Clinical Reports

Transoesophageal echocardiogram may fail to diagnose perioperative myocardial infarction

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We report a case in which a 55-yr-old man undergoing aortocoronary bypass was monitored with electrocardiogram and transoesophageal echocardiogram. Intraoperative electrocardiogram and simultaneous ECG recordings using the Holter monitor showed an ST elevation of greater than 2 mm and new Q wave formation in leads AVF and V₅ during skin closure. However, the transoesophageal echocardiogram showed no wall motion abnormalities. No significant haemodynamic abnormalities were observed during the period of intraoperative ECG changes. He was treated with nitroglycerine infusion. Confirmation of a perioperative myocardial infarct was documented by postoperative 12-lead ECG and CPK-MB. A postoperative transthoracic echocardiogram showed a hypokinetic left ventricle with an anteroapical infarct. Thus transoesophageal echocardiography failed to detect an apical wall motion abnormality when the probe was placed at the midpapillary level. This limitation can be overcome by periodically obtaining apical views or by using probes with more than one imaging plane.

On rapporte le cas d'un homme de 55 ans devant subir un pontage aortocoronarien et qui fut surveillé par l'électrocar-

diogramme et l'échocardiogramme transoesophagiens. L'électrocardiogramme peropératoire et l'enregistrement de l'ECG utilisant un Holter ont démontré une élévation du segment ST supérieur à 2 mm et la formation d'une onde Q récente dans les dérivation AVF et V₅ durant la fermeture sternale. Cependant, l'échocardiogramme transoesophagien n'a pas démontré d'anomalies de mouvement de la paroi. Aucune anomalie hémodynamique significative ne fut observée durant cette période peropératoire. Le patient fut traité avec une perfusion de nitroglycérine. La confirmation d'un infarctus myocardique périopératoire a été documenté par un électrocardiogramme 12 dérivation et du CPK-MB. Un échocardiogramme transthoracique en période postopératoire a démontré un ventricule gauche hypokinétique avec un infarctus antéroapical. Ainsi l'échocardiogramme transoesophagien n'a pas détecté d'anomalie d'un mouvement de la paroi apicale quand la sonde fut placée entre les muscles papillaires. Cette limitation peut être évitée par l'obtention périodique d'une vue apicale ou par l'utilisation de sondes fournissant plus qu'un plan de section.

Key words

MONITORING: transoesophageal echocardiography;
HEART: myocardial infarction.

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The use of echocardiograms has greatly facilitated the investigation and management of common cardiac abnormalities. The more common transthoracic echocardiograms are limited in their application when intraoperative thoracic procedures are contemplated. Thus, the advent of the transoesophageal echocardiogram has provided a new acoustic window to the heart and mediastinum.

Transoesophageal echocardiography provides a stationary and reproducible position of the probe. It does not interfere with the surgical procedure and continuous intraoperative monitoring of myocardial function for extended periods is possible. Kremer *et al.*¹ applied transoesophageal echocardiography in 43 high-risk patients. Seventy per cent of the patients with new persistent wall motion abnormalities had evidence of a myocardial infarction. No myocardial infarction occurred in patients

with transient wall motion abnormalities. The sensitivity of transoesophageal echocardiography in detecting myocardial infarction was 100 per cent in the studies of Kremer *et al.* and Smith *et al.*² When patients with severely impaired baseline systolic function were studied, Shively *et al.* found the sensitivity to be 46 per cent. Thus the sensitivity of transoesophageal echocardiography in infarction detection in a segment with a severe wall motion abnormality is hampered.

Although the short-axis view at the level of the papillary muscles includes segments of myocardium representing all three coronary arteries, wall motion abnormalities beyond this cross-section can be missed. Furthermore, myocardial infarction can occur after surgery when the transoesophageal probe is removed. In this case report, a patient had an intraoperative electrocardiogram which indicated ST segment elevation, but the transoesophageal echocardiogram failed to show any segmental wall motion abnormality.

Case report

The patient, a 55-yr-old male, was scheduled for elective coronary artery bypass grafting. His past medical history consisted of a myocardial infarction eight years before admission which was followed by angina pectoris on exertion.

Medical therapy consisted of diltiazem 60 mg bid and nitroglycerine 2.6 mg tid. Cardiac catheterization and angiography performed six months before admission showed a normal ventricle with no regional wall motion defects. The inferior wall was normal. The right coronary artery was dominant with a total occlusion of his right coronary artery, a 95 per cent obstruction in the circumflex vessel and a 40 per cent obstruction in the left anterior descending vessel.

On admission, his heart rate was 80 beats per minute and arterial pressure was 140/90 mmHg. His chest was clear and the heart sounds were normal with no added sounds. Neurologically no defects were encountered. Routine laboratory investigations and a chest x-ray were within normal limits. A preoperative 12-lead ECG showed left axis deviation. The CPK-MB was negative.

He received premedication with morphine 15 mg, perphenazine 5 mg, and diazepam 10 mg. He also consented to participate in a study and was therefore given metoprolol 100 mg the night before surgery and 100 mg 1½ hours before surgery. As part of the study, he was connected to an ambulatory ECG device (Holter performer, Model 8500, Applied Cardiac Systems Inc.) 45 min before surgery for monitoring leads AVF and V₅ which was continued intraoperatively and postoperatively for 24 hr. Intraoperatively, he was monitored using a radial artery catheter, pulmonary artery (PA) catheter, oesopha-

geal temperature probe, EGG, oximeter, and capnograph. The insertion of the arterial line and the PA catheter were performed under local anaesthesia and were uneventful. Anaesthesia was induced using a high-dose fentanyl technique (4000 µg), diazepam 5 mg, and the trachea was intubated after administering of pancuronium 10 mg. Following an easy intubation, the transoesophageal probe was passed under direct vision into the oesophagus and positioned so that the transducer was just posterior to the heart to obtain a short-axis, cross-sectional view of the left ventricle at the level of the papillary muscles. Anaesthesia was maintained with fentanyl 3000 µg, diazepam 25 mg, and pancuronium 5 mg. The patient also required propranolol 2 mg, nitroglycerine 7 mg, and nitroprusside 6 mg to maintain haemodynamic variables within 20 per cent of baseline.

The cross-clamp time was 74 min, and the duration of bypass was 106 min. Quadruple bypass grafting was done with the left internal mammary artery being anastomosed to the left anterior descending coronary artery, and grafts of autologous long saphenous veins to the first and second obtuse marginal branches of the circumflex and to the right coronary arteries. Cardiopulmonary bypass was discontinued with temporary pacing for ten minutes. No other inotropic support was needed.

Intraoperatively, the heart was monitored by an experienced echocardiographer using the transoesophageal echocardiogram which showed no wall motion abnormalities during the procedure. The echocardiographic images at predetermined intervals were recorded for 120 sec on half-inch VHS video tape. This was read by a cardiologist who confirmed no wall motion abnormalities. The left ventricle was classified as normal at baseline, and after skin incision, sternotomy, aortic cannulation, bypass, pericardial closure, sternal wiring, and at the end of surgery. However, intraoperative electrocardiograms and simultaneous ECG recordings using the Holter monitor showed an ST elevation of greater than 2 mm and new Q wave formation in leads AVF and V₅ during skin closure. No significant haemodynamic abnormalities were observed during the period of intraoperative electrocardiographic changes. Nitroglycerin infusion was given at skin closure and in the postoperative period in an attempt to revert the electrocardiographic changes.

Postoperatively, he was haemodynamically stable, but confirmation of a perioperative myocardial infarct was provided by the ST elevation in leads V₂–V₆, and new Q waves in leads II–III, AVF, V₂–V₄. The CPK-MB was strongly positive. In addition, a two-dimensional trans-thoracic echocardiogram done on the third postoperative day showed a hypokinetic left ventricle with an antero-apical infarct. He also developed a right occipital infarct resulting in some loss of vision. The clinical diagnosis

was confirmed by CT scan of the head. The patient was discharged from hospital one week after surgery.

Discussion

During transoesophageal echocardiography, the normal ventricle is seen to thicken during systole and the endocardial surface moves inwards. Abnormal wall motion is described by the terms hypokinesia, akinesia and dyskinesia. Hypokinesia refers to contraction that is less vigorous than normal, akinesia refers to the complete absence of wall motion during systole and dyskinesia describes paradoxical motion, the outward movement of a segment of myocardium during systole when it should be moving inwards.

Heger and Weyman correlated regional wall asynergy with the site of myocardial infarction in 37 patients.⁴ The most frequently involved segments were those in the midpapillary muscle at the short-axis view, followed by the apical segment. Thus the short-axis view at the midpapillary muscle appears to be the best single view with which to monitor regional wall motion abnormalities.

Smith *et al.* compared the incidence and importance of ischaemia as detected by two-dimensional transoesophageal echocardiography and electrocardiography during anaesthesia and surgery in patients known to have coronary artery disease.² During surgery, 24 of the 50 patients studied had new segmental wall motion abnormality, whereas only six had ST-segment changes. The standard surface ECG may not reveal subendocardial ischaemia.⁵ Even severe transmural ischaemia may go undetected if a single electrocardiogram lead is monitored.⁶ In addition, most studies also show that the sensitivity of regional wall motion abnormalities for the detection of myocardial ischaemia is generally superior to that of an electrocardiogram.^{7,8}

In this case report, the intraoperative transoesophageal echocardiogram failed to detect any regional wall motion abnormality, whereas electrocardiographic recording showed ST elevations greater than 2 mm in leads AVF and V₅. The transducer was placed at the midpapillary level to obtain a short-axis, cross-sectional view of the left ventricle. Thus, the transoesophageal echocardiogram failed to detect wall motion abnormality occurring in the apical region. Postoperatively, the patient had new Q-wave formation and ST elevation in leads V₂-V₆. There was also evidence of an anteroapical infarct on a postoperative transthoracic echocardiogram.

Usually the transoesophageal probe was placed at the midpapillary section of the heart to monitor left ventricular function. By not moving the probe, the process of monitoring is simplified, the chances of introducing false positives secondary to changes in orientation of the

transducer are minimized, and the remote possibility of oesophageal trauma is reduced.²

The onset of visible wall motion abnormality occurs within 10–15 sec of coronary occlusion.⁹ The physiological changes responsible for continuing abnormality will be accompanied by histological changes after 15–20 min of ischaemia.¹⁰ Thus, there may be a need for the transoesophageal probe to be moved at least every 10–15 min so that wall motion abnormality at either the midpapillary level or the apical region can be detected and pharmacological intervention attempted. Alternatively, we have to be aware of the possible failure to detect apical wall motion abnormalities if the probe is kept at midpapillary level.

Transoesophageal echocardiography has several technical pitfalls. In approximately 8–10 per cent of patients, the short-axis view of the left ventricle cannot be obtained because of horizontal displacement of the heart by the contents of the abdomen. The accurate detection of segmental wall motion abnormalities is also dependent on correct gain settings and the ability to maintain a stable probe position, to monitor the same left ventricular segments.¹¹ The development of biplane and multiplane probes may help to increase the number of LV segments that can be visualized without significant repositioning of the probe.

Anaesthetists are interested in using echocardiography during surgery to guide pharmacological therapy because it is a sensitive and specific method of detecting myocardial ischaemia. This case report demonstrates the failure of transoesophageal echocardiography to detect an apical wall motion abnormality when the probe was placed at the midpapillary level. This limitation can be overcome by periodically obtaining apical views or by using probes with more than one imaging plane.

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