—Original—

Repair of an Infrarenal Abdominal Aortic Aneurysm is Associated with Persistent Left Ventricular Diastolic Dysfunction

Hiroyasu Bitoh[1,2], Kazuhiro Nakanishi[1], Shinhiro Takeda[1], Chol Kim[1], Masaki Mori[1] and Atsuhiro Sakamoto[1]

[1]Department of Anesthesia and Pain Control, Graduate School of Medicine, Nippon Medical School
[2]Department of Anesthesiology, Nippon Medical School Musashi Kosugi Hospital

Abstract

Background: Left ventricular (LV) diastolic function has received much attention recently. However, few studies have evaluated LV diastolic function in the perioperative period. The aim of this study was to elucidate perioperative changes in diastolic function using tissue Doppler imaging (TDI) in patients undergoing repair of an infrarenal abdominal aortic aneurysm (AAA).

Methods: Eight patients undergoing repair of an infrarenal AAA were studied prospectively using transesophageal echocardiography. Doppler echocardiographic examinations were performed before the surgical procedure (T1), immediately before aortic unclamping (T2), 30 minutes after aortic unclamping (T3), and at the end of surgery (T4).

Results: Pulmonary edema developed in two patients on postoperative day 1. These two patients had the lowest early diastolic mitral annular velocity (Ea) of the study group at the end of surgery. The ratio of the peak velocity of early mitral inflow (E) to the peak velocity of atrial inflow was significantly decreased at T3 and T4. The systolic ejection velocity was significantly decreased at T3, but returned to the baseline value at T4. The Ea was significantly decreased at T3 and T4. The E/Ea ratio showed a progressive rise and was significantly increased at T3 and T4.

Conclusions: In patients undergoing repair of an infrarenal AAA, the Ea derived using TDI decreases at T3 and is still reduced at T4. The E/Ea ratio, which is used to estimate LV filling pressures, is significantly increased at T3 and T4. Further research is required to confirm the development of diastolic dysfunction and determine its possible association with increased postoperative morbidity and mortality.

(J Nippon Med Sch 2007; 74: 393–401)

Key words: left ventricular diastolic function, tissue Doppler imaging, infrarenal abdominal aortic aneurysm repair, early diastolic mitral annular velocity

Correspondence to Kazuhiro Nakanishi, Department of Anesthesiology, Nippon Medical School, 1–1–5 Sendagi, Bunkyo-ku, Tokyo 113–8603, Japan
E-mail: n-kazu@nms.ac.jp
Journal Website (http://www.nms.ac.jp/jnms/)

Introduction

Left ventricular (LV) diastolic function has received much attention recently. It plays an important role in determining LV filling pressures and symptoms of pulmonary congestion in patients with or without decreased LV ejection fraction (EF)[1]. Nearly half of patients with congestive heart failure (CHF) have diastolic dysfunction despite a normal EF[2], suggesting that the LV systolic and diastolic functions may be uncoupled[3]. In contrast to systolic dysfunction, the positive response to a fluid challenge is limited as a result of decreased compliance, sensitivity to increased afterload is lower, and compensatory neuroendocrine activation is less marked[4]. Recently, coupled to the growing prevalence of CHF and the elderly population is the dramatic increase in the number of surgical procedures[5]. In addition, the elderly have a high prevalence of CHF with preserved systolic function[6]. These features may be important in the perioperative setting because of the poor tolerance to volume overload, a common perioperative event. However, few studies have evaluated alterations in myocardial diastolic function in the perioperative period because its importance and characteristics have only recently become apparent.

The operative risk for conventional open repair of nonruptured infrarenal abdominal aortic aneurysm (AAA) has steadily decreased over the past several decades[7]. Nevertheless, population-based studies suggest that the mortality rate for open AAA repair is still nearly 7% in many communities[7]. Cardiac complications are the major cause of death after conventional AAA repair, accounting for up to 60% of all deaths[8–10]. Adverse cardiac events have been attributed to excessive stress on the myocardium caused by the combined effects of anesthesia; surgical procedures, such as aortic clamping and unclamping; operative blood loss; inadequate volume management; and associated hemodynamic and metabolic changes[11,12]. In particular, the hemodynamic alterations that occur during and after infrarenal aortic clamping are often very severe. However, no clinical reports have described the effects of these manipulations on myocardial diastolic function in patients undergoing repair of an infrarenal AAA.

Tissue Doppler imaging (TDI) is a new ultrasound modality that records systolic and diastolic velocities within the myocardium[13–15] and at the corners of the mitral annulus[16,17]. The early diastolic mitral annular velocity (Ea) has been used to evaluate LV relaxation[16–18] and predict LV filling pressure in several different populations[18–21]. Such clinical observations suggest that Ea is less load-dependent than conventional Doppler variables[16,17]. The systolic mitral annular ejection velocity (Sa) is the clinical variable most suitable as a surrogate for LVEF, because of its accuracy and reproducibility[22]. The aim of this study was to elucidate perioperative alterations in systolic and diastolic function on TDI during and after clamping of the aorta in patients undergoing repair of an infrarenal AAA.

Materials and Methods

Patient Population

This was a prospective study of consecutive patients undergoing repair of an infrarenal AAA with transesophageal echocardiography (TEE) monitoring from April 2005 through April 2006. The study was approved by the Committee on Human Subjects of Nippon Medical School, and all participants provided informed consent. All patients underwent a comprehensive preoperative examination by cardiologists. This examination had a fixed protocol consisting of a physical examination, standard 12-lead electrocardiography, and dobutamine stress echocardiography (DSE). The exclusion criteria were: esophageal or gastric disease, cervical spine instability, pulmonary insufficiency, lack of sinus rhythm, right or left bundle branch block, and significant mitral valve disease. Patients were excluded if they had cardiac dysfunction (defined as LVEF <40%), symptomatic CHF, significant segmental wall motion abnormalities before DSE, or new wall motion abnormalities detected on DSE.

Operative Procedures

All patients underwent the same anesthetic procedure. General anesthesia was induced with fentanyl (5 μg・kg^{-1}), propofol (2.5 mg・kg^{-1}), and vecuronium (0.1 mg・kg^{-1}). An endotracheal tube was inserted and attached to a ventilator; the tidal volume and ventilation rate were adjusted to maintain normocapnia (arterial carbon dioxide tension, 35～40 mmHg). Anesthesia was maintained with sevoflurane in a mixture of oxygen/air, and muscle relaxation was maintained with a continuous infusion of vecuronium (0.1 mg・kg^{-1}・min^{-1}). Standard intraoperative monitoring included a radial artery cannula to continuously record blood pressure, central venous pressure, and heart rate, and a I-II-V5/6 ST-segment electrocardiogram monitor. End-tidal carbon dioxide and oxygen saturations were monitored to assess the adequacy of ventilation, and arterial blood gas was analyzed to assess acid-base balance. Blood losses were recorded and replaced with adequate amounts of crystalloid and colloid solutions. Monitoring was continued throughout the period that the patient remained in the high-dependency unit after the operation.

The infrarenal AAA was repaired through xiphopubic laparotomy. The aorta and iliac vessels were prepared, isolated, and clamped below the renal arteries after systemic heparinization and were then reconstructed with either a tube or bifurcated Dacron graft, depending on the extent of the aneurysm. All procedures were performed by the same surgeon.

Echocardiographic Studies

All echocardiographic studies were performed with the patient in the supine position. Complete TEE was performed with a Sonos 5500 ultrasound system (Hewlett-Packard, Andover, MA, USA) equipped with TDI capability. Echocardiographic examinations were performed before the surgical procedure (T1), immediately before aortic unclamping (T2), 30 minutes after aortic unclamping (T3), and at the end of surgery (T4). Data were obtained using standard views and techniques, and images were stored digitally on magnetic optical disks (Hewlett-Packard) for later playback and analysis. Echocardiographic measurements were performed off-line by an observer who had no knowledge of the clinical data or other hemodynamic measurements. All measurements were made during the end-expiratory phase over five consecutive cardiac cycles.

American Society of Echocardiography recommendations[23] were followed to determine LV volumes and EF. The mitral inflow was recorded using pulsed Doppler echocardiography, with the sample volume placed at the mitral valve tips in the midesophageal 4-chamber view. The Doppler beam was aligned to produce the narrowest possible angle between the beam and the blood flow vector. The mitral inflow velocity was traced, and the peak velocity of early (E) and late (A) filling, and deceleration time (DT) of the E wave velocity were derived. The E/A ratio and DT are the most widely used indices and are recommended in the latest guidelines for diagnosing diastolic dysfunction[24,25]. The midesophageal 4-chamber view was used to determine the pulmonary venous flow, with the sample volume placed in the left upper pulmonary vein 1 to 2 cm proximal to its entrance into the left atrium. The peak systolic and diastolic velocities were recorded, and peak systolic/diastolic (S/D) velocity ratio was computed.

Velocities of the mitral annulus were recorded using a TDI program, with a 5-mm sample volume placed at the septal and lateral corners of the mitral annulus and averaged as previously described[18]. The following measurements were made from the DTI recordings: systolic mitral annular ejection velocity (Sa), systolic mitral annular velocity during isovolumic contraction, Ea and late diastolic mitral annular velocity (Aa). All pulsed Doppler readings were recorded for 5 to 10 cardiac cycles at a horizontal sweep speed of 100 mm/s.

Statistical Analysis

All values are reported as mean (± SD) for variables with a normal distribution and as median (interquartile range) for variables without a normal distribution. Data without a normal distribution were subjected to log-normalization before parametric analyses were performed. *Post hoc* analysis was

performed of multiple comparison testing to determine differences from control values (Dunnett). A correlation analysis (Pearson) was used to test for significant linear relationships between changes in myocardial velocities and the aortic occlusion time. Differences with $P<0.05$ were considered as statistically significant.

Results

Eight patients were enrolled in the study. The clinical characteristics and perioperative data are summarized in **Table 1**. There were 6 men and 2 women, aged 60 to 79 years (mean, 68 ± 8 years). None of the patients undergoing repair of traditional/open AAA required suprarenal clamping. There were no in-hospital deaths. Pulmonary edema developed in two patients on postoperative day 1. It is noteworthy that these patient had the lowest Ea of the study group at the end of surgery (5.3 and 6.5 cm/s), and that the E/Ea ratio in these patients increased to 11.8 and 11.3 cm/s. One of these patients required noninvasive positive pressure ventilator support. No patients had ST-segment changes on electrocardiography at any time during the study.

The hemodynamic and echocardiographic data are shown in **Table 2**. Heart rate was significantly increased at T3 and T4. Systolic blood pressure, diastolic blood pressure, left ventricular end diastolic volume index (LVEDVI), and stroke volume were slightly but not significantly lower at T3. The LVEF was significantly decreased at T3 but returned to the baseline value at T4. The changes in LVEF are shown in **Figure 1**. Compared with the baseline value, the LVEF decreased by 6.8% at T3 but inversely to increase at T4. Before surgery, all patients demonstrated a normal pattern of mitral inflow with an E/A ratio >1 and DT <200 ms, and a normal pulmonary venous flow S/D ratio of >1. The mitral E/A ratio and the pulmonary venous S/D ratio were significantly decreased at T3 and T4. Six patients had a decrease in E/A ratio to <1 at T3, and 2 of these patients had an increase in DT to > 200 ms with acute mild-to-moderate diastolic dysfunction. Four patients had a persistent decrease in E/A ratio to <1 at T4, and one of these patients had an increase in DT to >200 ms. This patient had a decrease in S/D ratio to <1 at T3 and T4. The Sa was significantly decreased at T3 and returned to the baseline value at T4. The Ea was significantly decreased at T3 and T4. All patients showed acute and consistent decreases in Ea after aortic unclamping (**Fig. 2**). Compared with the baseline value, the Ea decreased by 2.0 cm/s at T3 and by 1.9 cm/s at T4. The E/Ea ratio showed a progressive rise and was significantly increased at T3 and T4. There were no significant correlations between changes in myocardial velocities and the aortic occlusion time.

Table 1 Clinical Characteristics and Perioperative Data

Baseline characteristics	
Patients (n)	8
Age (yrs)	68 ± 8
Weight (kg)	59.5 ± 9.7
Height (cm)	163 ± 5
Men/women (n)	6/2
Hypertension (n)	8
Diabetes mellitus (n)	5
Hyperlipidemia (n)	7
Smoking history (n)	7
Preoperative medications	
ACE inhibitors (n)	7
Calcium channel blocker (n)	6
Diuretics (n)	3
Oral hypoglycemic (n)	3
Beta-blocker (n)	7
Operative details	
Operation time (min)	288 ± 60
Aortic cross-clamping time (min)	75 ± 12
Intraoperative blood losses (mL)	350 ± 80
Fluides administered (mL/kg/hr)	12.8 ± 3.5
Colloids/crystalloids ratio	0.30 ± 0.08
Postoperative coutcomes	
Pulmonary edema (n)	2

Values are presented as number and means ± SD.
ACE = angiotensin-converting enzyme.

Discussion

The Ea and Sa derived from TDI data were significantly decreased at T3 in patients undergoing repair of an infrarenal AAA. The Sa returned to the preoperative value at T4, whereas the Ea remained

Table 2 Hemodynamic and Echocardiographic Variables

Variables	T1	T2	T3	T4
Heart rate (beats/min)	60.0 (7.5)	65.0 (8.5)	68.0 (10.0) **	68.0 (15.5) ***
Systolic blood pressure (mm Hg)	104.0 (11.5)	106.0 (12.5)	99.0 (20.0)	105.0 (12.0)
Diastolic blood pressure (mmHg)	51.5 (10.5)	44.5 (12.0)	45.5 (10.5)	50.0 (7.5)
Central venous pressure (mmHg)	8.0 (4.0)	9.0 (2.0)	7.0 (5.0)	8.0 (3.5)
LVEDVI (mL/m^2)	62.5 (10.2)	62.8 (15.1)	61.3 (13.7)	59.1 (6.2)
Stroke volume index (mL/m^2)	39.8 (7.0)	38.3 (12.0)	35.4 (12.6)	40.2 (9.5)
Left ventricular ejection fraction (%)	63.7 (7.9)	61.0 (8.4)	57.9 (14.8) **	68.0 (7.3)
Mitral E/A ratio	1.43 (0.52)	1.32 (0.54)	0.98 (0.48) **	1.08 (0.38) **
DT (ms)	158 (27)	165 (67)	140 (37)	146 (38)
Pulmonary venous S/D ratio	1.51 (0.35)	1.55 (0.31)	1.38 (0.53) *	1.33 (0.45) *
Average Sa (cm/s)	7.7 (1.9)	7.4 (1.6)	6.6 (1.3) **	7.1 (0.9)
Average Ea (cm/s)	10.5 (2.3)	9.5 (1.9)	7.8 (1.2) ***	7.7 (1.2) ***
Average Aa (cm/s)	8.3 (2.7)	7.1 (2.7)	8.5 (3.5)	8.2 (3.2) *
E/Ea ratio	7.0 (2.6)	8.0 (2.0)	8.2 (2.0) *	9.1.(2.5) **

* $p<0.05$, ** $p<0.01$, *** $p<0.001$, vs. T1 (repeated measure one-way ANOVA followed by Fisher's PLSD for multicomparison).
Values are median (interquartile range).
LVEDVI = left ventricular end diastolic volume index; E = peak velocity of early mitral inflow; A = peak velocity of atrial mitral flow; DT = deceleration time of early mitral flow; S = peak systolic pulmonary venous flow velocity; D = peak diastolic pulmonary venous flow velocity; Sa = systolic mitral annular ejection velocity; Ea = early diastolic mitral annular velocity; Aa = late diastolic mitral annular velocity; T1 = before surgical procedure; T2 = immediately before aortic unclamping; T3 = 30 minutes after aortic unclamping; T4 = at the end of surgery.

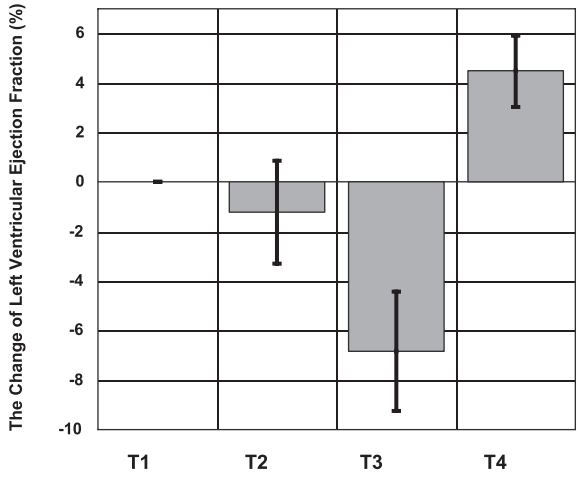

Fig. 1 The changes in the LVEF from baseline values at each point of different perioperative times. Values are expressed as means ± SEM. The change in LVEF = LVEF [Tn] − LVEF [T1], n = 2, 3, and 4. Compared with the baseline value, the LVEF decreased by 6.8% at T3 but tended to increase at T4.

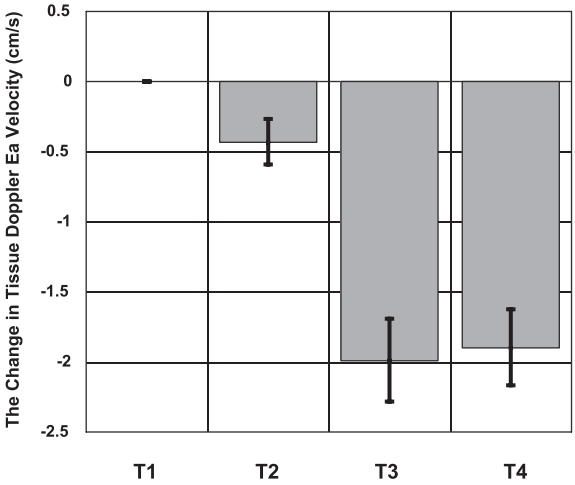

Fig. 2 The changes of tissue Doppler Ea from baseline values at each point of different perioperative times. Values are expressed as means ± SEM. The change of Ea = Ea [Tn] − Ea [T1], n = 2, 3, and 4. Compared with the baseline value, the Ea decreased by 2.0 cm/s at T3 and by 1.9 cm/s at T4.

consistently reduced. The E/Ea ratio was significantly increased at T3 and at T4.

Diastolic dysfunction plays an important role in determining LV filling pressures and symptoms of pulmonary congestion in patients with and without a low LVEF[1]. It is related to a variable combination of abnormal myocardial relaxation and reduced ventricular compliance[4]. In contrast to systolic

dysfunction, the positive response to a fluid challenge is limited as a result of decreased compliance, sensitivity to increased afterload is lower, and compensatory neuroendocrine activation is less marked[4]. Patients may be faced with numerous triggers of acute CHF in the perioperative period, including the withdrawal of drugs to treat CHF, hypertension, inadequate volume management, severe bleeding, tachyarrhythmias, and myocardial ischemia[4]. Diastolic dysfunction may be the primary mechanism responsible for acute perioperative CHF, regardless of the presence or severity of systolic dysfunction. The elderly have a particularly high prevalence of CHF with preserved systolic function[6], which may be important because of their poor tolerance to volume overload, a common perioperative event. However, previous studies did not evaluate diastolic dysfunction because its importance and characteristics did not become apparent until recently.

Doppler echocardiography has become the noninvasive technique of choice for the evaluation of LV relaxation[16–18] and filling pressures[18–21]. Although mitral inflow and pulmonary venous flow velocities can often provide valuable information, they have limitations. They are most useful in patients with a low EF, but are much less reliable when the EF is normal (>50%)[26,27]. The strong influence of loading conditions on these velocities also precludes their application to drawing inferences about LV relaxation in situations where filling pressures are increased[28]. The new ultrasound modality of TDI records systolic and diastolic velocities within the myocardium[13–15] and at the corners of the mitral annulus[16,17]. The velocity of annular motion reflects shortening and lengthening of the myocardial fibers in a longitudinal plane. The Ea has recently been demonstrated to decline progressively with age, and to be reduced in pathologic LV hypertrophy[16] and restrictive cardiomyopathy[17]. It is therefore commonly believed that Ea behaves as a preload-independent index of LV relaxation. In the present study, the Ea was significantly decreased at T3 and was consistently lower at T4 than the baseline value. The ratio between the early mitral inflow measured by conventional Doppler and the displacement of the mitral annulus measured by TDI was also significantly increased at T3 and at T4. This ratio has been shown to accurately reflect pulmonary capillary wedge pressure in cardiac patients[18,27]. The LVEF and Sa were significantly decreased at T3, but returned to the baseline value at T4. These findings indicate that left ventricular systolic and diastolic function were impaired after aortic unclamping in patients undergoing repair of an infrarenal AAA, and that diastolic function remained consistently impaired despite the complete recovery of systolic function at the end of surgery.

Why the reduced diastolic function persisted for a longer time after aortic unclamping is unknown. Diastolic dysfunction frequently occurs in the absence of systolic dysfunction. It has also been reported that LV relaxation and filling is sensitive to ischemia[29] and that Ea decreases before LVEF becomes abnormal[30]. LV diastolic dysfunction may be a primary abnormality if the EF is preserved. In patients with preoperative LV diastolic dysfunction, biventricular filling patterns are impaired initially but return to preoperative status 6 months after coronary artery bypass grafting[31]. Diastolic function may increase the susceptibility to myocardial injury.

The pathophysiologic disturbances that occur during aortic clamping and unclamping in repair of an infrarenal AAA may be closely related to the hemodynamic alterations described and the high complication rates[32]. During aortic cross-clamping, the blood volume redistribution and increase in preload and afterload require appropriate adjustments in myocardial contractility and coronary blood flow[32]. Many observers have reported no increase in cardiac output in response to the increased preload, induced volume redistribution and increased afterload during infrarenal aortic cross-clamping[33,34], suggesting a lack of the required increase in myocardial contractility. Observations with nuclear ventriculography have shown depressed myocardial performance and systolic function during cross-clamping of the abdominal aorta[35]; the authors interpreted this observation as being due to myocardial ischemia. In the present study, we excluded patients with CHF and coronary artery disease who had significant segmental wall

motion abnormalities before DSE or had new wall-motion abnormalities detected on DSE; no patients had ST-segment changes on electrocardiography throughout the study. The cardiovascular response to infrarenal aortic cross-clamping is less significant than that to high aortic cross-clamping. Therefore, we did not find any alterations in LVEF or Sa, which is a load-independent index, during cross-clamping.

Unclamping of the aorta is consistently associated with substantial decreases in vascular resistance and arterial blood pressure. The most likely reasons for the unclamping hypotension include central hypovolemia caused by the pooling of blood in reperfused tissues distal to the aortic occlusion, hypoxia-mediated vasodilatation with a subsequent increase in vascular (venous) capacity in the extremities below the occlusion, and accumulation of vasoactive and myocardial-depressant metabolites[32]. Furthermore, ishemia-reperfusion injury following aortic cross-clamping leads to activation of cytokines and inflammatory pathways[36,37], resulting in injury to distant organs, such as the heart, lungs and kidneys, which may dysfunction and eventually fail[37]. The release of inflammatory cytokines and the expression of inducible nitric oxide synthase have recently been suggested to play important roles in mediating cardiac dysfunction by direct actions on myocytes[37]. We have reported that esophagectomy is associated with transient depression of myocardial function. Interleukin (IL)-6 may contribute to this postoperative myocardial dysfunction[38]. Finkel et al. have clinically confirmed that IL-6 levels in pulmonary venous effluent markedly increase immediately after aortocoronary bypass surgery; they have also shown experimentally that the same concentrations of IL-6 can cause reversible myocardial depression in the human heart[39]. Furthermore, increasing evidence suggests that both systolic and diastolic functions are affected in severe sepsis and septic shock[40]. An excessive and prolonged increase in the levels of circulating myocardial-depressant metabolites may, therefore, be associated with a protracted deterioration of left ventricular diastolic function after repair of an infrarenal AAA. anticytokine therapy may attenuate the elevated release of myocardial-depressant metabolites, leading to improved myocardial performance. Further studies are required to clarify the association between increased cytokine release and postoperative diastolic dysfunction.

In the present study, we found no significant correlation between changes in myocardial velocities and the aortic occlusion time. The duration of aortic cross-clamping may be correlated with the amount of circulating myocardial-depressant factors released from ischemic tissues. However, we did not measure circulating myocardial depressants. In AAA repair, ischemia and reperfusion can occur for several reasons, such as direct ligation of vessels, mesenteric traction, bowel hypothermia, and the release of vasoactive mediators from the vascular endothelium within the gastrointestinal tract. Therefore, we cannot find an association between decreased myocardial function and the duration of aortic clamping.

In the present study, we found prolonged postoperative diastolic dysfunction with preserved systolic function using TDI in all patients undergoing repair of an infrarenal AAA. postoperative pulmonary edema without systolic dysfunction developed in two patients, one of whom required postoperative ventilatory support. It is noteworthy that these patients had the lowest Ea of the study group at T4 (5.3 and 6.5 cm/s) and that the E/Ea ratio in these patients increased to 11.8 and 11.3 cm/s, Previous echocardiographic studies have suggested that diastolic dysfunction contributes to perioperative hemodynamic instability and adverse outcomes following cardiac surgery[41]. Our sample size was not large enough to document clinical outcome variables, but our results suggest that the development of diastolic dysfunction may be closely related to postoperative cardiac complications, regardless of the presence or severity of systolic dysfunction. Furthermore, the prolonged diastolic dysfunction identified by TDI may predict adverse outcomes.

Previous studies have shown that CHF is an important risk factor, but the magnitude of the risk may be underappreciated[42]. Recent studies have shown that among patients 65 years or older undergoing major noncardiac surgery, those with

CHF have higher morbidity and mortality rates despite advances in perioperative care, whereas those with coronary artery disease without CHF have a similar mortality rate to the general population[42]. Nearly half of patients with CHF have diastolic dysfunction despite a normal EF[2]. There are few guidelines for clinicians on how to treat patients with CHF during the perioperative period. Our results may explain why patients with CHF who are 65 years or older and undergoing major noncardiac surgery have poor outcomes. Attention should be focused on the treatment of patients with CHF without systolic dysfunction through the perioperative period. Further research is required to confirm the development of diastolic dysfunction and determine its possible association with increased postoperative morbidity and mortality.

In patients undergoing repair of an infrarenal AAA, the Ea derived from TDI data decreased after aortic unclamping and remained consistently reduced at the end of surgery. The E/Ea ratio, which is used to estimate LV filling pressures, was significantly increased after aortic unclamping and at the end of surgery.

References

1. Zile MR, Brutsaert DL: New concepts in diastolic dysfunction and diastolic heart failure: Part I. Diagnosis, prognosis, and measurements of diastolic function. Circulation 2002; 05: 1387–1393.
2. Vasan RS, Larson MG, Benjamin EJ, Evans JC, Reiss CK, Levy D: Congestive heart failure in subjects with normal versus reduced left ventricular ejection fraction. Prevalence and mortality in a population-based cohort. J Am Coll Cardiol 1999; 33: 1948–1955.
3. Bella JN, Palmieri V, Liu JE, et al.: Relationship between left ventricular diastolic relaxation and systolic function in hypertension. The Hypertension Genetic Epidemiology Network (HyperGEN) Study. Hypertension 2001; 38: 424–428.
4. Toller WG, Metzler H: Acute perioperative heart failure. Current Opinion in Anaesthesiology 2005; 18: 129–135.
5. National Center for Health Statistics: Impatient Surgery. Available at: http://www.cdc.gov/nchs/fastats/insurg.htm. Accessed April 1, 2004.
6. Hogg K, Swedberg K, McMurray J: Heart failure with preserved ledt ventricular systolic function; epidemiology, clinical characteristics, and prognosis. J Am Coll Cardiol 2004; 43: 317–327.
7. Hertzer NR: Current status of endovascular repair of infrarenal abdominal aortic aneurysms in the context of 50 years of conventional repair. Ann N Y Acad Sci 2006; 1085: 175–186.
8. Johnston KW: Multicenter prospective study of nonruptured abdominal aortic aneurysm. Part II. Variables predicting morbidity and mortality. J Vasc Surg 1989; 9: 437–447.
9. Amundsen S, Trippestad A, Viste A, Soreide O: Abdominal aortic aneurysms—a national multicentre study. Eur J Vasc Surg 1987; 1: 239–243.
10. Diehl JT, Cali RF, Hertzer NR, Beven EG: Complications of abdominal aortic reconstruction. An analysis of perioperative risk factors in 557 patients. Ann Surg 1983; 197: 49–56.
11. Falk JL, Rackow EC, Blumenberg R, Gelfand M, Fein IA: Hemodynamic and metabolic effects of abdominal aortic crossclamping. Am J Surg 1981; 142: 174–177.
12. Hudson RJ, Wurm WH, O'Donnell TF, et al.: Hemodynamics and prostacyclin release in the early phases of aortic surgery: comparison of transabdominal and retroperitoneal approaches. J Vasc Surg 1988; 7: 190–198.
13. Sutherland GR, Stewart MJ, Groundstroem KWE, et al.: Color Doppler myocardial imaging: a new technique for the assessment of myocardial function. J Am Soc Echocardiogr 1994; 23: 1441–1458.
14. Miyatake K, Yamagishi M, Tanaka N, et al.: New method for evaluating left ventricular wall motion by color-coded tissue Doppler imaging: in vitro and in vivo studies. J Am Coll Cardiol 1995; 25: 717–724.
15. Uematsu M, Miyatake K, Tanaka N, et al.: Myocardial velocity gradient as a new indicator of regional left ventricular contraction: detection by a two-dimensional tissue Doppler imaging technique. J Am Coll Cardiol 1995; 26: 217–223.
16. Rodriguez L, Garcia M, Ares M, Griffin BP, Nakatani S, Thomas JD: Assessment of mitral annular dynamics during diastole by Doppler tissue imaging: comparison with mitral Doppler inflow in subjects without heart disease and in patients with left ventricular hypertrophy. Am Heart J 1996; 131: 982–987.
17. Garcia MG, Rodriguez L, Ares M, Griffin BP, Thomas JD, Klein AL: Differentiation of constrictive pericarditis from restrictive cardiomyopathy: assessment of left ventricular diastolic velocities in longitudinal axis by Doppler tissue imaging. J Am Coll Cardiol 1996; 27: 108–114.
18. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quinones MA: Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol 1997; 30: 1527–1533.
19. Nagueh SF, Mikati I, Kopelen HA, Middleton KJ, Quiñones MA, Zoghbi WA: Doppler estimation of left ventricular filling pressure in sinus tachycardia. A new application of tissue Doppler imaging. Circulation 1998; 98: 1644–1650.
20. Nagueh SF, Lakkis NM, Middleton KJ, Spencer WH III, Zoghbi WA, Quiñones MA: Doppler estimation of left ventricular filling pressures in patients with hypertrophic cardiomyopathy. Circulation 1999; 99: 254–261.
21. Sohn DW, Kim YJ, Kim HC, Chun HG, Park YB, Choi YS: Evaluation of left ventricular diastolic function when mitral E and A waves are completely fused:

role of assessing mitral annulus velocity. J Am Soc Echocardiogr 1999; 12: 203–208.
22. Ruan Q, Nagueh SF: Usefulness of isovolumic and systolic ejection signals by tissue Doppler for the assessment of left ventricular systolic function in ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 2006; 97: 872–875.
23. Schiller NB, Shah PM, Crawford M, et al.: Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989; 2: 358–367.
24. European Study Group on Diastolic Heart Failure: How to diagnose diastolic heart failure. Eur Heart J 1998; 19: 990–1003.
25. Ceconi M, Manfrin M, Zanoli R, et al.: Doppler echocardiographic evaluation of left ventricular end-diastolic pressure in patients with coronary artery disease. J Am Soc Echocardiogr 1996; 9: 241–250.
26. Yamamoto K, Nishimura RA, Chaliki HP, Appleton CP, Holmes DR Jr, Redfield MM: Determination of left ventricular filling pressure by Doppler echocardiography in patients with coronary artery disease: clinical role of left ventricular systolic function. J Am Coll Cardiol 1997; 30: 1527–1533.
27. Rivas-Gotz C, Manolios M, Thohan V, Nagueh SF: Impact of left ventricular filling pressures using tissue Doppler and flow propagation velocity. Am J Cardiol 2003; 91: 780–784.
28. Thomas JD, Weyman AE: Echo Doppler evaluation of left ventricular diastolic function: physics and physiology. Circulation 1991; 84: 977–990.
29. Najos-Valencia O, Cain P, Case C, et al.: Determinants of tissue Doppler measures of regional diastolic function during dobutamine stress echocardiography. Am Heart J 2002; 144: 516–523.
30. Nagueh SF: Search for non-invasive load-independent indices of left ventricular relaxation. Clin Sci (Lond) 2003; 105: 395–397.
31. Shi Y, Denault AY, Couture P, et al.: Biventricular diastolic filling patterns after coronary artery bypass graft surgery. J Thorac Cardiovasc Surg 2006; 131: 1080–1086.
32. Simon G: The pathophysiology of aortic cross-clamping and unclamping. Anesthesiology 1995; 82: 1026–1057.
33. Grindlinger GA, Vegas AM, Manny J, Bush HL, Mannick JA, Hechtman HB: Volume loading and vasodilators in abdominal aortic aneurysmectomy. Am J Surg 1980; 139: 480–486.
34. Hjalmarson A: Myocardial metabolic changes related to ventricular fibrillation. Cardiology 1980; 65: 226–247.
35. Kalman PG, Wellwood MR, Weisel RD, et al.: Cardiac dysfunction during abdominal aortic operation: The limitation of pulmonary wedge pressure. J Vasc Surg 1986; 3: 773–781.
36. Norwood MGA, Bown MJ, Sayers RD: Ischaemia-reperfusion injury and regional inflammatory responses in abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2004; 28: 234–245.
37. Bown MJ, Nicholson ML, Bell PRF, Sayers RD: Cytokines and inflammatory pathways in the pathogenesis of multiple organ failure following abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2002; 22: 485–495.
38. Nakanishi K, Takeda S, Terajima K, et al.: Myocardial dysfunction associated with proinflammatory cytokines aftere esophageal resection. Anesth Anakg 2000; 91: 270–275.
39. Finkel MS, Hoffman RA, Shen L, et al.: Interleukin-6 as a mediator of stunned myocardium. Am J Cardiol 1993; 71: 1231–1232.
40. Rabuel C, Mebazaa A: Septic shock: a heart story since the 1960s. Intensive Care Med 2006; 32: 799–807.
41. Bernard F, Denault A, Babin D, et al.: Diastolic dysfunction is predictive of difficult weaning from cardiopulmonary bypass. Anesth Analg 2001; 922: 291–298.
42. Hernandez AF, Whellan DJ, Stroud S, Sun JL, O'Connor CM, Jollis JG: Outcomes in heart failure patients after major noncardiac surgery. J Am Coll Cardiol 2004; 447: 1446–1453.

(Received, June 6, 2007)
(Accepted, September 13, 2007)