the general population but remains undiagnosed in 80-90% of cases. The main surgical risk stratification models only recognise a Creatinine >200µmol/l or >2.26 mg/dL as independent risk factors. Creatine however is an insensitive measure of renal function. Creatine clearance can be calculated from the serum creatine taking into account age, sex, ethnicity and body mass. Chronic kidney disease, defined as a CC <60mls/min has been shown to be an independent predictor of increased early mortality, an increasing complication profile including low cardiac output state requiring inotropes, bleeding and renal failure. In addition it has been shown to negatively influence late survival. The addition of creatine clearance to the EuroSCORE may improve model fit.

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3. Chronic Lung Disease

Initial work by Samuels demonstrated that patients with COPD, defined as requiring long term treatment or FEV1 <75%, had significant increased hospital mortality and decreased late survival following CABG. Further work by Further work by Fuster demonstrated a stepwise increase in mortality with decreasing lung function 0.9% in patients with FEV1: >80%, 0.4% in FEV1: 60–80%, 10.8% in FEV1: 40–59% and 54% in FEV1: <40%. Additionally the Northern New England group analysing outcomes in 33,137 patients undergoing isolated CABG demonstrated that patients with both COPD and at least one other significant co-morbidity had very poor late outcomes indeed.

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4. Diabetes, Extremes of BMI, Smoking & Social Deprivation

The link between poverty, socioeconomic inequalities and increased mortality is well established and cardiovascular disease is the commonest cause of premature death in the UK. We have shown that social deprivation has a substantial independent negative effect on both in-hospital survival and late survival following cardiac surgery.

Smoking (current or ex), extremes of BMI and diabetes also affect survival following cardiac surgery. They are strongly related with social deprivation. Adjustments for these factors reduces the effect of deprivation by ~ 30% but does not abolish the effects of deprivation on survival.

Pagano et al. BMJ 2009;338:b902

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

NOTES

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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RECENT EVIDENCE ON THE VALUE OF CAROTID SURGERY PRIOR TO CORONARY SURGERY

Much of the background to this debate springs from the 2004 AHA Guidelines[1], which very firmly advocate pre-operative carotid endarterectomy (CAE) as being a "proven" manoeuvre to reduce the risk of post-operative stroke. This conclusion is reached by examining the risk of stroke with known carotid disease, quoting papers from 12 and 14 years ago. Risks are said to range from 10% when stenoses are between 50 and 80% , and 11% to 18.8% with stenoses greater than 80%. If there is bilateral severe disease, or an occlusion with severe contralateral stenosis, the risk is 20%. This last observation comes from 1984. The numbers in these studies are small. On the other hand, there are three separate studies quoted showing that CAE done pre-operatively can have a stroke rate of less than 4% and a mortality rate of 3%. Ergo it must be safer than doing nothing!

The same guidelines also recommend epi-aortic echo to detect mobile plaque. They state that 1 in 3 strokes are probably related to aortic plaque, and echo, with subsequent manoeuvres to reduce aortic manipulation, has been shown to reduce stroke rate.

They identify post-op AF as increasing the stroke rate by a factor of 2-3, and recommend anticoagulation with heparin and then warfarin for AF that persists for more than 24 hours.

Few centres in the UK routinely do either epi-aortic echo or early anticoagulation for AF, despite these being recommendations from the AHA.

Finally, they document the increase of stroke risk with age, suggesting the risk is 2-6% at 70, and between 8 and 10% at 80.

All of these risk factors, and indeed much of the literature, is from papers 10-20 years old. The stroke rate in Newcastle 20 years ago, from Pam Shaw's 1985 study, was 5%. She studied a group of 312 patients, but the mean age was 53.4 and the oldest was just 70. Our rate last year was 1.9%, admittedly from the PATS database, but with a much older mean age of 67.

Relatively little has been published since the AHA document, but there is very good, critical analysis of the literature from Prof Ross Naylor, a vascular surgeon in Leicester. He has done a number of detailed systematic reviews[2,3]. The conclusions are best summarised in his 2004 paper. Although carotid disease is clearly an important aetiological factor, there is little published data on the risk of *ipsilateral* stroke in a screened patient with disease who undergoes CABG alone. It is likely that carotid disease is responsible for no more than 40% of strokes. On the other hand, from the literature, 9-11% of patients who undergo a staged or synchronous procedure will die, have a stroke or an MI. Naylor states "***Because of the paucity of natural history data no systematic evidence exists that staged or synchronous operations confer any benefit over CABG alone."*** If a procedure is done, there is some evidence to suggest that staged, ie CAE followed by CABG is safer than synchronous procedures are safer, although this may just represent a trend to select the sickest patients for combined operations.

There are two more recent publications worth noting. Dubinsky and Lai analysed the risk of CEA plus CABG compared with CABG alone, from a large database – the US Nationwide Inpatient Sample[4]. They had data on about 180,000 CAE patients and just under 500,000 CABG's, with risk-stratification data on both groups. Between them, there were 1230 done on the same day and almost 6000 on the same admission. The Odds Ratio for death or stroke was more than 2.0 for all the combined procedures – this is not surprising, given the high risk nature of patients needing two procedures. It was the same whether on the same day or same admission.
But after controlling for all the risk factors, the odds ratio was still 1.38, which for a population of this size,

is a very significant risk. Whilst the methodology may be flawed, the numbers are huge, and the paper was accepted by a major journal. The conclusion was that a randomised study is required.

In the real world, there is a fascinating small study from Sheffield[5]. It comes from a group of neurologists and neuro-radiologists who work with cardiac surgeons, some of whom demanded intervention for carotid disease pre- CABG. Since 1998 they have had a policy of carotid stenting in this setting, and report on 52 patients who had this intervention. Criteria for stenting were rigorous - >80% stenosis on the dominant hemisphere, >150% combined stenosis, symptomatic stenosis>70% or an occlusion with contralateral stenosis >50%. There were no strokes related to stenting. Three patients died before surgery, despite no patient waiting more than 2 months. There were 3 non-fatal and 2 fatal strokes, together with one other cardiac death, in the 30 days post surgery. All the non-fatal strokes were ipsilateral to the stent.

In this small group of patients, there was a combined major stroke, minor stroke and death rate of 19%! All of the complications were due either to surgery or delay in surgery, and there was an 8% ipsilateral stroke rate despite stenting!. The authors again suggest this can only be resolved with a multicentre trial.

There is no doubt about the situation with regard to *symptomatic* patients; urgent treatment is required. The benefit from surgery is greatest is within the first two weeks of an ischaemic event. In the trials of symptomatic patients, five patients were required to undergo CEA to prevent one stroke in the first two weeks. The figure for patients treated after 12 weeks was 125 patients![6]

The patient without neurological symptoms with planned cardiac surgery represents a conundrum. Surgery may be delayed because of a perceived need to investigate asymptomatic patients. If carotid disease is found there is then the question of what to do about it. The various large studies of asymptomatic patients have established a role for endarterectomy (but not yet for stenting) in preventing a stroke over a *5 year* time period. There is about a 3% chance of stroke or death in these patients if there is no cardiac disease, so it is probably higher in our patients. In a recent review of largely asymptomatic patients referred for carotid stenting prior to CABG there was 4.7% incidence of death or stroke associated with the stent alone, and a combined death and stroke rate of 12.3% for the two operations![7] These authors yet again suggested the need for a trial

There is no good evidence that any intervention on the carotids will reduce the peri-operative risk of stroke for cardiac surgery.

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Insert PPT entitled: TAG2

SO, WHAT IS THE ROLE OF CARDIOPULMONARY BYPASS IN SURGICAL REVASCULARISATION?

Depending who you address this question to, your answer will range from "none" to "essential". Off pump CABG usage appears to have levelled out at between 25% to 30% of CABG activity in the US and ≈20% (and possibly falling) in the UK.

Previously, cardiac surgeons have been proud to develop their practice utilising the available evidence base (e.g. adoption of LIMA over saphenous vein as conduit of choice for LAD grafting).

Given these facts, one can, perhaps, make one of several conclusions:

1. Some cardiac surgeons are, for some reason, ignoring current evidence of an overall superior effect of off pump surgery.
2. There is still a perception that the case for off pump is yet to be proven.

My assessment of the large amount of published data on this topic is that it is very confusing! The randomised trials thus far completed have demonstrated broadly comparable outcomes for on and off pump surgery.

Some studies have demonstrated improvements in certain aspects of care with on and off pump surgery. These will be discussed during my talk.

There are very few surgeons who when referred a patient would have no preference as to whether to treat him/her with either off and on pump surgery. This means we all, quite rightly, have an opinion. On occasion, these opinions can become quite firmly held beliefs. This fact alludes to one of the difficulties that has been encountered during the development of off pump surgery; one person's "early adopter" is another's Zealot. This taking of positions has, in fact, sometimes discouraged a full debate with the protagonists each retreating to their corners.

Overall the rate of off pump surgery appears to be, at best, static, in the UK. A conclusion might be that what seems to be a formidable learning curve, in an environment of tight scrutiny, has put many people off taking on this new endeavour.

However, large retrospective trials continue to show some benefits of OPCABG over on pump. Is there some sort of bias in these trials or is there really something in off pump surgery?

Perhaps a third possibility (to 1 and 2 above) is that there are cases for which off pump is more suitable and those for which on pump is better. Surgeons clearly need more data to be able to take an informed decision and offer the appropriate treatment to patients on an individualised basis.

Providing the clear evidence base for this decision making process is the challenge that lies ahead.

SURGICAL MANAGEMENT OF ATRIAL FIBRILLATION

THE RATIONALE OF TREATMENT

SURGICAL MANAGEMENT OF ATRIAL FIBRILLATION

CONCOMITANT ATRIAL FIBRILLATION SURGERY: THE LESION SETS

Inserted PPt entitled: Lone AF Birmingham 2009

: Gaynor
: Three AF Papers

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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NOTES

Inserted PPt entitled: 2008-10-02

AORTIC STENOSIS - THE EVIDENCE BASE FOR SURGERY

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

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 REGURGITATION, AORTIC VALVE REPAIR AND AORTIC ROOT PRESERVATION TECHNIQUES

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

AHA and ESC guidelines recommend corrective surgery for severe aortic regurgitation (AR) in symptomatic patients (recommendation class/level of evidence, I/B), in asymptomatic pts with resting LVEF </=50% (I/B), in asymptomatic pts with LVEF >50% with LVED dimension >70mm or LVESD >50mm (IIa/C); in pts undergoing CABG, ascending aortic or other valve surgery (I/C). Corrective surgery for any degree of AR is indicated for pts with aortic diameter >/= 45mm for Marfan pts (I/C); >/= 50mm for bicuspid valve pts (IIa/C) and >/= 55mm for other pts (IIa/C).

Experience is growing with aortic valve repair for cusp prolapse, perforation, retraction/thickening and commissural disruption along with uni- and bicuspid aortic valves, rendering these techniques realistic alternatives to aortic valve replacement (AVR), especially in young pts and in pts undergoing aortic root repair.

Aortic root preservation techniques including remodeling, reimplantation and tailoring (sinotubular reconstruction) have evolved over time and the modified versions now demonstrate good mid-term durability. The Sinus of Valsalva graft for root reimplantation has proved to be easy to use and versatile with 90.8%±3.3% freedom from AVR at 5yrs. It appears to be particularly effective in preventing postoperative annular dilatation in pts with connective tissue disorders.

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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.

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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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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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ISCHAEMIC MITRAL VALVE REGURGITATION – SURGICAL TREATMENT AND ADJUVANT THERAPIES

Ischaemic heart disease can cause mitral regurgitation (MR) in several ways – acute myocardial infarction, which will not be considered here, can cause acute papillary muscle rupture or dysfunction and MR. Chronic ischaemic heart disease can lead to myocardial infarction that can in turn cause MR. The mechanisms of MR in this setting is dysfunction and distortion of left ventricular myocardium and papillary muscles, which may lead to tethering, usually of the posterior leaflet of the mitral valve which may be further compounded by failure of systolic contraction of the mitral annulus. Ischaemic cardiomyopathy may cause a spectrum of symptoms including angina, heart failure and a combination of the two. Analysing the data is made more complex as some trials have been performed for patients purely with angina, others purely for heart failure.

Mitral regurgitation in this setting may be clinically silent with little or no audible murmur and its significance is often underestimated by echo – significant regurgitation for degenerative MR is defined by a regurgitant volume of greater than 45 mls, but in ischaemic MR a regurgitant volume of greater than 30 mls is thought significant. MR is common in patients with impaired LV function, and isolated CABG alone does not improve moderate or severe MR in most cases[1]. The severity of CABG is underestimated by TOE at the time of surgery, unless provocative tests are used. Leaving patients with residual MR after coronary artery surgery adversely affects life expectance[2,3] – this is not just an effect of MR being a marker of more severe LV dysfunction – mild to moderate residual MR is an independent predictor of poor prognosis.

There is mixed data on whether mitral repair at the time of CABG improves prognosis in patients with moderate to MR and angina, [4,5,6,7,8]. On balance the data suggests survival is better, and that symptoms are better controlled if the mitral valve is repaired.

There is evidence that some additional treatments may help improve prognosis in patients with poor LV function. Implantable cardiac defibrillators improve outcome in this group [9], and this applies to patients who have also undergone CABG. Cardiac resynchronization therapy (biventricular pacing) improves MR in patients with poor LV function [11] and for patients with symptoms of heart failure combined treatment with ICDs and resynchronisation therapy improves outcome [12].

Patients with poor LV function and MR due to ischaemic heart disease are a difficult group to treat with bad longterm outlook. Surgery with revascularization and mitral repair improves outcome, but many of these patients have high degrees of predicted operative risk. Some of these patients will also benefit from ICD +/- resynchronisation therapy. Some may benefit from these therapies instead of surgery. Optimal treatment strategies are best defined by a team approach include surgeon and cardiologist with expertise in EP treatments.

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ISCHEMIC MITRAL VALVE REGURGITATION:

THE LIMITS OF OUR KNOWLEDGE

Mr. A. Anyanwu – Consultant Cardiothoracic Surgeon, New York, U.S.A.

The field of ischemic mitral regurgitation continues to evolve with recent studies improving our understanding of the pathophysiology and outcomes. Current approach to therapy should consist of definitive diagnosis by clinical and echocardiographic criteria, complete surgical revascularization, and restrictive annuloplasty using complete rigid or semi-rigid rings. Consistent application of surgical technique and collection of robust outcomes data is crucial to guiding our understanding of the natural history of surgically treated disease. Unfortunately limitations in surgical techniques and flaws in study methodology have restricted the ability of the literature to determine the efficacy of mitral valve annuloplasty and although many papers suggest annuloplasty is of limited efficacy, these papers have critical flaws. The efficacy of alternative surgical techniques such as chordal cutting, valve replacement and papillary muscle relocation is undetermined. This lecture will review recent developments and controversies regarding the pathophysiology and management of ischemic mitral regurgitation and also discuss the limitations of the published literature.

Further reading:

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

CARDIOPULMONARY BYPASS

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

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

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

Suggested Reading

Taylor K M. Cardiopulmonary Bypass in Seminars in Thoracic and Cardiovascular Surgery 1990.2:291-415
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