Available online at www.sciencedirect.com
SCIENCE DIRECT
JNMU
Journal of Nanjing Mecical University, 2007, 21(1): 1-7
www.elsevier.com/locate/jnmu
Review

Role of intraoperative transesophageal echocardiography in coronary artery bypass grafting

Xinchun Chen[a,*]

[a]Department of Anesthesia, the First Affiliated Hospital of Nanjing Medical University, Nanjing 210029, China

Received 24 April 2006

Abstract

Transesophapeal echocardiography (TEE) can be used as a diagnostic tool during cardiac surgery to direct the surgical procedure and diagnose unanticipated problems. TEE has also been one of the most important means of monitoring myocardial ischemia during coronary artery bypas grafting procedures. The cardiac anesthesiologist can apply intraoperative TEE in evaluating coronary artery anatomy and aorta atherosclerosis, assessing diastolic left ventricular function and preload, measuring intracardiac pressure and cardiac output, detecting ischaemic mitral regurgitation, intracardiac air and pericardial effusion.

Keywords: transesophageal echocardiography; coronary artery bypass grafting; anesthesia; mycardial ischemia; cardiac output; regional wall motion abnormality

Transesophageal echocardiography (TEE)is one of the most practicable tools to provide the instant information of myocardial ischaemia, though its use for coronary artery bypass grafting (CABG) surgery is a Category Ⅱ indication based on the lack of direct evidence that TEE detection of intraoperative myocardial ischaemia leads to improvement of patients' outcome [1]. In the last decade, the use of TEE in the operating room had seen rapid expansion. It is a powerful and informative cardiac monitor and an important diagnostic tool during cardiac operations[2].

After induction of general anaesthesia and oral tracheal intubation, the probe is inserted into the esophagus. A thorough and careful examination should be completed at the beginning of the operation in order to set up a baseline and make sure the abnormity of the heart. If unexpected problem arise later in the case, this baseline record can be reviewed to determine whether subsequently noted findings are new, pre-existing or worse. If the patient is unstable, the main purpose of the TEE examination should be performed firstly to answer the urgent question for the case. The remainder of the comprehensive examination should be completed if the situation permits.

Assessment of ventricular function

Haemodynamic abnormalities of the heart are often accompanied by changes in the size of its chamber. With chronically elevated loading conditions, qualitative evaluation of left ventricule can be recognized as enlarged, normal (about 5.5 cm), or underfilled with experience. TEE can not only yield information about the structural integrity of the heart, but also offer continuous and accurate information about ventricular performance and function. Ventricular function, assessed by ejection fraction, is more important in determining survival than the number of diseased vessels[3]. Left ventricular function has proved to be an important predicator of outcome in patients undergoing CABG. An experienced observer can detect changes in cardiac output and left ventricular filling before significant changes in blood pressure appear[4].

*Corresponding author.
E-mail address: xinchun6665822@sohu.com

During operation, ventricular function is often assessed by ejection fraction (EF) or cardiac output (CO). Both can be estimated by TEE. Fractional shortening (FS) is measured in an M -mode mid-papillary view as the difference between end-diastolic diameter (EDD) and end-systolic diameter (ESD) normalized for end -diastolic diameter.

Fractional area change(FAC) is derived from two-dimensional images and calculated as end-diastolic area-end-systolic area normalized for end-diastolic area and allows estimation of ejection fraction.

Stroke volume(SV) can be calculated by multiplying the time-velocity-integral(VTI), the area under the curve of the Doppler flow profile (***Fig. 1***), with the aortic valve area(AVA). AVA can be calculated from a midesophageal 30^0 view of the aortic valve as $AVA=0.433 \times L^2$.

Where L is the average length of the free side of the aortic valve cusp during ejection(***Fig. 2***).

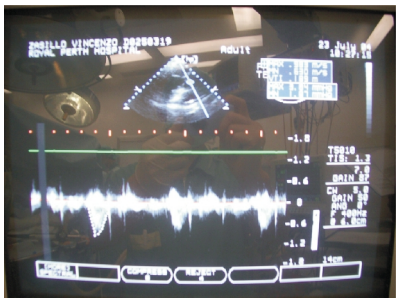

Fig. 1 The area under the curve

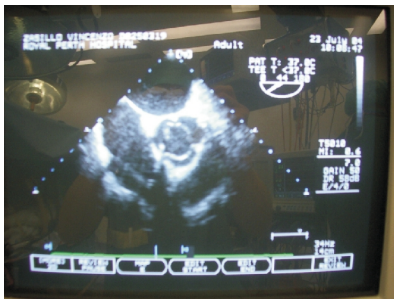

Fig. 2 The length of the aortic valve cusp

CO can be derived simply by multiplying stroke volume by heart rate, which has been shown that SV and CO can be derived with high accuracy, high reproducibility, and a low failure rate using this technique [5]. New imaging modalities with special software are promising approaches to automatic measurement of CO requiring fewer assumptions and necessitating only one image view[6].

In markedly reduced preload as due to hypovolaemia, it is possible to underestimate systolic function. Reduced left ventricular diastolic dimension will make the inward movement occurring in systole appear to be less.

Assessment of Diastolic function

As many patients with ischaemic cardiac disease (IHD) presenting to cardiac operating theatres will have pre-existing diastolic dysfunction, and cardiopulmonary bypass itself is implicated in acute reversible diastolic dysfunction, these are increasing interests to cardiac anaesthetists.

During coronary procedure, diastolic function of LV can be assessed by mitral valve velocities and left upper pulmonary venous inflow with pulse wave Doppler. Mitral inflow occurs in two phases, passive early diastolic (E-wave) and active late diastolic(A-wave). Diastolic dysfunction progresses the severe phase of restrictive filling that occurs with a very high E-wave velocity with steep deceleration and a diminutive A-wave. These result from the development of high LV end diastolic pressure (LVEDP) and LA pressure.

It was observed that some patients with normal mitral inflow patterns before CPB had signs of impaired ventricle filling after bypass[7]. Recently, Rinder *et al* [8]reported that diastolic filling was significantly impaired two hours after CPB. They found that it tended to recover in four hours and returned to baseline by eighteen hours.

The left upper pulmonary venous inflow is easily obtained by leftward rotation of the probe from ME four-chamber view or two-chamber view. Pulse Doppler interrogation of pulmonary venous inflow is characterized by three wave: AR-wave(flow reversal during atrium systole), S-wave (systole), D-wave (diastole). Normal pulmonary venous flow pattern is illustrated in ***Fig. 3***. High left atrial pressure produces diastolic predominance in the pulmonary venous flow pattern (***Fig. 4***).

Several changes occur with increased diastolic dysfunction:

1. Blunting of the S wave velocity (such as D> S) reflecting a non-compliant atrial system and high LAP and LVEDP.

2. Increasing atrial reversal (AR) velocity reflecting high LVEDP and LAP and increasing reliance on the “atrial kick”.

3. Increased AR wave duration as compared to

mitral A wave duration. As LVEDP increases, the LV pressure exceeds the LAP earlier, shortening the mitral A component.

In cases of hypovolemia, systolic dominance becomes exaggerated and diastolic flow becomes nearly undetectable[9].

Assessment of right ventricular function

Right ventricular function can be affected by myocardial ischaemia. This is usually associated with inferior LV dysfunction. During the operation, evaluation of right ventricular function has historically been based largely on visual inspection. The measurements of volume and area can only be alone by estimation due to the crescent shape of the right ventricle, which are usually compared with size of left ventricle.

The systolic excursion of tricuspid annulus has been noted as potentially more accurate index of right ventricle systolic function, as measured from the lateral portion of the annulus to the apex. Tricuspid annular motion has been shown to be reflective of nuclear-derived ejection fraction and circumvents issues of right ventricular geometry[10]. The datum in every patient at a risk of RV dysfunction should be noted. This is best observed by TEE in the ME four-chamber view at the level of the coronary sinus at about 20^0 rotation. With multiplane imaging, the views of four-chamber, two-chamber and long-axis show all segments of the right ventricle and allow the assessment of right heart function.

Regional wall motion abnormalities

Before considering ventricular function it is necessary to firstly consider whether there are any regional wall motion abnormalities (RWMAs), which occur almost exclusively in the setting of ischaemia heart diseases. RWMAs exist when different regions of the left ventricle exhibit different grades of systolic function. This is best considered initially in the TG mid SAX view, although obviously all segments of the ventricle must be examined before pronouncing the heart free of RWMAs. The base and apex of the left ventricle can be seen in the ME 2-chamber LAX view (base 2, 5 and apex 13,15)(***Fig. 5***) and the ME 4-chamber view(base 3, 6 and apex 14,16)(***Fig. 6***). The anteroseptal (1,7) and posterior(4, 10) wall of base and mid segment can be checked in the ME LAX view, TG basal SAX and TG mid SAX.

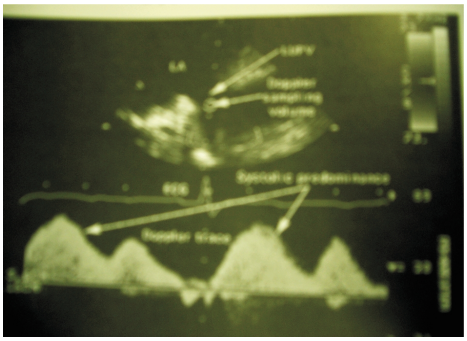

Fig. 3 Normal pulmonary venous flow pattern

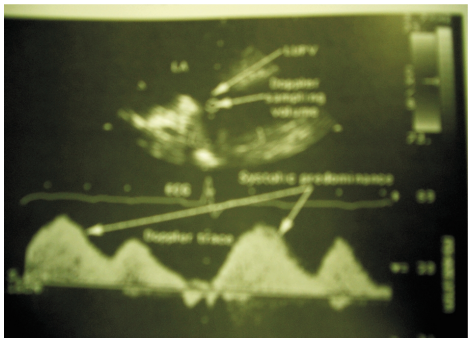

Fig. 4 The flow pattern of high left atrial pressure

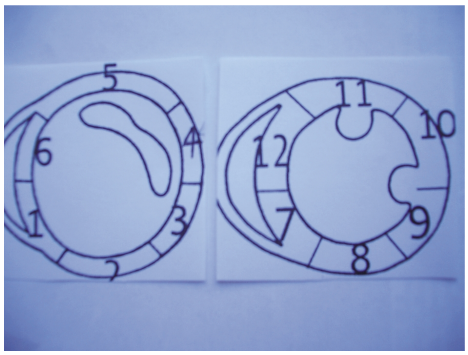

Fig. 5 TG basal SAX and TG mid SAX

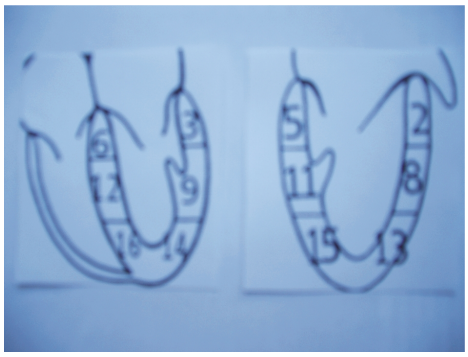

Fig. 6 ME 4-Chamber and ME 2-Chamber LAX

When the heart is displaced for vessel graft construction (LCX, OM1, OM2, PDA etc) during OPCABG, It is a little difficult to get good images. Usually, transient RWMAs during vessel occlusion disappear within 30 seconds. More attention should be paid to persistent new RWMAs after OPCAB, for

it may be an indication of a technically flawed graft. When the heart is left, ECG may show small complexes or conduction abnormalities[11]. Standard TEE views sometimes are not obtainable, and stabilizers may produce mechanical wall motion abnormalities [12]. It may partially overcome this limitation by withdrawing the probe into the esophagus and using the two-chamber and four-chamber views to image the ventricle.

There is good evidence that intraoperative TEE is a more sensitive monitor for myocardial eschaemia than ECG. Coronary occlusion incites RWMAs within about 20 seconds up to several minutes before ECG changes occur [13]. Ischemia may also induce wall motion abnormalities without any of 12-lead ECG changes [14]. About 20% of wall motion abnormalities are seen only in long axis view of LV. Only 17% of all RWMAs can be detected in the short axis mid-papillary view, emphasizing the importance of evaluating the ventricle in multiple planes with careful standardization of location and edge definition[15]. Training will increase the operator's ability to attain the required images and to recognize new RWMAs in real time[16]. When a wall motion abnormality is identified, the knowledge of the coronary circulation enable the anaesthetist to predict which vessel views may be involved.

To judge contractility, it is best to use the transgastric short-axis view at the tips of the papillary muscles. The systolic thickening of the ventricular walls is the best way to assess regional function. Wall motion is commonly graded as normal(marked wall thickening), mild hypokinetic (moderate wall thickening), severe hypokinetic (minimal wall thickening), akinetic (no wall thickening), or dyskenic (wall thinning in systole)[10]. A change in radius(an imaginary line from endocardium to center of LV cavity in TG mid papillary SAX view)refers to decrease in length, during systole. A worsening of segmental wall motion by at least two grades is required to diagnose ischaemia, less severe changes are not reliably interpretable [17]. Care must be taken in case of increased wall thickness arising from hypovolemia with true hypertrophy. Recently, color kinesis has been shown to be as sensitive but not as specific as conventional wall motion analysis[18].

Not all RWMAs are indicative of ischaemia. Nonischaemic myocardium adjacent to ischemic or infracted myocardium may be interpreted as RWMA, a phenomenon known as "tethering". RWMAs also occur with pacing, bundle branch blocks, and myocarditis[19]. Tachycardia may elicit new RWMAs. Wall motion abnormalities may also be apparent that disappear on increasing preload and these have been shown to be unrelated to myocardial ischaemia[20].

RWMAs are common during CABG surgery and have important prognostic and management implications. Persisting RWMAs have been associated with a regraft rate of around 1%-5%[21]. TEE can influence the decision as to which vessels should be regrafted. Another potential benefit of intraoperative TEE is that it may provide reassurance whether regrafting is necessary.

Evaluation of coronary artery anatomy and aorta atherosclerosis

With the development of digital imaging capabilities, the emergence of multiplane TEE, TEE has improved visualization of the coronary artery tree and enhanced detection of its abnormalities [22]. The ostium of the left main(LM) coronary artery is visualized in ME short-axis view above the aortic cusp and rotated slightly to the left. The LM arising from the left sinus at 3 o'clock is about 2 cm in length. Small adjustments in the transducer orientation are often necessary to view the vessel along its full length from the aortic root to its bifurcation, which is Y-shaped with the left anterior descending (LAD) artery and the left circumflex(LCx) in the atrioventricular groove.

Normally, the ostium of the right coronary artery arises(RCA) at 7 o'clock position on the ME short-axis view of the aorta and is visualized as the probe is withdrawn 1 to 2 cm above the level of the aortic valve and is flexed anteriorly. The right coronary ostium and a 1 to 2 cm segment of the proximal RCA also can be visualized in longitudinal plane typically at 110° to 130° of rotation.

Intraoperative TEE is a sensitive method for detecting atherosclerotic disease of ascending aorta with the ME ascending aorta SAX and ME ascending aorta LAX view. It can tell the site of severe atherosclerosis so that surgical modifications can be made if possible to reduce postoperative stroke.

Stroke is a serious complication after CABG. Roach et al[23] evaluated 2108 cases after elective CABG surgery and found an incidence of adverse cerebral outcome of 6.1%. Konstadt et al[24] suggested that the presence of atheroma in descending aorta was an indicator of atheroma elsewhere. They used TEE to screen descending aorta and found that if there was moderate or severe atheroma, then there was a 34% risk of similar disease in the ascending aorta. Conversely, if there was no atheroma detected, then

more than moderate(> 3 mm) atheroma in ascending aorta was highly unlikely. The tactics to avoid postoperative stroke include not clamping aorta and using hypothermia to induce cardiac arrest, or changing the aortic cannula to the femoral or axillary artery. Other option is to perform CABG without CPB on the beating heart. More aggressive methods include replacing the diseased segment of aorta under deep hypothermic circulatory arrest. Wareing et al[25] performed this procedure on 27 patients with severe ascending aortic atheroma without postoperative stroke. A new aortic connector can provide a precise and reproducible alternative for proximal anastomosis between vein graft and the aorta. This does not require sutures, cross clamp or side bite of the aorta. It is very easy to use and achieves instant haemostasis at the site in a few seconds.

Hypotension

Access to the different coronary artery during OPCABG requires displacement of the heart. The haemodynamic changes caused by heart displacement may vary depending on the vessel to be grafted (eg, LAD, LCx, or RCA), preoperative ventricular function, preload, and heart size. Displacement of the heart vertically for anastomosis to the marginal branches causes the greastest haemodynamic compromise[26]. The increase in CVP and decreased mean PAP and PCWP and arterial hypotension can be noted. These changes can be due to right ventricular compression and result in an increase in CVP. Grundeman *et al*[27] found that the deteriorated circulation during displacement was 44% reduction by an elevated right ventricular preload without a concomitant increase in left ventricular preload. TEE can observe the right ventricular outflow tract obstruction in ME RV inflow-outflow view. And in a few patients with decreased CVP, TEE showed a collapse of SVC and IVC. It is possible that the haemodynamic deterioration can be due to kinking of the SVC and IVC, which decrease the venous return to the right heart. Geskes et al [28] also suggested the impairment of right ventricle function could be the result of inflow obstruction of the blood by tract on the IVC.

Hypotension due to reduction in systolic vascular resistance (SVR)manifests as a normal EDA in the setting of hyperdynamic systolic function. Other cardiac causes of low CO such as VSD, severe MR or AR will also show a normal EDA and hyperdynamic LV, but these may be readily distinguished from each other on TEE views.

An experienced anesthetist can consistently and rapidly distinguish severe ventricular dysfunction from other causes of hypotension by using TEE. The ability to distinguish causes of shock using TEE will rely on the ability to assess the LV end diastolic area (EDA) as an assessment of LV preload[29].

Hypovolemia

Hypovolaemia manifests as marked disease in LV EDA and normal or increased LV systolic function. As 90% of the stroke volume is due to contraction of the LV in its short axis, the best view utilized for this purpose is the TG mid SAX view. Studies have shown that decrease in blood volume as little as 1.5% could be detected by TEE [30] and that correlation between EDA and cardiac output was better than that between pulmonary artery occlusion pressure and cardiac output [31]. In fact, cardiac anaesthetist often assesses the patient's volume status with experience in TG mid SAX view on whether end systolic cavity obliteration may be seen (kissing papillary muscles) or not.

Ischaemic mitral regurgitation

RMWA is one of several pathogenic mechanisms by which ischaemic heart diseases may lead to regurgitation.. Ischaemi a can cause dysfunction of the papillary muscles, which may be reversible. The improvement may occur with vasoconstriction due to improved perfusion of the ischaemic segments.

Fibrosis of papillary muscles cause foreshortening of the mitral apparatus and regurgitation due to failure of coaptation without flail. Rupture of the papillary muscle can cause flail with a mass (the remnant of the papillary muscle) seen in the left atrium during ventricular systole. The major determinants of functional MR after MI are changes in LV shape(LV dilation and mitral annular dilatation) and regional LV function(mitral leaflet restriction and inferoposterior SWMAs)[32]. Intraoperative TEE has an important surgical impact in the management of patients with ischaemic MR. Cancellation of mitral valve surgery occurs in 9%-18% of patients scheduled for combined CABG and mitral procedures [33,34]. Functional MR discovered intraoperatively may lead to unscheduled mitral s urgery. Unscheduled valve surgery performed due to intraoperative TEE findings may be in 2%-3% of high-risk CABG cases and changed to conventional CABG with CPB[35].

Pericardial effusion and tamponade

If patient with CID suffers from myocardial infarction or uraemia, pericardial fluid may be detected by TEE in transverse sinus. A pericardial reflec-

tion (an echolucent space)exists between anterior LA wall and posterior wall of the ascending aorta. You can also use M mode to find the flat posterior pericardial echo reflection and the moving epicardial echo with separation between the two in both systole and diastole. The size of effusion is arbitrarily considered small on echocardiography when the separation between parietal and visceral pericardium is < 0.5 cm, moderate when the distance is 0.5 -2 cm and large when it is > 2 cm.

The incidence of postoperative cardiac tamponade is significantly low, however, with reported rates of 0.5 to 5.8%[36]. Reichert *et al* [37] estimated that cardiac tamponade was responsible for 10% of all cases of persistent hypotension after cardiac surgery. TEE is highly sensitive and specific for detecting pericardial effusion and diagnosing cardiac tamponade. After chest closure, when unexpected hypotension occurs, TEE can possibly find tamponade compressing the left atrium due to supine position.

Intracardia air

Air within the chamber is easily seen as hyperdense with TEE in ME 4-chamber view. In supine patient, air in left atrium accumulates along the IAS and adjacent to the right upper pulmonary vein. Air in the left ventricle accumulates along the apical septum. Tiny white spots seen floating within the chambers are microscope bubbles and are not of great concern except in cases of PFO and atrial or ventricular septal defect. Large bubbles have air-fluid levels visible on TEE as straight lines perpendicular to the direction of gravity that wobbles as the heart beats. They typically have shimmering artifacts extending from the air-fluid level away from the transducer, which should be evacuated before discontinuing CPB. If akinesis occurs in left ventricular inferior wall and right ventricular wall after CPB, air embolism of right coronary artery should be suspected.

Intracardiac pressure

Measuring flow velocities with Doppler echocardiography and applying the modified Bernoulli equation allow calculation of gradients between chambers of the heart at various locations. If the absolute pressure of one of these chambers is known, the pressure of the other chamber can be calculated. Thus, by measuring the peak velocity of TR with CWD, one can estimate the peak RV systolic pressure as the RV to RA gradient ($\triangle P=4V_{tr}^2$) plus the RA pressure. This is equal to the PA systolic pressure if there is no pulmonic stenosis. Similar logic can be used to measure LA, PA, and AR jets, and the LV systolic (same as systolic BP), RV diastolic(same as CVP), and aortic diastolic(same as diastolic BP) pressures are known.

Other use for CABG

Intra aortic balloon counterpulsation has been used to support the left ventricle without aortic regurgitation(AR) before induction or during the operation in conventional CABG and some high-risk OPCAB patients. These patients usually suffer from left main coronary artery disease, unstable angina or poor ventricular function[38]. It has been demonstrated that the device can facilitate OPCAB surgery by enhancing haemodynamic stability, reducing complications and promoting quick recovery [39]. The tip of balloon -pump can be checked or assisted just below the ostium of left subclavian artery with TEE in a UE aortic arch SAX view.

During conventional CABG with CPB, intraoperative TEE also can guide retrograde cannula for cardiaplegia by screening coronary sinus.

In conclusion, during OPCAB, TEE monitoring of LV function is necessary to determine the degree and persistence of myocardial ischaemia produced by the clamping of coronary vessels. Particularly, TEE can provide useful information to the anaesthetist and surgeon in cases with high risks caused by poor left ventricular function or haemodynamically significant mitral regurgitation (MR). In critically ill patients, assessments of global function and volume status aid the anaesthetist in determining the need for inotropic or vasodilator drugs, fluid resuscitation, or more aggressive measures, such as intra aortic balloon pump (IABP), ventricular assist devices (VAD), and extracorporeal membrane oxygenation.

References

[1] Practice guideline for perioperative TEE,A report of the American Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography, *Anesthesiol* 1996; 84:986-1006.

[2] Shanewise JS, Cheung AT, Aronson S,Steward WJ,Weiss RL,Mark J, *et al*. ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: Recommendations of the American Society of Echocardiography Council for intraoperative echocardiography and the Society of Cardiovascular Anaesthesiologists Task Force for certification in perioperative transesopeageal echocardiography. *Anesth Analg* 1999; 89:884-900.

[3] Mock MB, Ringqvist I, Fisher LD,Davis kB,Chaitman BR, Kouchoukos NT, *et al*. Survival of medically treated patients in the coronary artery surgery study (CASS) registry. *Circulation* 1982; 66:562-8.

[4] Reich DL, Konstadt SN,Ngiat M, Abrams HP,Bucek J. Intraoperative transesophageal echocardiography for the detection of

cardiac preload changes induced by transfusion and phlebotomy in pediatric patients. *Anesthesiology* 1993; 79: 10-5.

[5] Poelaert J, Schmidt C, Van Aken,Hinder F,Mollhoff T,Loick HM. A comparison of transoesophageal echocardiographic Doppler across the aortic valve and the thermodilution technique for estimating cardiac output. *Anasthesia* 1999; 54:128-36.

[6] Sun JP, Pu M, Fouad FM,Christian R,Steward WJ,Thomas JD. Automated cardiac output measurement by spatiotemporal intergration of color Doppler data. *Circulation*, 1997; 95:932-39.

[7] Thys DM, Hillel Z, Konstadt S. The intraoperative evaluation of left ventricular filling by esophageal Doppler echocardiography, AbstrActs of the English Annul Meeting of the Society of Cardiovascular Anaesthesiologists, *Montreal*,1986; 80.

[8] Rinders CS, Wheeler CR, Alpern WD. Pulse Doppler assessment of diastolic function before and after cardiopulmonary bypass. *Anesthesiology* 1988; 69:2.

[9] Rossvoll O,Hatle LK. Pulmonary venous flow velocity recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures J Am Coll *Cardiol* 1993; 21:1687-96.

[10] Clements F. Monitoring ventricular function in OR: Impact on clinical outcome. In Otto CM (ed): The practice of clinical echocardiography. Philadelphia, WB *Saunders*, 1997; 167-78.

[11] Greenspun HG, Adourian UA, Fonger JD,Fan JS. Minimally invasive direct coronary artery bypass: Surgical techniques and anesthetic considerations: decrease in monitoring and anesthetic considerations. *J cardiothorac Vasc Anesth* 1996; 10: 507-9.

[12] Mehta Y, Juneja R, Dhole S. Transesophageal echocardiography in MIDCAB: pitfalls. *J Cardiothorac Vasc Anesth* 1999; 13: 115-6.

[13] Watanabe S, Buffington CW. Speed and sensitivity of mechanical versus electrographic indicators to mild or moderate myocardial ischaemia in the pig, *Anesthesiology* 1994; 80:582-94.

[14] Wohlgelernter D, Jaffe C, Cabin H,Yeatman LA,clemen M. Silent ischemia during coronary occlusion produced by balloon inflation: relation to regional myocardial dysfunction. *J Am Coll Cardiol* 1987; 10: 491-8.

[15] Shah PM,Kyo S, Matsumura M,Omoto R. Utility of biplane transesophgeal echocardiography in left ventricular wall motion analysis. *J Cardiothorac Vasc Anesth* 1991; 5:316-9.

[16] Thomson I. Con: intraoperative myocardia is not benign. *J Cardiothorac Vasc Anesth* 1994;8:593-5.

[17] Rouine -Rapp K, Cahalan M, Ionescu P. Comparison of segmental wall motion analysis by anaesthetists and an echocardiographer, *Anesth Analg* 1983; 76:361-6.

[18] Hartman T, Kolev A, Blaicher C. Validity of acoustic quantification colour kinesis for detection of left ventricular regional wall motion abnormalities: a transesophageal echocardiography studiy, *Bri J Anaesth* 1997; 79:482-7.

[19] Weyman A. Principles and Practice of Echocardiograph, 2nd ed, Philadephia, Lea & Febiger, 1994.

[20] Seeberger MD, Cahalan MK,Rouine-Raoo K,Foster E,Ionescu P, Merrick S. Acute hypovolaemia may cause segmental wall motion abnormalities in the absence of myocardial ischaemia. *Anesth Analg* 1997; 85:1252-7.

[21] Mishra M, Chauhan R, Sharma K,Dhar A, Bhise M, Dhole S. Real -time intraoperative echocardiography - how useful? Experience of 5016 cases. *J Cardiothorac Vasc Anesth* 1998; 12(6): 625-32.

[22] Memmola C, Iliceto S,Rizzon P. Detection of proximal stenosis of left coronary artery by digital transesophageal echocardiography: feasibility, sensitivity and specificity. *J Am Soc Echocardiogr* 1993; 6:149-57.

[23] Roach G, Kanchuger M,Mangano C,Newman M,Nussmeier N, Wolman P, *et al.* Adverse cerebral outcome after coronary bypass surgery. *N Engl J Med* 1996; 335:1857-63.

[24] Konstadt SN, Reich DL, Kahn R,Viggiani RF. Transesophageal echocardiography can be used to screen for ascending aortic atherosclerosis. *Anesth Analg* 1995; 81: 225-8.

[25] Wareing TH, Davila -Roman VG, Daily BB,Murphy SF, Schechtman kB,Barzilai B *et al.* Strategy for the reduction of stroke incidence in cardiac surgical patients, *Ann Thorac Surg* 1993; 55: 1400-7.

[26] Nierich AP, Diephuis J, Jansen EW,Borst C,Knape JT. Heart displacement during off-pump CABG: How well is it tolerated? *Am Thorac Surg* 2000; 70:466-72.

[27] Grundeman PF, Borst C, Van Herwaarden JA,Mansvelt Beck HJ,Jansen EW. Hemodynamic changes during displacement of the beating heart by the Utrecht Octopus method. *Ann Thorac Surg* 1997: 63: 588-92.

[28] Geskes GG, Dekker AL, Vander Veen FH,Cramers AA, Maessen JG,Shoshani D. The enabler right ventricular circulatory support system for beating heart coronary artery bypass graft surgery. *Ann Thorac Surg* 1999: 68:1558-61.

[29] Cahalan M. Ventricular systolic function: regional and global LV and RV function. In course notes: Comprehensive review of intraoperative TEE, San Diego, CA, 2000.

[30] Cheung AT,Savino JS, Weiss SJ,Aukburg SJ,Berlin JA. Echocardiographic and haemodynamic indexes of left ventricular preload in patients with normal and abnormal ventricular function. *Anesthesiology* 1994; 81:376-87.

[31] Thys DM, Hillel Z, Goldman ME,Mindich BP,Kaplan JA. A comparison of hemodynamic indices derived by invasive monitoring and two -dimensional echocardio graphy. *Anesthesiology* 1987; 67:630-4.

[32] Van Dantzig J, Delemarre B, koster R, *et al.* Pathogenesis of mitral regurgitation in acute myocardial infarction: Importance of changes in left ventricular shape and regional function. *Am Heart J* 1996; 131: 865-71.

[33] Forrest A, Lovelock ND, Hu JM,Fletcher SN. The impact of intraoperative echocardiography on an unselected cardiac surgical population: A review of 2343 cases. *Anaesth Intensive Care* 2002: 30: 734-41.

[34] O'Rourke D, Palac R,Malenka DJ, Marrin C Arbuckle BE, Plehn JF. Outcome of mild periprosthetic regurgitation detected by intraoperative transesophageal echocardiography. *J Am Coll Cardiol* 2001; 38: 163-6.

[35] Aklog L, Filsoufi F, Flores K,Chen RH,Cohn LH,Nathan NS,Byme JG,Adams DH. Does coronary artery bypass grafting alone correct moderate ischaemic mitral regurgitation? *Circulation* 2001; 104: I68-75.

[36] Ball JB, Morrison WL. Experience with cardiac tamponade following open heart surgery. *Heart Vessels* 1996; 11:39-43.

[37] Reichert CL,Visser CA, Koolen JJ,vd Brink RB, van Wezel HB,Meyne NG,Dunning AJ. Transesophageal echocardiography in hypotensive patients after cardiac operation: Comparison with hemodynamic parameters, *J Thorac Cardiovasc Surg* 1992;104:321-6.

[38] Kim KB, Lim C, Ahn H,Yang JK. Intraaortic balloon pump therapy facilitates posterio vessel off-pump coronary artery bypass grafting in high-risk patient. *Ann Thorac Surg* 2001; 71: 1964-8.

[39] Guyton RA, Thourani VH, Puskas JD,Shanewise JS,steele MA,Palmer-steele Cl,Vintn-Johansen J. Perfusion-assisted direct coronary artery bypass: selective graft perfusion in off -pump cases. *Ann Thorac Surg* 2000; 69(1): 171-5.