Transoesophageal echocardiography from the cardiac surgeon's perspective

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We first started using routine epiaortic and transoesophageal echocardiography (TEE) in late 1995. It revolutionised our cardiac surgical practice only partly, because it provided a full and comprehensive diagnostic study routinely.

The predominant reason that it revolutionised practice was that it provided a real-time haemodynamic monitor, where acute changes, minute to minute, could be detected and compensated for. More significantly, it radically improved our understanding of haemodynamics, resulting in far superior intraoperative and postoperative haemodynamic management than in the intensive care unit, where there was only Swan-Ganz catheter monitoring and no TEE monitoring. Paradoxically, this created conflict between the cardiac surgical team and the intensive care unit team, where there was a progressive discordance in the understanding and management of haemodynamic derangement based on the experience gained using echocardiography.

For example, in 1995, all patients would be routinely "filled" prior to the anaesthetic, during the anaesthetic, during the bypass pump run, after bypass and in the intensive care unit. Haemodilution was the order of the day. Our understanding was that patients who were fasting came to the operating theatre "dry", and at all other times were "empty". Now this view is shared by only some intensive care unit consultants, and very few of the cardiac surgical team. Indeed, for the past four years, we have commenced a routine low-dose vasoconstrictor (noradrenaline) as an infusion when the anaesthetic begins. This is maintained throughout the remainder of the operation and into the postoperative period in the intensive care unit. We are simply compensating for the vasodilation caused by the anaesthetic and maintaining vascular tone. Later, we are compensating for the additional inflammatory vasodilation. No fluid is generally given prior to cardiopulmonary bypass. Equally, little (if any) fluid is given during bypass (other than for the pump prime), and little (if any) is given following weaning from bypass. Any scavenged blood is processed in the cell saver and retransfused as packed red blood cells, raising the haemoglobin concentration following bypass.

The difficulty occurs in the intensive care unit, where the systemic inflammatory response syndrome peaks at about six to eight hours postoperative. Despite the presence of low-dose vasoconstrictors, the patient progressively vasodilates, which is misinterpreted as the patient being under-volumed, rather than vasodilated. Over time, we have observed a shift in practice towards the earlier use of higher doses of vasoconstrictors to compensate for the vasodilation, rather than the primary use of crystalloid or colloid solutions; but this remains an area of inconsistent practice. Our frequency of homologous blood transfusion in the operating room has markedly diminished over time.

There are many other areas of benefit which, no doubt, will be well described elsewhere in the symposium. Of greater interest would be to concentrate on two specific areas of valve repair and respective echocardiography assessment.

Mitral valve repair

The most important information that the surgeon needs is the mechanism for the regurgitation, and secondarily the underlying cause of this mechanism. However, with new approaches to mitral valve repair which are different to the

traditional techniques of Carpentier, different sorts of information is required.

One intraoperative diagnosis that is important to establish is whether there is an acute, reversible form of mitral valve regurgitation which, therefore, does not require any specific treatment. Usually this relates to acute reversible diastolic dysfunction of the left ventricle, whereby mitral valve regurgitation can be trivial or even mild, becomes severe and is then resolved within the space of 10 to 20 minutes. This may be seen with acute reversible coronary ischaemia, poor intraoperative myocardial protection during cardioplegic arrest, inadequate period of time allowed for reperfusion before weaning from cardiopulmonary bypass, or air embolism of the coronary arteries. Typically, no acute mitral valve or left ventricular shape or size change is seen, yet mitral valve regurgitation may be severe. Clearly, this is problematic, and attention to the context of the situation, more so than the echocardiography findings, is important.

Determining the mechanism for the regurgitation will vary somewhat, depending on whether the surgeon plans to use a classical Carpentier repair technique, or one of the newer approaches, to repair. Principally, Carpentier methodology seeks to restore a more normal geometry to the mitral valve annulus and to resect redundant leaflet tissue to restore more normal-sized leaflets. Alternatively, specialised distorted mitral valve annuloplasty rings have been devised for ischaemic mitral valve regurgitation to displace the mitral valve annulus towards the apex of the left ventricle, in the region of restricted leaflet motion caused by myocardial infarction. Another alternative approach includes an attempt to convert a bileaflet mitral valve into a monoleaflet valve by radical resizing of the mitral valve posterior annulus.

The mitral valve annulus dimensions should be measured in two separate planes, since the shape-of-three annulus is not circular. Generally, this can be better appreciated with three-dimensional echocardiography. The distance over which coaptation of the leaflets takes place is important. Even if the mitral valve is competent, minimal leaflet coaptation is likely to result in regurgitation subsequently. The relationship between the point of coaptation and the annulus plane is also important for estimating the length of artificial chordae.

The degree of redundancy has not generally been measured, but simply taken as a reflection of myxomatous disease. However, the height of the posterior leaflet in particular is useful to know

when performing a leaflet plication repair, whereby part of the leaflet is folded (rather than resected) to reduce the height of the leaflet. Additionally, the appreciation of restricted leaflet motion, consequent to transmural myocardial infarction, is important in ischaemic regurgitation.

A variety of miscellaneous conditions require separate assessments, including perforation due to endocarditis or systolic anterior motion of the mitral valve.

Aortic valve repair

Aortic valve repair has traditionally been associated with poorer success rates. A number of new approaches are being published, with significantly improved success. Nevertheless, it is still "early days" for aortic valve repair. Principally, this is due to the inexperience of surgeons repairing the aortic valve.

The most important information relates to the mechanism of the regurgitation. Specifically, the precise relationship between the leaflets needs to be established because, unless this is specifically addressed by the repair, it will not be successful.

Broadly, there are two main groups. One of these is aortic wall disease resulting in aortic dilation, and therefore splaying of the aortic valve commissures away from the vertical orientation (parallel to the left ventricular outflow tract). This then results in tensioning of the free edges of the aortic valve leaflets, which reduces the coaptation zone of the leaflets. Usually, the situation of the regurgitation is central. This is because, even though the coaptation of the leaflets is adequate for much of the length of the leaflets, the leaflets do not meet perfectly in the central region. It is generally thought that the aorta is always uniformly dilated, but frequently it is dilated in an eccentric manner, whereby the posterior aspect of the aorta (in relation to the left coronary artery and left coronary cusp) rarely dilates. Usually dilation occurs in both the right and the non-coronary cusp areas, with the non-coronary cusp area usually dilating the most. Perhaps this is because the non-coronary cusp sinus of Valsalva is unsupported by any adjacent tissue.

The treatment of aortic dilation mechanisms relates to correction of the aortic valve geometry, so that the commissural posts parallel with the left ventricular outflow tract and the sinotubular junction and the annulus of the aortic valve dimensions are within 1-2 mm of each other. Plication of the aortic wall, aortic vascular prosthetic grafting or resection of

the aortic wall including the sinus of Valsalva and replacement with vascular graft material are all surgical options.

Aortic dissection may result in central displacement of one or more of the commissural posts, and repair will involve a combination of reattachment of the commissural posts to the dissected aortic tissue and reconstruction of the sinotubular junction with the vascular graft.

The assessment of aortic valve leaflet prolapse is less well described and more problematic. This is because it is always assumed that the prolapsing leaflet is the cause of the regurgitation and that, once the aorta is opened, the surgeon finds it almost impossible to recreate or establish the mechanism of the leaflet dysfunction. Therefore, assessment prior to cardiopulmonary bypass using echocardiography is critical to establishing the mechanism. Equally, following repair, the surgeon finds it very difficult to assess the competency of the repair whilst the valve is still on view. In particular, where leaflet tissue is increased in size and prolapse is apparent, it is common that one leaflet is assessed to be prolapsed whilst it is assumed that the other two leaflets are normal or not prolapsing. Usually, however, all three leaflets are affected by this process, and the principal cause of regurgitation is that one leaflet is not prolapsing as much as the other two leaflets. Hence, there is a loss of coaptation, which results in regurgitation. Even more problematic is determining the mechanism for regurgitation with abnormal valves, such as a bicuspid aortic valve, which may or may not have areas of calcification. Repair of the bicuspid or other congenital abnormal valves is certainly possible.

The techniques for repairing the valve other than reconstructing the commissural post geometry of the valve include plication of the valve leaflets to reduce the length of the free edges of the valve, reinforcement of the free edge with Gor-Tex® sutures, and decalcification of the valve leaflet tissue.