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CLINICAL INVESTIGATIONS

Use of myocardial tissue Doppler imaging for intraoperative monitoring of left ventricular function

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Background. Detection of myocardial ischaemia during surgery is usually by assessment of regional wall motion using two-dimensional transoesophageal echocardiography (TOE). Tissue Doppler imaging (TDI) may assist this assessment and improve its accuracy.

Methods. We measured peak myocardial velocities in the anterior mid-wall of the left ventricle by TOE and pulsed-wave TDI in addition to transmitral flow velocity, two-dimensional echocardiography and cardiovascular variables. We studied 42 patients before and after coronary bypass surgery with left internal mammary artery grafts.

Results. Peak systolic and early and late diastolic velocity measurements of the anterior mid-wall were obtained in all patients. Variation between and within observers was small (<6%). Peak systolic thickening velocity correlated with visual assessment of anterior wall motion score, fractional area change of the left ventricle and left ventricular systolic wall stress. Because of the wide overlap of systolic velocity between the segments with normal and abnormal wall motion, it was not possible to separate normal from abnormal segments on the basis of TDI-derived velocity alone. The diastolic velocity in the anterior wall reflected the transmitral filling pattern. After surgery, the peak systolic and late diastolic anterior wall velocities increased (from 4.2 (95% confidence interval 4.0, 4.7) to 5.7 (4.8, 6.3) cm s^{-1} and from 3.5 (3.2, 3.9) to 6.0 (5.1, 6.9) cm s^{-1} respectively), while the ratio of early to late diastolic velocity decreased from 1.5 (1.2, 1.7) to 1.0 (0.8, 1.2). TDI changes characteristic of new myocardial ischaemia were not seen in any patient.

Conclusion. Intraoperative measurement of TDI in the anterior wall of the left ventricle is feasible and provides additional quantitative information on both regional and global systolic and diastolic function. We found changes in myocardial velocities indicating improvement in the systolic and impairment in the diastolic function of the anterior wall of the left ventricle immediately after mammary artery grafting.

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Transoesophageal echocardiography (TOE) is used during surgery to monitor left ventricular (LV) function and to detect intraoperative myocardial ischaemia. TOE diagnosis of myocardial ischaemia is based on visual assessment of regional LV wall motion. Automatic computer-aided systems to analyse LV wall motion have been developed,[1 2] but the operator's experience and visual assessment remain indispensable. However, despite adequate training and experience there can be significant observer variation in the visual assessment of LV wall motion.[3 4] Decisions based on the assessment of regional wall motion are often critical. For example, during cardiac surgery a new abnormality of

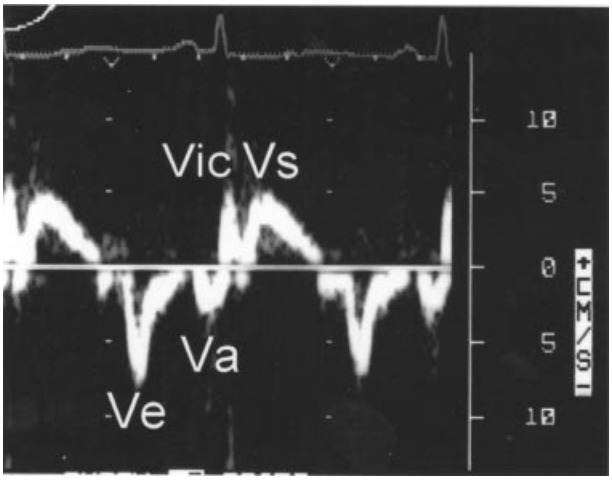

Fig 1 Pulsed-wave tissue Doppler recording of myocardial velocities in the LV anterior mid-wall. Systolic velocities are directed upwards (positive) and diastolic velocities are directed downwards (negative). V_{ic}=velocity of isovolumic contraction; V_s=systolic thickening velocity; V_e=early diastolic velocity; V_a=late diastolic velocity. The scale is set at ± 10 cm s^{-1}.

wall motion in the revascularized territory can indicate graft failure and the need for revision of the anastomosis or another graft.[5] Given the importance of such decisions, objective support of visual assessment would be useful. Tissue Doppler imaging (TDI) allows quantitative assessment of LV wall motion[6 7] and may become such a method. Transthoracic TDI detects myocardial ischaemia in animals[8] and in awake patients during coronary angioplasty[9] or dobutamine stress testing.[10 11] In this study, we examined the feasibility of transoesophageal TDI for assessing LV wall motion during surgery in addition to standard TOE monitoring. We chose the LV anterior wall for TDI and measured myocardial velocity in this segment before and after revascularization of the left anterior descending coronary artery (LAD) using the left internal mammary artery (LIMA).

Patients and methods

We studied 42 patients having elective aortocoronary bypass surgery, including LIMA graft to LAD. The institutional ethics committee of the University of Basel approved the study and we obtained written informed consent. During surgery, we monitored leads II and V5 of the ECG with automatic ST segment analysis, radial artery, pulmonary artery and pulmonary capillary wedge pressures and thermodilution cardiac output (PCMS Workstation 19845-15-03; Spacelabs, Chatsworth, CA, USA). Anaesthesia was induced with either etomidate 0.2 mg kg^{-1} or thiopental 3–5 mg kg^{-1} and fentanyl 5 μg kg^{-1} and maintained with isoflurane (0.2–1.0 vol%) and additional fentanyl as needed. Pancuronium was used for muscle relaxation. The ventilator was adjusted to obtain an end-tidal P_{CO_2} of 4.5–5.0 kPa, oxygen saturation >95% and peak inspiratory pressure <20 cm H_2O. During cardiopulmonary bypass, isoflurane (up to 2.0 vol%) was administered via the oxygenator. With the onset of rewarming, an infusion of midazolam 0.6–1.0 μg kg^{-1} min^{-1} and fentanyl 0.03–0.06 μg kg^{-1} min^{-1} was started and continued into the postoperative period. The total mean (SD) dose of fentanyl, which was given until the final measurements were taken, was 1.8 (0.4) mg.

Cardiopulmonary bypass was with a Sarns pump, hollow-fibre oxygenator and two-stage right atrial cannula. Aortic root vent, single aortic cross-clamp and hypothermic cardioplegic arrest were used. Twenty-six patients (62%) received blood and 16 patients (38%) received crystalloid cardioplegia, given intermittently by the antegrade route. The perfusate temperature was allowed to drift to 32–34°C. The distal venous graft anastomoses were performed first, followed by LIMA anastomosis to the LAD artery. Proximal anastomoses were made during reperfusion and rewarming to 37°C. In all patients, both right atrial and ventricular bipolar pacing wires were placed and the heart was paced at 80 beats min^{-1} if the spontaneous rate was less than 75 beats min^{-1}. If support was needed during weaning, low-dose epinephrine alone or with a loading dose of amrinone was given and the dose was adjusted according to the response.

An omniplane TOE probe (4–7 MHz) and a Sonos 5500 ultrasound system (Agilent, Andover, MA, USA) were used to monitor LV function and TDI. First, a transgastric LV short-axis cross-section was obtained at mid-papillary muscle level and the image stored as a base-line cine loop on an optical disc. Then, after appropriate adjustment of the power, gain, scale and filter controls, the sampling volume (0.3 cm) of the TDI was placed in the anterior mid-wall and the myocardial velocity in this segment was recorded. Finally, transmitral blood flow velocities were recorded by pulsed-wave Doppler. For the off-line analysis, three representative cardiac cycles were recorded in end-expiration and stored on an optical disk. The recordings and the off-line analysis of the two-dimensional TOE loops and of the Doppler velocity signals were made according to the recommendations of the American Society of Echocardiography.[12 13] Depending on the extent of systolic wall thickening and radial shortening, the four segments of the mid-papillary short-axis cross section (anterior, lateral, inferior wall and septum) were graded on a 1–4 point scale as normal (grade 1), hypokinetic (2), akinetic (3) or dyskinetic (4). A global short-axis wall motion score was calculated by summation of the points assigned to each segment. The cross-sectional area of the LV was measured in end-diastole (EDA) and end-systole (ESA) and fractional area change was calculated as FAC%=(EDA–ESA/EDA)×100.[2] End-systolic wall stress was calculated as ESWS=0.334×SBP×ESD/ESWT(1+ESWT/ESD), where ESWT is end-systolic wall thickness of the anterior wall and ESD end-systolic internal diameter of the LV.[2] ESWT and ESD were obtained from M-mode TOE recorded in the same short-axis view.

Table 1 Patient characteristics. Values are median (95% confidence interval) or number (%) of patients

Characteristic	Value
Age (yr)	67 (60, 71)
Female	7 (17)
Weight (kg)	78 (75, 80)
Height (cm)	170 (168, 174)
One-vessel disease	1 (2)
Two-vessel disease	10 (24)
Three-vessel disease	31 (74)
Degree of LAD stenosis	
50–75%	2 (5)
75–95%	19 (45)
>95%	21 (50)
Left main stenosis (>50%)	10 (24)
LV ejection fraction	57 (45, 70)
Medication	
β-blockers	28 (67)
Calcium entry blockers	18 (43)
ACE inhibitors	5 (12)

LAD, left anterior descending coronary artery; ACE, angiotensin-converting enzyme.

Table 2 Haemodynamic variables. Values are median (95% confidence interval). *$P< 0.05$

	Before revascularization	After revascularization
HR (min^{-1})	62 (58, 65)	86 (84, 89)*
SBP (mm Hg)	110 (105, 115)	110 (102, 118)
DBP (mm Hg)	60 (57, 65)	63 (60, 65)
MAP (mm Hg)	78 (74, 82)	79 (72, 82)
RAP (mm Hg)	7 (6, 9)	10 (9, 11)*
PAP (mm Hg)	25 (24, 27)	27 (24, 30)*
PAOP (mm Hg)	12 (10, 14)	12 (11, 14)
CI ($l\ min^{-1}\ m^{-2}$)	2.1 (1.9, 2.3)	2.4 (2.3, 2.6)*
SVR ($dyn\ s\ cm^{-5}$)	1418 (1252, 1653)	1157 (1045, 1443)*

HR, heart rate; SBP, systolic arterial pressure; DBP, diastolic arterial pressure; MAP, mean arterial pressure; RAP, right atrial pressure; PAP, pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; CI, cardiac index; SVR, systemic vascular resistance.

From the TDI tracings, the following peak velocities were measured: velocity of isovolumic contraction (V_{ic}), systolic thickening velocity (V_s), early diastolic velocity (V_e) and late diastolic velocity (V_a)[8] (Fig. 1). In addition, the V_e/V_a ratio was calculated. From the transmitral flow velocity recordings we measured and calculated early (E) and late (A) filling velocities, E/A ratio, deceleration time (DT) of the E wave, diastolic filling time (FT), FT corrected for heart rate (FT_c = FT/vRR) and FT as a percentage of the RR interval of ECG.[13]

The baseline cardiovascular and TOE studies were done after induction of anaesthesia before surgical incision. The study after revascularization took place between sternal closure and the end of surgery, 79 (18) min after the release of the aortic cross-clamp.

Statistical methods

Data are presented as median and 95% confidence intervals. All statistical analyses were performed using StatView 5.0 (SAS Institute, Cary, NC, USA). Because the variables after revascularization were not normally distributed, non-parametric statistical tests were used for all analyses. Accordingly, the Wilcoxon signed rank test was used to compare continuous variables before and after revascularization. Correlations between continuous variables were analysed using the Spearman rank correlation test, whereas the relations between nominal and continuous data were analysed using logistic regression.

Reproducibility of TOE measurements

One experienced observer did the off-line analysis. The intra-observer variability [coefficient of variation, Bland–Altman analysis with mean difference (SD) and 95% limits of agreement, and kappa (κ) test] was tested by repeating the measurements in 12 randomly selected patients 6 months later.[14] In addition, a second masked observer independently analysed the TDI data of 12 randomly selected patients and the interobserver variability of TDI measurements was determined.

Results

The patients' characteristics are given in Table 1. All patients underwent uneventful bypass surgery. The median aortic cross-clamp time was 49.5 (range 23–101) min and three grafts (median; range 1–4) were performed, including LIMA graft to the LAD in all patients. In 17 patients the circulation was supported by epinephrine 1–4 $\mu g\ min^{-1}$ and amrinone 0.2–0.6 $mg\ kg^{-1}$ after cardiopulmonary bypass. During the final measurements, 32 patients (76%) had sinus rhythm, three patients (7%) sequential dual-chamber pacing and the remainder had atrial pacing. There was no difference in the spontaneous and paced heart rate (median 86 *vs* 84 beats min^{-1}). Logistic regression analysis did not indicate any effect of the pharmacological support, method of cardioplegia and cardiac pacing on any of the measurements. Haemodynamic variables are given in Table 2. After surgery, no patient had clinical, electrocardiographic or myocardial enzyme changes suggestive of intraoperative myocardial infarction.

Global LV function

Two-dimensional TOE showed a mild decrease in LV size and impairment in global wall-motion score (Table 3). Whereas the anterior wall motion tended to improve (P=0.05), there was a significant deterioration in the inferior wall motion (P=0.0007) and no significant changes in the two remaining segments. There were marked changes in transmitral flow at the end of surgery: an increase in A and a

Table 3 Global LV function. Values are median (95% confidence interval). *P<0.05

	Before revascularization	After revascularization
Systolic		
EDA (cm^{-2})	16 (14, 20)	15 (12, 16)*
ESA (cm^{-2})	7.2 (5.9, 10.3)	6.7 (4.6, 10.3)*
FAC (%)	52 (41, 58)	51 (40, 59)
ESWS (g cm^{-2})	83 (60, 102)	80 (60, 108)
LVWMI	4 (4, 6)	5 (5, 7)*
Diastolic		
E (cm s^{-1})	56 (49, 61)	49 (43, 56)*
A (cm s^{-1})	50 (46, 55)	56 (49, 65)*
E/A	1.1 (0.9, 1.2)	0.8 (0.8, 1.0)*
DT (ms)	179 (153, 189)	153 (133, 168)*
FT_c (ms)	468 (431, 520)	362 (332, 383)*
FT%	48 (46, 51)	42 (41, 45)*

EDA, end-diastolic area; ESA, end-systolic area; FAC, fractional area change; ESWS, end-systolic wall stress; LVWMI, LV wall motion score index (four-segment model of the short-axis view, with grades 1–4); E, peak early filling velocity of mitral inflow; A, peak atrial filling velocity of mitral inflow; DT, deceleration time of E velocity; FT_c, diastolic filling time (corrected for heart rate); FT%, duration of filling as a percentage of RR interval.

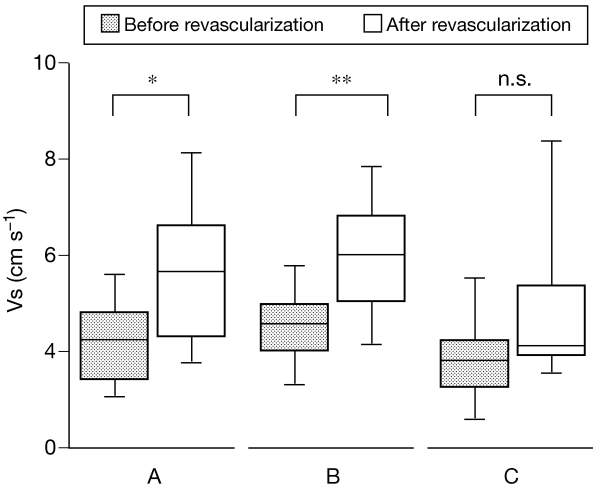

Fig 2 Box plot of changes in V_s before and after revascularization in all 42 anterior wall segments (A), in 29 initially normokinetic segments (B) and in nine initially hypokinetic or akinetic segments (C).V_s=thickening velocity recorded by tissue Doppler imaging. *P<0.05; **P<0.01; n.s.=not significant.

decrease in E velocity, reduction in E/A ratio and reduction in DT, FT_c and FT%.

Regional (anterior wall) LV function

Visual assessment

Before surgery, the anterior wall motion was graded as normokinetic, hypokinetic or akinetic in 29, nine and four patients respectively. After revascularization, the anterior wall motion grade did not change in 32 patients, improved in seven and became worse in three. Four of the nine hypokinetic segments and three of the akinetic segments improved to normokinesia and hypokinesia respectively. Two segments with normal wall motion before surgery became hypokinetic and one hypokinetic segment became akinetic. The anterior wall-motion grade did not change and correlated with FAC% both before and after surgery (r=0.72 and 0.55, P<0.0001 and P<0.001 respectively).

TDI: systolic velocities

We obtained good quality recordings of anterior wall velocity in all patients. During LV ejection, TDI showed positive (inwardly directed) thickening velocity in all segments, even in those visually graded as akinetic. Logistic regression analysis showed an inverse relationship between increasing anterior wall motion abnormality and decreasing V_s (P<0.01). Furthermore, when we compared segments graded visually as normal (n=33) with those considered abnormal (n=15), we found a smaller V_s in abnormal than in normal segments (3.8 (3.2, 4.4) *vs* 4.5 (4.3, 4.8) cm s^{-1}, P<0.05). After revascularization, both V_{ic} and V_s increased. The increase in V_s was the result of a marked response of the initially normal segments, whereas segments that were initially abnormal showed only a trend to an increase in V_s (Fig. 2). The median change in V_s (ΔV_s%) was +40% in segments with improved wall-motion grade, +27% in segments with unchanged wall motion and 14% in segments when wall motion worsened. V_s showed an inverse correlation with end-systolic wall stress (r=–0.56 and –0.37, P<0.001 and <0.05 before and after surgery respectively); V_s also correlated directly with FAC% (r=0.42 and 0.36, P<0.01 and <0.05 before and after surgery respectively). In contrast, we found no association between V_s and EDA. Postsystolic shortening was detected by TDI in three patients before surgery; this disappeared in one patient after surgery and a further patient developed this feature.

TDI: diastolic velocities

During diastole, TDI recorded two outwardly directed velocities, V_e and V_a, in the anterior wall in all patients. Before surgery V_e was higher than V_a and the V_e/V_a ratio was >1.0. The correlation between transmitral E/A and anterior wall V_e/V_a ratio was significant (r=0.41, P<0.01).

After surgery V_e did not change, whereas V_a increased, so the V_e/V_a ratio decreased (Table 4). The diastolic anterior wall velocities and their ratios were similar in normal and abnormal segments.

Reproducibility

Intra-observer variation in measurements of the two-dimensional TOE had the following coefficients of variation and mean (SD) differences: 4.7% and 0.8 (0.9) cm^2 for EDA, 5.5% and –0.2 (0.7) cm^2 for ESA, 4.8%, 0.2 (2.9)% for FAC% and 2%, –0.03 (0.2) for anterior wall-motion grade. The intra-observer agreement for evaluation of LV wall

Table 4 LV anterior wall function by TDI. Values are median (95% confidence interval). *$P<0.05$

	Before revascularization	After revascularization
V_{ic} (cm s^{-1})	3.9 (3.3, 4.5)	5.2 (4.0, 5.8)*
V_s (cm s^{-1})	4.2 (4.0, 4.7)	5.7 (4.8, 6.3)*
V_e (cm s^{-1})	4.9 (4.4, 5.9)	5.4 (4.3, 6.4)
V_a (cm s^{-1})	3.5 (3.1, 3.9)	6.0 (5.1, 6.9)*
V_e/V_a	1.5 (1.2, 1.7)	1.0 (0.8, 1.2)*

V_{ic}, isovolumic contraction velocity; V_s, systolic thickening velocity; V_e, early diastolic velocity; V_a, late diastolic velocity.

Table 5 Reproducibility of tissue Doppler imaging

	Variability (%)	Mean difference (cm s^{-1})	SD	95% limits of agreement
Intra-observer				
V_{ic}	5.3	0.1	0.4	–0.7 to 0.9
V_s	4.1	0.1	0.3	–0.5 to 0.7
V_e	5.9	0.1	0.4	–0.7 to 0.9
V_a	5.7	0.1	0.3	–0.5 to 0.7
Interobserver				
V_{ic}	6.2	–0.1	0.4	–0.9 to 0.7
V_s	3.4	–0.1	0.2	–0.5 to 0.3
V_e	5.9	–0.2	0.4	–1.0 to 0.6
V_a	3.6	–0.1	0.2	–0.5 to 0.3

V_{ic}, isovolumic contraction velocity; V_s, systolic thickening velocity; V_e, early diastolic velocity; V_a, late diastolic velocity.

motion was very good, with a score of $\kappa=0.93$. These data and the results of the Bland–Altman analysis of the TDI measurements are given in Table 5.

Discussion

We showed that TDI can be used for quantitative analysis of LV anterior wall motion by TOE during surgery. We obtained reproducible myocardial velocity measurements in all patients and the spectral velocity patterns were comparable with those measured by the transthoracic approach at mid-ventricular level in awake subjects, as described in previous studies.[6 15–17] The value of this novel method for monitoring LV function needs to be further evaluated. Preliminary conclusions from our study are that TDI of the anterior wall gives additional and easily accessible information on both regional and global systolic and diastolic LV function. TDI, even limited to the anterior wall, could be a useful intraoperative monitoring tool.

Systolic velocities

During systole, TDI showed an inward thickening velocity in all the segments examined. No outward (dyskinetic) velocities were observed. In contrast, no systolic motion was seen in some patients, and therefore these segments were graded as akinetic. This disagreement can be explained by the fact that the observer evaluates the motion of the entire segment encompassing one-fourth of the entire LV endocardial circumference in the short axis, whereas the V_s is measured within the small sampling volume. Nevertheless, we found a difference in V_s between normal and abnormal segments and a significant correlation between segmental wall motion grade and V_s. However, there was a wide overlap of V_s values between normal and abnormal segments. Because of this overlap and the small number of abnormal segments, it was neither possible to define the V_s values associated with different grades of wall motion nor to distinguish abnormal from normal function.[6] Previous studies using transthoracic TDI found more obvious differences in V_s among segments with different grades of wall motion. However, the inwardly-directed systolic velocities were still present even in severely hypokinetic and akinetic segments.[16 18–21] The wide range of absolute velocities described in the literature can be explained by the different techniques of TDI that are used: for instance, pulsed-wave TDI records peak velocities whereas colour-coded TDI provides mean velocities, and circumferential and longitudinal velocities are dissimilar even within one segment. Furthermore, velocity increases from the apex towards the base and is greater in the posterior wall.[22] Consequently, the absolute value of V_s at rest may not distinguish between normal and dysfunctional segments. However, this important separation has been shown to be possible with TDI using an inadequate increase in V_s during increased myocardial oxygen demand, such as during exercise or the dobutamine stress test.[10 11 16 19 21] We found an overall increase in V_s after revascularization in both unchanged and improved segments but only a marginal V_s increase in the three segments with deterioration in wall motion after surgery. We speculate that the post-bypass period, with increased catecholamines and other mediators and higher heart rates, stressed the myocardium, which responded with an increase in V_s, depending on its functional status. The increase in V_s could also be a real improvement in function brought about by LAD revascularization, or the successful revascularization may be necessary for an increase in V_s when oxygen demand increases. During experimental coronary occlusion (supply type of ischaemia), V_s decreases towards zero and eventually changes its direction (paradoxical outward motion) with increasing ischaemia.[8] Comparable changes have also been reported during angioplasty of the LAD.[9] We did not observe any such changes in our patients and conclude that no relevant ischaemia occurred immediately after LIMA grafting (with the possible exception of the three patients mentioned above).

Theoretically, an increase in V_s could also be caused by the greater motion of the entire heart that occurs after open-heart surgery and can be recorded by TDI.[1] However, this systolic motion is anterior and would therefore be expected to reduce the velocity of the anterior wall, which moves

posteriorly towards the oesophageal transducer during systole.

V_s showed no association with EDA, which is an index of LV preload, but correlated inversely with ESWS, which is an index of LV afterload. Therefore, V_s might also have been influenced by intraoperative changes in LV afterload. This is unlikely because ESWS did not change after surgery and there was no association between percentage changes in ESWS and V_s.

Diastolic velocities

At baseline, the diastolic myocardial velocities in the normal and abnormal anterior wall segments were similar. This unexpected finding could have resulted from diastolic dysfunction even in segments with normal systolic function. The association of preserved systolic function with abnormal diastolic function at rest has been detected by TDI in myocardial segments supplied by stenotic coronary arteries.[23] With increased myocardial oxygen demand in the normal myocardium, an increase in V_s is accompanied by an increase in V_e.[19] In contrast, despite the increase in V_s, V_e did not change in our patients, whereas V_a increased, resulting in a fall in V_e/V_a ratio. This suggests impairment in regional diastolic function and occurs at the same time as the mitral flow changes. Transient diastolic dysfunction after coronary artery bypass surgery, associated with a decrease in E/A ratio, has been described.[24] The change in both the regional and global (transmitral) diastolic velocity pattern in our study may result from several factors, such as myocardial stunning, increased heart rate and cardiac output, and shortened filling time, causing a change in LV filling to late diastole.

Clinical implications

By visual assessment, 54% of the dysfunctional anterior segments improved after revascularization. Only 7% of all segments deteriorated. Previous studies of wall thickening also reported improved function of abnormal segments after revascularization with venous grafts.[25–27] On the other hand, transient impairment in the region supplied by the LAD has been described after LIMA grafting.[28 29] The rarity of this impairment in our study may reflect improvements in surgical technique and in myocardial and LIMA preservation. In the past, internal mammary hypoperfusion syndrome has been described, seen as postoperative myocardial ischaemia in the LAD territory, recurrent ventricular dysrhythmias and/or life-threatening haemodynamic compromise.[5 30–32] These patients can be saved if the diagnosis is made quickly and a venous graft is placed in the same region.[5] More recently, the use of the radial artery as a bypass conduit has again raised concern about increased incidence of intraoperative ischaemia.[33] Monitoring of the anterior wall motion by TDI may help in such critical situations after arterial grafting, and in all patients undergoing non-cardiac surgery who are at risk of ischaemia in the LAD supply region.

Limitations

This first use of TDI to assist monitoring has important limitations. First, the study was limited to the anterior wall of the LV and the transgastric short-axis cross-section for several reasons. In the anterior wall optimal alignment between the ultrasound beam and the moving object is possible, and the anterior wall function indicates the success of the LIMA grafting. In addition, the mid-papillary short-axis view is the most useful and reliable cross-section for continuous intraoperative TOE monitoring[4] and anterior wall function is an important determinant of the global LV function.[34] However, the feasibility of using TDI in other regions of the LV, and hence in other TOE views, should be examined in future studies. The anatomy and function of the myocardium means that in the anterior mid-wall TDI measures the velocity of circumferential fibres. These fibres dominate during ejection and early relaxation.[35] Myocardial velocity would have been greater if measured in the subendocardium and less if it had been measured in the subepicardium.[22 36] Because of this physiological transmural velocity gradient, a small difference in the position of the sampling volume could affect the accuracy of measurement. Thus, TDI recordings from several positions and depths could provide additional information. We measured only peak velocity of the TDI curve because it is simple, expeditious and easily done on-line. Although additional measurements of early and late ejection velocity, mean velocity, velocity time integral and of timing variables may allow more insight into the regional function, they appear unsuitable for intraoperative on-line monitoring.

Myocardial velocity recorded by pulsed-wave tissue Doppler indicates the true shortening and relaxation velocity and the velocity of the translational and twisting motion of the ventricle. To avoid these confounding factors, velocity gradient rather than absolute velocity or wall strain and strain rate can be measured.[37] Such techniques, using colour-coded tissue Doppler, require special equipment not generally available at present.[22] For serial measurements, however, colour and pulsed-wave TDI and velocity and strain measurements offer comparable information on changes in regional wall motion.[16 38 39]

Conclusions

TDI complements standard intraoperative TOE monitoring of LV function by recording myocardial velocities in the LV anterior wall. We found that systolic thickening velocity correlated with anterior wall motion and global LV systolic function, whereas the diastolic velocity reflected LV filling. However, it was not possible to define the velocities associated with normal and different grades of abnormal

anterior wall function. Nevertheless, the measurement of peak anterior wall velocity and particularly of the changes after surgical revascularization appears to be a valuable addition to visual assessment of wall motion. Because of the paucity of new wall motion abnormalities in our patients, the value of TDI in the detection of myocardial ischaemia after LIMA grafting is not yet clear and should be studied further.

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