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Relationship between echocardiographic index of ventricular filling pressure and intraoperative haemodynamic changes during off-pump coronary bypass surgery

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Background. The ratio of mitral velocity to early-diastolic velocity of the mitral annulus (E/e') is an indicator of diastolic function representing acute loading conditions of the left ventricle. We tested the efficacy of E/e' as a predictor of haemodynamic derangement during off-pump coronary artery bypass surgery (OPCAB), when heart displacement causes loading changes.

Methods and results. Fifty patients with left ventricular (LV) ejection fraction $\geq$50% were divided into two groups; $E/e'<8$ (normal LV filling pressure, $n=25$) and >15 (increased LV filling pressure, $n=25$). Haemodynamic measurements were recorded after induction of anaesthesia, during grafting, and after sternum closure. Patients' characteristics and operative data were similar between the groups. Cardiac index and mixed venous oxygen saturation were significantly lower during grafting and after sternum closure in the $E/e'>15$ group, compared with $E/e'<8$ group and with the baseline values. The $E/e'>15$ group required significantly longer ventilation time and length of stay in the intensive care unit.

Conclusions. Even in patients with preserved systolic LV function, patients with $E/e'>15$ were more prone to undergo a significant decrease in cardiac output during OPCAB, which did not return to baseline level after completion of grafting. Whether this finding is associated with increased morbidity and mortality should be validated.

Exposure of the grafting site during off-pump coronary bypass surgery (OPCAB) requires displacement of the heart, resulting in a decreased cardiac index (CI) and mixed venous oxygen saturation (Sv_{O_2}).[1 2] Although these haemodynamic consequences are usually transient and well tolerated,[3 4] haemodynamic collapse unresponsive to corrective measures does occur, requiring emergent cardiopulmonary bypass that is associated with poor prognosis.[5 6]

Underlying mechanisms for these iatrogenic haemodynamic changes during grafting are mainly ascribed to impaired filling and diastolic dysfunction of the right and left ventricle.[1 2 4 7 8] Thus, even with preserved systolic function, patients with diastolic dysfunction would be more prone to undergo significant haemodynamic derangement during grafting, however, evidence to support this hypothesis is lacking.

The ratio of early transmitral flow velocity to early-diastolic velocity of the mitral annulus (E/e') is an indicator of the diastolic function that correlates well with left ventricular (LV) filling pressure.[9 10] In addition, a number of studies have demonstrated increased morbidity, mortality or both in patients with elevated E/e' value after myocardial infarction.[11 12]

We therefore evaluated the relationship between preoperative E/e' value and intraoperative changes of haemodynamic variables and patients' outcome in patients with preserved systolic LV function undergoing OPCAB, in a prospective observational study.

Methods

After approval of Institutional Review Board and getting consent from patients, 50 patients undergoing elective, isolated, multivessel OPCAB between January and September 2007 were studied. Basic inclusion criteria were patients with stable angina having triple vessel disease, NYHA class ≤ 2, LV ejection fraction (LVEF) $\geq 50\%$. Patients with pre-existing pulmonary, hepatic disease or both, concomitant valvular heart disease, pulmonary hypertension, serum creatinine (Cr) >1.3 mg dl^{-1}, and myocardial infarction within 1 month were excluded.

Patients were divided into two groups according to their E/e' value measured 1 day before surgery, by the cardiologists using transthoracic echocardiography; $E/e'<8$ (normal LV filling pressure, $n=25$) and $E/e'>15$ (elevated LV filling pressure, $n=25$).[9][10] Early transmitral inflow velocity (E) was assessed by pulsed-wave Doppler from the apical four-chamber view with $1-2$ mm sample volume. Early-diastolic velocity of mitral annulus (e') was assessed by pulsed-wave Doppler tissue imaging of the septal mitral annulus from the same view with $2-4$ mm sample volume.

All patients received $0.05-0.1$ mg kg^{-1} of morphine i.m. as premedication 1 h before operation. Upon arrival at the operating theatre, standard monitoring devices were applied including a pulmonary artery catheter (PAC, Swan-Ganz CCOmbo CCO/Sv$_{O_2}$™, Edwards Lifesciences LLC, Irvine, CA, USA), which was inserted through the right internal jugular vein and connected to an analysis system (Vigilance™, Edwards Lifesciences LLC, Irvine, CA, USA) for continuous monitoring of CI and (Sv$_{O_2}$). Anaesthesia was induced with i.v. midazolam ($0.03-0.07$ mg kg^{-1}) and sufentanil ($1.5-2.0$ µg kg^{-1}), and maintained with sevoflurane ($0.8-1.5\%$) and continuous infusion of sufentanil ($0.5-1.5$ µg kg^{-1} h^{-1}). Neuromuscular block was achieved by administering rocuronium (0.9 mg kg^{-1}) and maintained with continuous infusion of vecuronium ($1-2$ µg kg^{-1} min^{-1}). Isosorbide dinitrate 0.5 µg kg^{-1} min^{-1} was infused in all patients throughout the study period. The patients' lungs were ventilated with a tidal volume of $8-10$ ml kg^{-1}, *I:E* ratio of 1:2, at a rate of $8-12$ breaths min^{-1} in 60% oxygen with air and no PEEP during the surgery. After induction of anaesthesia, a transoesophageal echocardiography probe was inserted to assess the global cardiac function and detect newly developing segmental wall motion abnormalities. Intravascular volume replacement was managed with crystalloid and colloid solutions to maintain the pulmonary capillary wedge pressure (PCWP) between 8 and 16 mm Hg according to the baseline values before enucleation of the heart and after completion of grafting. During the period of heart displacement, a crystalloid solution was infused at a fixed rate of $6-8$ ml kg^{-1}, whereas a colloid solution was infused to compensate for the amount of blood loss collected by a cell salvage device. Salvaged blood by the cell salvage device was re-infused to the patient before the end of the surgery. Haemodynamic management during the period of heart displacement and grafting was as follows: (i) maintenance of a mean systemic arterial pressure (MAP) above 70 mm Hg either with $10-20°$ Trendelenburg position or with norepinephrine (NE) infusion, (ii) infusion of milrinone in patients with Sv$_{O_2}$ < 60% for >10 min, development of mitral regurgitation $\geq$Grade 3 with a concomitant increase in mean pulmonary arterial pressure (MPAP) >30 mm Hg or both. Allogenic-packed red blood cells (pRBCs) were transfused when the haematocrit (Hct) level was $<25\%$ throughout the study period. The central temperature measured by PAC was maintained $>36°C$ with a warm mattress, a forced warm air blanket, and a fluid warmer as necessary.

All surgical procedures were performed by one surgeon through a median sternotomy, and the heart was displaced using posterior pericardial stitch, large gauze (12×70 cm) swabs, and tissue stabilizer (Octopus Tissue Stabilization System®, Medtronic Inc. USA). Vertical right pericardiotomy was also performed to minimize compression of the right ventricle. The sequence of grafting was always the left internal mammary artery to left anterior descending coronary artery (LAD) first, followed by grafting on the circumflex coronary artery (LCx) and the right coronary artery (RCA) by way of composite Y graft consisting of radial artery or saphenous vein with left internal mammary artery, by use of right internal mammary artery as necessary or by both. An intracoronary shunt was used during grafting procedures on the LAD and distal RCA. All patients were transferred to the intensive care unit (ICU) after surgery.

Assessed operative data were duration of surgery, number of grafts performed, graft reconstruction time, amount of infused fluid, and urine output. Haemodynamic variables obtained from the PAC and arterial line were recorded 15 min after induction of anaesthesia (baseline, T1), 10 min after stabilizer application for LAD grafting (T2), LCx grafting (T3), RCA grafting (T4), and 15 min after sternum closure (T5), and include (Sv$_{O_2}$), CI, heart rate (HR), MAP, central venous pressure (CVP), MPAP, PCWP, and right ventricular ejection fraction (RVEF). Assessed laboratory variables were Cr, creatine kinase-MB (CK-MB), arterial oxygen tension (Pa$_{O_2}$) and Hct. Pa$_{O_2}$ and Hct were assessed 15 min after induction of anaesthesia and after sternum closure. Cr and CK-MB were assessed 1 day before surgery and 12 and 24 h after surgery. Of the postoperative Cr measurements, number of patients with Cr>2.0 mg dl^{-1} was recorded. Of the two postoperative CK-MB values, the highest value was recorded and also the number of patients with CK-MB elevations >5 times the upper limit of normal (>25 ng ml^{-1}) was recorded. In the ICU, the following variables in conjunction with five major morbidity endpoints were assessed and recorded: permanent stroke, renal dysfunction requiring dialysis, haemostatic re-exploration, deep sternal wound infection, number of patients requiring prolonged ventilation (>48 h), and the time to extubation and length

of stay in the ICU.[13] Decisions for extubation and discharge from the ICU were made at the discretion of the ICU staff, consisting of cardiothoracic surgeons and anaesthetists not aware of this study, according to the standard ICU protocols of our institution. Criteria for weaning from ventilatory support included an appropriate sensorium, haemodynamic stability ($CI > 2.2$ litre min^{-1} m^{-2}; MAP 60 mm Hg; pulmonary artery diastolic pressure <20 mm Hg; and no significant arrhythmias), $Pa_{O_2} > 10.7$ kPa with an $F_{I_{O_2}} = 0.4$, minimal chest tube drainage, urine output 0.5 ml kg^{-1} h^{-1}, and temperature $>35.5°C$. Discharge criteria from the ICU were as follows: stabilized patient's clinical status without the need for ICU monitoring and care (which include no further requirement for either inotropic or vasoactive agents except NE infusion <0.05 μg kg^{-1} min^{-1}), and no plan for further active intervention.

Statistical analyses were performed using SPSS 12.0 (SPSS Inc., Chicago, IL, USA). All data are expressed as number of patients or mean (SD). We determined that 22 patients would be required in each group with 90% power to detect a 0.5 litre min^{-1} m^{-2} difference in CI between the groups with a SD of 0.5 litre min^{-1} m^{-2} and alpha level of 0.05 using independent t-test.[14] Data between the groups were compared using χ^2 test, Fisher's exact test, or independent t-test where appropriate. For between-group comparisons of haemodynamic data, independent t-test with Bonferroni *post hoc* test was used. Changes between time points within the groups were compared using repeated measures of analysis of variance. A P-value of <0.05 was considered statistically significant.

Results

Off-pump coronary bypass surgery could be successfully performed in all 50 patients without requiring emergent cardiopulmonary bypass. Patients' characteristics were similar between the groups with a trend towards older age ($P=0.096$) in the $E/e' > 15$ group (Table 1). Operative data were also similar between the groups with none of the patients requiring infusion of milirinone during the surgery, except the total amount of infused NE to maintain MAP >70 mm Hg during the surgery, which was significantly larger in the $E/e' > 15$ group (Table 2). Post-induction Pa_{O_2} [32.9 (8.8) and 32.7 (8.8) kPa in the $E/e' < 8$ and >15 group, respectively, $P=0.913$] and Hct [34 (5)% in both groups, $P=0.855$] was similar between the groups. Hct after sternum closure was also similar between the groups [28 (4)% and 29 (3)% in the $E/e' < 8$ and >15 group, respectively, $P=0.719$], whereas Pa_{O_2} was significantly lower in the $E/e' > 15$ group [27.9 (5.6) *vs* 21.3 (5.1) kPa, $P<0.001$].

Baseline haemodynamic variables recorded at T1 were all similar between the groups with a trend towards higher MPAP ($P=0.079$) and PCWP ($P=0.078$) in the $E/e' > 15$ group. In intergroup comparisons of haemodynamic variables, Sv_{O_2} and CI were significantly lower in the $E/e' > 15$

Table 1 Patient characteristics. Data are given as mean (SD) or absolute numbers. ACEIs, angiotensin converting enzyme inhibitors; Cr, creatinine; CK-MB, creatinine kinase-MB; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; RCA, right coronary artery

	$E/e' < 8$ (n=25)	$E/e' > 15$ (n=25)	P-value
Age (yr)	62 (9)	66 (6)	0.096
Gender (M/F)	19/6	15/10	0.364
Body surface area (m^2)	1.7 (0.1)	1.7 (0.2)	0.568
Diabetes	3	6	0.289
Hypertension	13	19	0.140
Preoperative medication			
β-Blockers	17	22	0.171
Calcium channel blockers	13	15	0.776
ACEIs	8	9	1.000
Preoperative Cr (mg dl^{-1})	1.1 (0.3)	1.0 (0.2)	0.327
Preoperative CK-MB (ng ml^{-1})	2.8 (1.1)	3.3 (1.4)	0.174
LVEF (%)	63 (8)	62 (6)	0.540
LVEDD (mm)	48 (4)	48 (5)	0.993
LVESD (mm)	31 (4)	33 (6)	0.189
Degree of stenosis (%)			
LAD	84 (9)	88 (9)	0.233
LCx	88 (12)	83 (12)	0.309
RCA	83 (15)	80 (13)	0.601

Table 2 Operative data. Data are given as mean (SD). NE, norepinephrine; pRBCs, packed red blood cells. *$P<0.05$ compared with the $E/e' < 8$ group

	$E/e' < 8$ (n=25)	$E/e' > 15$ (n=25)	P-value
Operation time (min)	325 (64)	323 (66)	0.915
Number of grafts per patient	3.5 (0.7)	3.6 (0.7)	0.693
Total graft reconstruction time (min)	35 (5)	36 (4)	0.175
Input			
Crystalloid (ml)	2902 (1694)	3025 (1268)	0.795
Colloid (ml)	1220 (247)	1172 (275)	0.548
pRBCs (unit)	0.3 (0.7)	0.2 (0.5)	0.826
Salvaged blood (ml)	232 (153)	258 (238)	0.682
Urine output (ml)	867 (140)	920 (181)	0.256
Amount of infused NE (μg)	124 (140)	250 (243)*	0.029

group at T2, T3, T4, and T5 (all $P \leq 0.01$). Other haemodynamic variables did not show any significant intergroup differences at each time point of measurements (Table 3). In intragroup comparisons of haemodynamic variables, Sv_{O_2} was significantly decreased at T3 ($P=0.016$) and T4 ($P=0.001$) and CI was significantly decreased only at T3 ($P=0.001$) compared with the baseline values in the $E/e' < 8$ group. In contrast, Sv_{O_2} and CI at T2, T3, T4, and T5 were significantly decreased compared with baseline value in the $E/e' > 15$ group (all $P \leq 0.001$). HR was significantly increased at T5 ($P=0.002$) in the $E/e' < 8$ group and at T4 ($P=0.009$) and T5 ($P<0.001$) in the $E/e' > 15$ group compared with baseline values of each group. CVP was significantly increased at T2 ($P=0.028$), T3, ($P=0.002$), and T4 ($P<0.001$) in the $E/e' < 8$ group and at T3 ($P=0.001$) and T4 ($P=0.023$) in the $E/e' > 15$ group compared with baseline values of each group. MPAP was significantly increased at T3 in both groups ($P=0.001$ and 0.005 in the $E/e' < 8$ and >5 groups, respectively)

Table 3 Haemodynamic data. Data are given as mean (SD). T1, 15 min after induction of anaesthesia; T2, 10 min after stabilizer application for left anterior descending coronary artery grafting; T3, 10 min after stabilizer application for left circumflex coronary artery grafting; T4, 10 min after stabilizer application for right coronary artery grafting; T5, 15 min after sternum closure. Sv_{O_2}, mixed venous oxygen saturation; CI, cardiac index; HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; MPAP, mean pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; RVEF, right ventricular ejection fraction. *$P<0.05$ compared with the $E/e'<8$ group at each time point of measurement; †$P<0.05$ compared with values at T1 in each group

	E/e' group	T1	T2	T3	T4	T5
Sv_{O_2} (%)	<8	78 (6)	76 (5)	73 (5)†	72 (6)†	75 (5)
	>15	77 (4)	69 (7)*†	64 (7)*†	65 (6)*†	70 (5)*†
CI (litre min⁻¹ m⁻²)	<8	2.9 (0.5)	2.7 (0.5)	2.4 (0.4)†	2.5 (0.5)	2.8 (0.5)
	>15	2.8 (0.5)	2.3 (0.5)*†	1.9 (0.3)*†	1.9 (0.3)*†	2.3 (0.4)*†
HR (beats min⁻¹)	<8	63 (9)	66 (9)	67 (8)	68 (7)	72 (10)†
	>15	59 (7)	62 (7)	63 (7)	67 (9)†	68 (7)†
MAP (mm Hg)	<8	79 (10)	77 (7)	76 (6)	77 (9)	81 (8)
	>15	79 (11)	78 (6)	78 (5)	76 (5)	81 (13)
CVP (mm Hg)	<8	8 (2)	10 (1)†	10 (2)†	11 (3)†	9 (2)
	>15	8 (2)	10 (3)	11 (2)†	11 (3)†	9 (2)
MPAP (mm Hg)	<8	17 (3)	18 (3)	20 (2)†	18 (3)	17 (2)
	>15	18 (2)	20 (3)	21 (2)†	19 (3)	18 (3)
PCWP (mm Hg)	<8	12 (3)	14 (3)†	14 (2)†	14 (2)†	13 (2)
	>15	13 (2)	14 (3)	15 (2)†	14 (3)	14 (2)
RVEF (%)	<8	35 (5)	32 (4)	30 (5)†	31 (5)	32 (5)
	>15	35 (6)	32 (3)	31 (5)†	32 (2)	32 (4)

compared with baseline value of each group. PCWP was significantly increased at T2 ($P=0.048$), T3 ($P=0.005$), and T4 ($P=0.033$) in the $E/e'<8$ group and at T3 ($P=0.018$) in the $E/e'>15$ group compared with the baseline values of each group. RVEF was significantly decreased at T3 in both groups ($P=0.022$ and 0.019 in the $E/e'<8$ and >15 group, respectively), compared with baseline values of each group (Table 3).

In the ICU, postoperative CK-MB concentrations were similar between the groups [8.0 (6.9) and 9.4 (6.3) ng ml⁻¹ in the $E/e'<8$ and >15 group, respectively, $P=0.463$] and one patient in each group had CK-MB elevations more than five times the upper limit of normal. None of the patients developed permanent stroke, Cr>2.0 mg dl⁻¹, renal dysfunction requiring haemodialysis and deep sternal wound infection or all. Also, none of the patients required haemostatic re-exploration or prolonged ventilation. Time to extubation [657 (199) *vs* 856 (290) min, $P=0.007$] and length of stay in the ICU [2.8 (0.6) *vs* 3.4 (0.8) days, $P=0.004$] were significantly longer in the $E/e'>15$ group.

Discussion

The current prospective trial, addressing the relationship between echocardiographic index of increased LV filling pressure assessed with E/e' and iatrogenic haemodynamic changes during heart displacement in OPCAB, demonstrated that, even with preserved systolic LV function, patients with elevated E/e' (>15) had significantly lower CI and Sv_{O_2} during grafting. Furthermore, the decrease in CI and Sv_{O_2} did not return to baseline values after completion of grafting and these patients required significantly larger amount of NE during surgery, and a longer time to extubation and length of stay in the ICU.

In order to expose the target coronary grafting site during OPCAB, the heart must be displaced resulting in various degrees of iatrogenic haemodynamic compromise. Alteration of the geometry of the heart has several consequences contributing to the haemodynamic compromise. First, the atria are placed below the corresponding ventricles resulting in increased atrial size and pressure.[7][8] Secondly, there is direct compression of the right heart leading to RV dysfunction.[4][7] Thirdly, distortion of the tricuspid and mitral annuli occur that may result in either stenosis or regurgitation of the corresponding valve.[8] Fourthly, the stabilizer device restricts the myocardium causing regional myocardial dysfunction.[3] In combination, these changes manifest mainly as impaired filling and diastolic dysfunction of the ventricles resulting in reduced stroke volume and thus decrease in CI, Sv_{O_2} and MAP, especially during exposure of the posterior wall.[1][2] Although earlier reports were more focused on the RV dysfunction,[4][7] the resultant haemodynamic compromise is considered to arise from bi-ventricular contribution.[5][8][15] George and colleagues[8] have demonstrated mitral and pulmonary venous flow of restrictive filling pattern and diastolic dysfunction of the left ventricle during mechanical displacement of the heart in OPCAB.

The haemodynamic consequences of heart displacement are usually transient, reversible, and well tolerated by augmenting preload and maintaining MAP;[3][4] however, decrease in CI to <2.0 litre min⁻¹ m⁻², $Sv_{O_2}<60\%$ for >15 min or both despite corrective measures do occur necessitating conversion to on-pump coronary artery bypass surgery.[1] Aborted OPCAB is associated with significantly higher morbidity and mortality mandating identification of patients' risk factors,[5][6] and it seems reasonable to assume that, even with preserved systolic function, patients with pre-existing diastolic dysfunction would be more prone to develop a significant haemodynamic compromise during grafting. Yet, evidence to support this hypothesis was lacking.

E/e' is an indicator of diastolic function, which correlates well with LV filling pressure, representing acute loading conditions of the left ventricle.[9] [10] $E/e'<8$ and >15 accurately predicted normal and increased mean LV diastolic pressure, respectively, for all levels of systolic function, whereas E/e' between 8 and 15 showed a poor correlation.[9] These two cut-off values of E/e' also have good correlation with B-type natriuretic peptide concentration, which has been shown to predict congestive heart failure with good correlation to LV filling pressure as well.[10] [16] [17] Elevation of filling pressure is almost always associated with a structural or functional abnormality of the heart and, haemodynamically, it is a unifying feature for heart failure.[18] Moreover, e' reflects the rate of myocardial relaxation and therefore, in terms of diastolic function, E/e' may be a more valuable parameter than directly measured LV end-diastolic pressure. In conjunction, a number of studies have validated an increased morbidity, mortality or both in patients with elevated E/e' value (>15) after myocardial infarction,[11] [12] whereas increased LV end-diastolic pressure after myocardial infarction was not an independent risk factor of heart failure and survival.[19]

In the current study, patients with $E/e'>15$ exhibited significantly lower CI and Sv_{O_2} compared with patients with $E/e'<8$ during mechanical displacement of the heart and after completion of grafting with the heart in the natural position. In addition, when CI and Sv_{O_2} were compared with baseline values of each group, both variables in the $E/e'>15$ group were decreased during the entire grafting period and did not return to baseline values after sternum closure. In contrast, CI and Sv_{O_2} in the $E/e'<8$ group were only decreased either during grafting at LCx or during RCA and returned to baseline value after sternum closure. Also, patients with $E/e'>15$ required a significantly larger amount of NE to maintain the MAP>70 mm Hg, while in both groups CVP and PCWP were maintained well above the baseline levels. Considering that haemodynamic consequence of right heart compression were similar between the groups as shown with similar RVEF between the groups throughout the study period, these results clearly validate our hypothesis.

Patients with preserved LVEF are usually considered as low-risk patients for OPCAB.[20] [21] However, although we could not observe any differences in development of five major morbidity endpoints, patients with $E/e'>15$ had significantly lower Pa_{O_2} after sternum closure and subsequently required longer ventilation time and also length of stay in the ICU. Patients with elevated LV filling pressure require a higher filling pressure to maintain adequate CI, and at the same time are more prone to development of pulmonary oedema with preload augmentation.[22] [23] As this was a prospective observational study, similar fluid management protocol was applied in both groups. However, fluid management was done generously including administration of colloids in both groups, which subsequently led to lower Pa_{O_2} in the $E/e'>15$ group compared with the $E/e'<8$ group.

Also, evidence exists with regard to association of intraoperative low cardiac output and postoperative renal replacement therapy or prolonged ICU stay.[24] [25] As none of the patients met the criteria for milrinone infusion during grafting and only NE was infused to maintain predefined MAP. Addition of inotropics with lusitrophic effect in patients with already elevated LV filling pressure would yield better cardiac performance and more appropriate than NE infusion alone. Also, improved lusitrophy and cardiac performance may lead to reduced risk of pulmonary oedema formation and length of ICU stay. However, to justify inotropic infusion in patients with coronary artery occlusive disease before completion of grafting not on the basis of decreased Sv_{O_2} ($<60\%$ as in this study) but only on the presence of diastolic dysfunction at the expense of the potential risk of aggravating ischaemia–reperfusion injury, further studies are required to validate the safety.

As this is the first study to address the relationship between E/e' value and intraoperative haemodynamic changes during OPACB, we did not include patients with E/e' between 8 and 15 to avoid the introduction of additional confounding factor, because E/e' between 8 and 15 shows poor correlation with the LV filling pressure and requires other information.[9] However, in extension of our results, to validate the prognostic importance of this easily measured echocardiographic indicator of diastolic function, as in studies with patients having myocardial infarction,[11] [12] further studies encompassing all range of E/e' value with larger number of sample size is required.

Limitation of this study is as follows. First, during grafting, haemodynamic compromise also occurs as a consequence of ischaemic insult to the myocardium elicited by transient coronary haemostasis. However, the contribution of an ischaemic insult to the haemodynamic compromise is minor and difficult to assess separately.[1] [2] [5] Considering the use of intracoronary shunt in all patients during LAD grafting and that the number of grafts performed, the total graft reconstruction time and postoperative CK-MB concentration were similar between the groups, this effect should have minimal influence on our results.

In conclusion, despite well-preserved LVEF ($\geq50\%$), patients with elevated E/e' value (>15) were more prone to develop a significant reduction in CI and Sv_{O_2} during grafting that did not return to baseline level after completion of grafting. These patients also required significantly larger amount of NE to maintain the MAP during the surgery, and longer ventilation time and length of stay in the ICU.

Funding

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