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Noninvasive assessment of left ventricular pressure–area relationship using transesophageal echocardiography and tonometry during cardiac and abdominal aortic surgery

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Abstract
Purpose. The purpose of this study was to noninvasively evaluate intraoperative left ventricular (LV) performance by an online pressure–area relationship using transesophageal echocardiography (TEE) and tonometry.
Methods. In study 1, LV pressure with a micromanometer catheter, LV cross-sectional area with TEE, direct radial pressure, and tonometric arterial pressure were simultaneously recorded in 5 patients (10 measurements) undergoing cardiac surgery. End-systolic elastance (E′es) was determined from pressure–area loops during inferior vena caval (IVC) occlusion. In study 2, in 16 patients undergoing repair of abdominal aortic aneurysm, LV performance (E′es; effective arterial load, E′a, and LV end-diastolic area, LV-EDA) was examined by noninvasive assessment of pressure–area loops using TEE and tonometry at aortic cross-clamping and unclamping.
Results. E′es by tonometric arterial pressure closely correlated with E′es by LV pressure ($r = 0.92$) in study 1. E′es at aortic clamping were not significantly different from those at unclamping. The clamping increased LV-EDA and E′a by approximately 13% and 44%, and the unclamping significantly decreased by 9% and 22%, respectively.
Conclusion. Our results demonstrated that online tonometric arterial pressure and LV area measured by automated border detection (ABD) of TEE might be used to calculate E′es to estimate LV contractility and allow the estimation of LV performance during aortic clamping and unclamping.

Key words Left ventricular contractility · Noninvasive monitor · Preload · Afterload

Introduction

Analysis of the left ventricular (LV) pressure–volume relationship is useful for characterizing the determinants of ventricular performance, including preload, afterload, and contractility [1,2]. The clinical application of such relationship has been, however, limited due to technical difficulties in acquiring LV pressure and volume data throughout the cardiac cycle. Standard imaging techniques require frame-by-frame manual analysis, whereas the use of invasive conductance catheter and sonomicrometry techniques is not well suited for clinical settings [3–6]. Transesophageal echocardiography (TEE) has been widely used for intraoperative monitoring of LV function, and automated border detection (ABD) data have been demonstrated using this approach in humans [7–10]. Automated echocardiographic measures of the LV cavity area have been shown to closely correlate with changes in LV volume [7–14]. Based on the assumption that LV pressure is similar to that of the aorta and femoral artery during the ejection phase, Gorcsan et al. [9] constructed LV pressure–area loops using automated echocardiographic LV area and recording femoral arterial pressure, and demonstrated a good correlation between end-systolic pressure–area relationship using LV pressure and femoral arterial pressure.

Arterial pressure waveforms can be measured noninvasively and continuously by the use of arterial tonometry [15–17]. Arterial tonometry provides accurate and reliable real-time monitoring of blood pressure even during induced hypotension [16]. The purpose of this study was to evaluate LV performance intraoperatively by online pressure–area relationship using ABD of TEE and arterial tonometry in a manner similar to pressure–volume analysis. Using this method, we evaluated LV performance at aortic clamping and unclamping, which are known to cause rapid changes in hemodynamic variables.

Materials and methods

After obtaining institutional approval and informed consent, two studies were performed.

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Study 1: Correlation between LV pressure–area relationship and radial arterial or tonometric pressure–area relationship

Five patients (two men and three women), aged 70 ± 5 years (range, 63–76), undergoing valve replacement or coronary artery bypass were enrolled in the study. General anesthesia was induced with diazepam 0.2 mg·kg^{-1} and fentanyl 20 μg·kg^{-1}. Endotracheal intubation was facilitated by vecuronium. Anesthesia and muscle relaxation were maintained by fentanyl, diazepam, and vecuronium. Ventilation was controlled with a tidal volume of 10 ml·kg^{-1} at a rate that maintained a stable Pa_{CO_2} at 30–40 mmHg.

Echocardiography was performed using a 5.0-MHz transesophageal transducer (model 21364A; Hewlett-Packard, Andover, MA, USA) and ultrasound system (Sonos 2500, model 2406A; Hewlett-Packard) with ABD capabilities [11]. Transgastric midshort-axis view was used, with the midpapillary muscle level as the anatomic landmark, by positioning the transducer to uniform wall thickness. This plane was selected because of the difficulties encountered in obtaining true long-axis lengths from the transesophageal approach. Previous studies have identified the presence of a linear relationship between cross-sectional area and LV volume [7,9–14]. The threshold for discriminating blood from tissue backscatter characteristics was directly influenced by manual gain settings, and thus overall transmit, time gain compensation, and lateral gain controls were carefully adjusted by visual inspection as a compromise between cavity clutter and wall dropout. A region of interest was manually drawn immediately beyond the LV endocardial border to exclude the right ventricular cavity. The area of pixels within the selected region of interest identified as blood density was calculated from each frame and displayed as a waveform in real time.

Patients were instrumented with 20-G fluid-filled radial arterial catheters, connected to strain-gauge pressure transducers (Baxter, Irvine, CA, USA). A continuous noninvasive tonometric blood pressure monitoring instrument (Jentow; Colin Electronics, Komaki, Japan) was also attached to the extended wrist pressured against the contralateral radial artery. A cuff was wrapped around the brachial artery. The output of tonometric sensor was calibrated by the oscillometric blood pressure measured on the brachial artery every 2.5 min. This device records radial arterial pressure by compressing the radial artery between the radius bone and the pressure sensor. The compressing force was adjusted so as not to narrow the artery but establish a steady contact with the artery through the flat surface of the sensor, with the force level adjust to be equivalent to the intravascular pressure [16]. A median sternotomy was performed, and a micromanometer catheter (Micro-Tip pressure transducer, MPC-500; Miller, Houston, TX, USA) was advanced into the LV through the right upper pulmonary vein.

The analog data of the LV area from ABD and arterial pressure waveforms from the tonometry were captured at a sampling frequency of 200 Hz using an analog-to-digital converter (Mac Lab; Analog Digital Instrument, Castle Hill, Australia). LV pressure and area signals were plotted using customized software and graphics routines, and the pressure–area loops were displayed on a monitor. The pressure signal was plotted after a delay of approximately 30 ms with respect to the area signal to allow for the time required for the automated system to calculate the area from each frame. The duration of the delay was adjusted for each run by aligning the point immediately preceding isovolumic contraction on the pressure waveform with the first occurrence of maximal area.

To construct LV pressure–area loops, LV end-diastolic pressure waveforms, defined as the point just after isovolumic contraction, were advanced to manually align the LV end-diastolic pressure with LV end-diastolic area (EDA), defined as the first occurrence of the maximum area. To construct arterial pressure–area loops, arterial pressure waveforms were advanced to manually align the minimum arterial pressure with LV-EDA just before the beginning of decreased point from the maximum area for each run. End-diastole was selected as the point for alignment because the majority of arterial pressure tracings did not have a clearly defined dicrotic notch to indicate end-systole. Slight adjustments in timing were made by visual inspection of the pressure–area loops to most approximate LV ejection pressure with the systolic pressure–area trajectory bounded by the range of area values. These adjustments were made by adding or subtracting approximately 30-ms increments to the pressure signals. LV contractility was then estimated by the method of Sagawa [1,2] for calculating the end-systolic pressure–volume relationship, or end-systolic elastance, by applying these calculations to the pressure–area loops.

Accordingly, end-systole was defined as the occurrence of the maximal pressure–area point in each pressure–area loop, and the slope of these points from differently loaded beats determined E′es by an iterative linear regression method [13]. Pressure-area elastance values were designated E′es to differentiate the term from the Ees values of end-systolic elastance from the pressure-volume data. E′es was obtained during cardiac surgery, and before and after cardiopulmonary bypass (CPB), using inferior vena caval (IVC) occlusion maneuvers by ten recordings in five patients. Echographic LV area, LV pressure, and radial and tonometric pressure data were recorded at end-expiratory period.

Study 2: LV performance during aortic clamping and unclamping

Studies were performed in 20 consecutive patients (14 men and 6 women), aged 72 ± 6 years (range, 61–83) undergoing repair surgery for infrarenal abdominal aortic aneurysm. Four patients were excluded because of poor-quality transgastric echocardiographic images and poor-quality tonometric blood pressure waveforms. Therefore, the study group consisted of 16 patients: 11 men and 5 women. After securing an intravenous access, epidural block was established to the mid-thoracic level with 1% or 2% mepivacaine. General anesthesia was induced with thiamylal and fentanyl. Endotracheal intubation was facilitated by vecuronium. Anesthesia and muscle relaxation were maintained by nitrous oxide-oxygen, isoflurane, fentanyl and vecuronium. Ventilation was controlled with a tidal volume of $10\,ml \cdot kg^{-1}$ at a rate to maintain Pa_{CO_2} between 30 to 40 mmHg.

Echocardiographic and arterial pressure data were recorded and analyzed using the same methods described for study 1. End-diastolic area (EDA) and end-systolic areas (ESA) were also collected before and after aortic clamping and unclamping. The effective arterial load (E′a) was calculated as [end-systolic pressure $\cdot (EDA - ESA)^{-1}$] [2], and the fractional area change (FAC) was calculated as [$(EDA - ESA) \cdot (EDA)^{-1}$]. E′es was determined as the slope of end-systolic points in the pressure-area loops during aortic cross-clamping and unclamping.

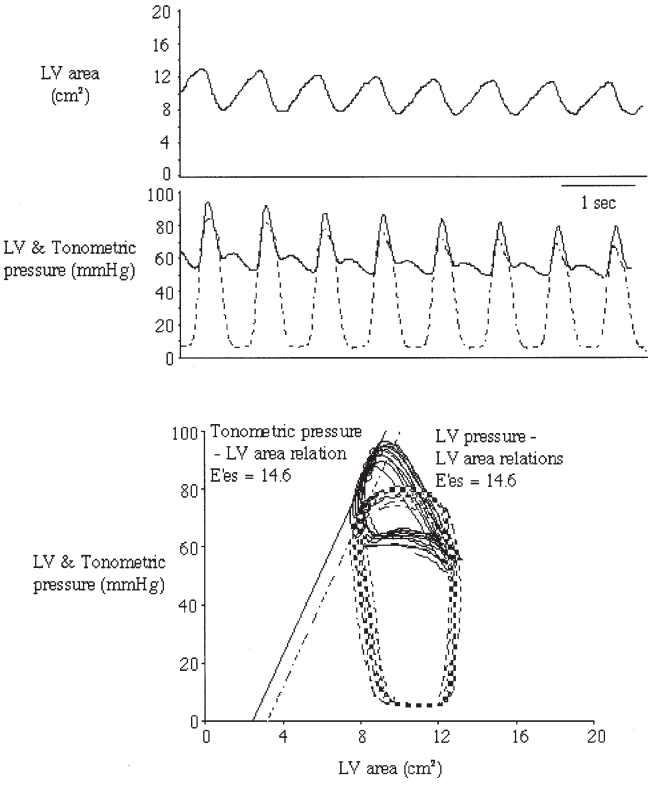

Fig. 1. An example of waveform data during inferior vena caval occlusion and corresponding simultaneous left ventricular (LV) pressure–area loops (*dotted lines*) and tonometric pressure–area loops (*solid lines*)

Statistical analysis

Data were expressed as mean ± standard deviation (SD). To assess the degree to which tonometric arterial blood pressure could be used to estimate E′es with LV pressure, linear regression analysis by the method of least squares was performed because E′es derived from high-fidelity LV pressure was the gold standard to which E′es by the arterial pressure signal was compared. Bias of E′es from the three methods was evaluated using Bland-Altman method. Data obtained after aortic clamping and unclamping were analyzed in relation to the indices measured before clamping and unclamping by using a Wilcoxon signed-rank test. $P < 0.05$ was defined as significant.

Results

Study 1

Ten IVC occlusion maneuvers were performed in the five patients. Figure 1 shows an example of waveform data during IVC occlusion and corresponding simultaneous LV pressure–area loops and tonometric pressure–area loops.

The mean fall in systolic blood pressure by IVC occlusion was 31 ± 11 mmHg for LV pressure, 35 ± 16 mmHg for radial pressure, and 36 ± 16 mmHg for tonometric pressure. The magnitude of the fall in systolic blood pressure was not significantly different among the three measurements. Estimates of E′es by tonometric pressure–area relationship closely correlated to the E′es estimates by LV pressure–area relationship (Fig. 2; $r = 0.92$, SE of the estimate = $4\,mmHg \cdot cm^{-2}$, $y = 0.86x + 1.26$). Estimates of E′es from radial arterial pressure also closely correlated with E′es from LV pressure ($r = 0.90$, SE of the estimate = $5\,mmHg \cdot cm^{-2}$, $y = 0.98x + 2.81$). Estimates of E′es from tonometric pressure also closely correlated with E′es from radial arterial pressure ($r = 0.84$, SE of the estimate = $6\,mmHg \cdot cm^{-2}$, $y = 0.73x + 3.43$). Analysis by the Bland–Altman method of the estimates of E′es from tonometoric pressure, E′es from LV pressure, and E′es from direct radial pressure are shown in Fig. 2. No systematic measurement errors were seen.

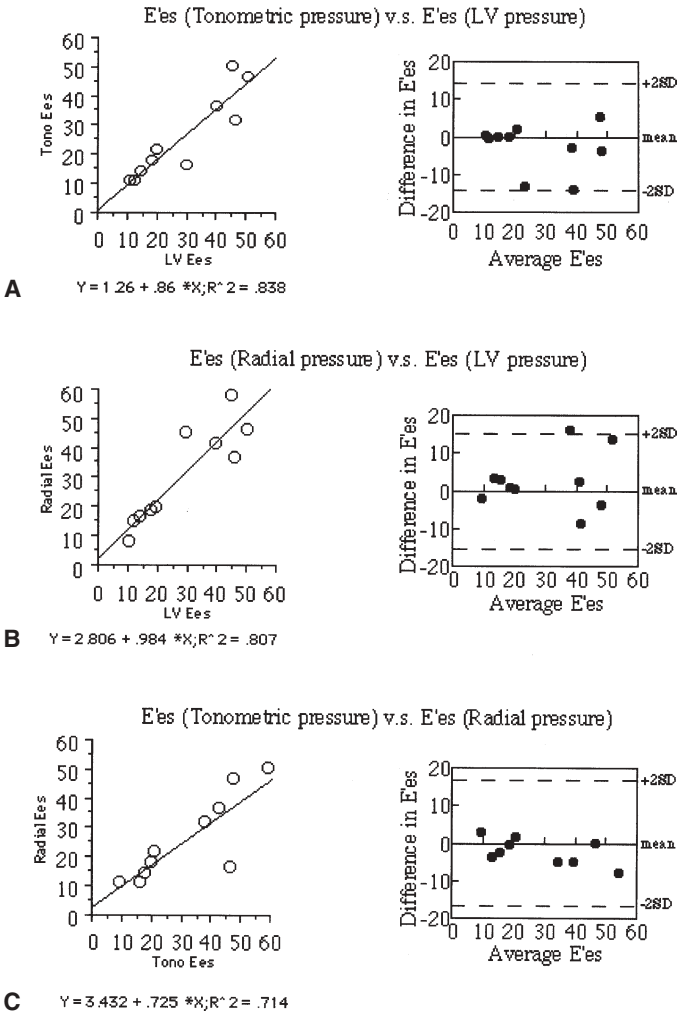

Fig. 2. Relationship between E′es (end-systolic elastance) values derived from LV pressure, radial pressure, tonometric pressures (*left*), and results of Bland–Altman bias analysis (*right*). **A** E′es from LV pressure versus E′es from tonometric pressure; **B** E′es from LV pressure versus E′es from radial pressure; **C** E′es from radial pressure versus E′es from tonometric pressure

Study 2

The surgical approach was transabdominal, and the average duration of aortic cross-clamping was 60 ± 15 min. Aortic cross-clamping significantly increased LV EDA by approximately 13%, and inversely, unclamping significantly decreased LV EDA by 9% (Table 1). E′a significantly increased by 44% by aortic clamping and significantly decreased 22% by unclamping. FAC was 56% before clamping. Aortic clamping significantly decreased FAC by 15%, whereas unclamping significantly increased FAC by 13% (Table 1).

Estimates of E′es from tonometric pressure closely correlated with E′es from radial arterial pressure (Fig. 3) during aortic clamping ($r = 0.95$, SE of the estimate = 2.4 mmHg·cm^{-2}, $y = 0.98x + 1.03$) and unclamping ($r = 0.96$, SE of the estimate = 2.4 mmHg·cm^{-2}, $y = 0.87x + 1.73$). There was no significant difference between E′es at clamping and at unclamping (Table 1).

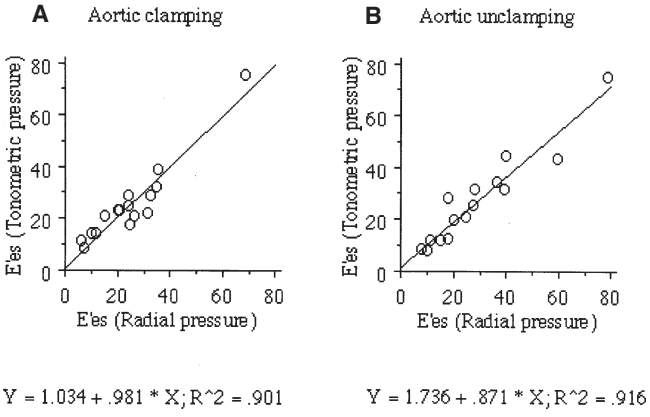

Fig. 3. Relationship between E′es values derived from radial pressure and tonometric pressures obtained at aortic cross-clamping (**A**) and unclamping (**B**)

Table 1. Changes in left ventricular end-diastolic area (EDA), fractional area change (FAC), effective arterial load (E′a), end-systolic elastance (E′es) from tonometric or direct radial pressure–area relations, and preload recruitable stroke force (PRSF) at aortic cross-clamping and unclamping

	Aortic clamping		Aortic unclamping	
	Before	After	Before	After
EDA (cm^2)	7.1 ± 2.6	8.0 ± 2.9*	7.7 ± 2.7	7.0 ± 2.6*
FAC (%)	55.7 ± 19.4	47.2 ± 17.6*	49.5 ± 19.0	56.0 ± 19.6*
E′a (mmHg·cm^{-2})	28.1 ± 13.1	40.5 ± 23.3*	36.8 ± 15.6	28.7 ± 13.1*
E′es (tonometric pressure) (mmHg·cm^{-2})	25.2 ± 15.6		25.9 ± 17.8	
E′es (direct radial pressure) (mmHg·cm^{-2})	24.6 ± 15.1		27.8 ± 19.6	

Data are mean ± SD
* $P < 0.05$ vs. before

Discussion

The LV end-systolic pressure-volume relationship or end-systolic elastance, Ees, is a measure for assessing cardiac contractility in animals and humans and is relatively independent of loading conditions [1,2,4,18]. Limitations in acquiring LV pressure as well as volume, however, have made it difficult to apply Ees as a routine intraoperative assessment of LV performance. Our study demonstrated that measures of LV cavity area by ABD of TEE and tonometric arterial pressure could be combined to construct online pressure–area loops, determining LV end-systolic pressure–area relations, E′es.

There are inevitable limitations in using two-dimensional data to represent three-dimensional volume and peripheral arterial pressure to represent LV pressure. LV area and volume cannot maintain a linear relationship over a wide range of values due to the determinants of each variable. However, previous investigators showed a linear relationship between changes in LV area and volume [7,9–14]. Gorcsan et al. [7,13] have shown the presence of a good relationship between LV area (using echocardiographic ABD) and LV volume (measured by electromagnetic flow probes). On the other hand, peripheral arterial pressure differ from LV pressure or aortic pressure due to a variety of factors, such as characteristics of arterial vascular system, viscous properties of blood, wave reflection, and quality of aortic valve. A small progressive rise in systolic pressure typically occurs from the ascending aorta to the peripheral arteries, and this pressure wave amplification is most pronounced in smaller, more distal arteries, such as the radial artery. Although differences in their absolute values existed also in our study, changes in radial arterial systolic pressure with IVC occlusions were similar to changes in LV pressure during an ejection phase. The E′es by tonometric pressure–area loops closely correlated with E′es by LV pressure–area loops (see Fig. 2). It has also been shown that LV end-systolic elastance can be estimated from pressure measurements in the femoral artery [9] and the radial artery [19]. Therefore, it is suggested that E′es using our noninvasive methods in clinical settings can substitute for LV end-systolic pressure–volume relationship.

Arterial pressure waveforms can be measured noninvasively and continuously by the use of arterial tonometry [15–17]. The latter method provides accurate and reliable real-time monitoring of blood pressure even during induced hypotension [16]. Sato et al. [17] performed a validation study of the tonometric blood pressure monitoring during Valsalva maneuver and the tilting test. They demonstrated that the beat-to-beat variability of tonometric pressure almost perfectly corresponded to that of intraarterial pressure in the physiologically significant frequency range. They concluded that the tonometric waveform was almost equal to the intraarterial waveform, except for the early systole, when a discrepancy between the two waveforms may exist. In this study, we were able to demonstrate that estimates of E′es from tonometric pressure closely correlated with E′es from LV pressure, and also with E′es by radial arterial pressure (see Fig. 2). Therefore, end-systolic pressure measured by tonometry can be used as a surrogate of LV pressure at end-systolic points during acute alterations in loading conditions to determine end-systolic pressure–area relations, E′es.

To estimate the LV end-systolic elastance, a rapid change in loading condition is necessary. In clinical settings, however, such interventions are not usually feasible. Accordingly, we utilized the aortic cross-clamping and unclamping technique, which is employed routinely during abdominal aortic surgery, to construct E′es. Several investigators have reported the hemodynamic changes in response to abdominal aortic clamping and unclamping [20–27]. To our knowledge, however, there are no studies that have used the aortic clamping and unclamping for intraoperative measurement of LV end-systolic elastance. Our results showed that aortic clamping and unclamping caused a rapid change in afterload thus allowing the determination of E′es. The data also showed that E′es did not differ between aortic clamping and unclamping. On the other hand, FAC, which is a two-dimensional substitute of ejection fraction (EF), was decreased by approximately 17% during clamping relative to that before clamping as well as after unclamping. The FAC can be influenced by loading conditions other than the contractile state. Thus, the decreases in FAC and EF during aortic clamping may be largely due to an increase in afterload rather than a decrease in LV contractility because of the lack of change in E′es.

In addition to LV contractility, changes in preload and afterload could be estimated during aortic clamping and unclamping by our methods. LV-EDA increased by about 20% at aortic clamping and recovered at aortic unclamping, indicating a significant increase in preload during aortic cross-clamping. This finding is consistent with other studies [20,28]. Previous studies have reported that aortic clamping is not associated with significant increases in pulmonary capillary wedge pressure [24] or central venous pressure [21], although this maneuver increased LV end-diastolic volume measured by radionuclide angiocardiograms by about 30% [20,28]. A decrease in LV diastolic compliance might render the pulmonary capillary wedge pressure a poor index of LV preload, and thus LV-EDA measured by TEE is a valuable adjunct in guiding preload evaluation in patients undergoing repair of abdominal aortic aneurysm [28]. Regarding changes in LV afterload, we obtained effec-

tive arterial load (E′a) as an alternative to afterload and found E'a to be increased by about 50% at aortic clamping and recovered after unclamping (see Table 1). These changes in afterload are in agreement with those shown in previous studies in which aortic cross-clamping increased systemic vascular resistance by about 25%–35% [21–25] or increased LV end-systolic wall stress measured echocardiographically by about 26% [20].

In conclusion, we demonstrated, in the present study, the feasibility of using tonometric pressure to simulate LV ejection pressure to determine LV end-systolic pressure–area relationship. These measurements were validated by the combined recording of ABD of TEE to simulate LV volume change. This noninvasive measurement of LV performance enables us to estimate LV performance during aortic cross-clamping and unclamping.

References

1. Sagawa K (1981) The end-systolic pressure-volume relation of the ventricle: definition, modifications and clinical use. Circulation 63:1223–1227
2. Sagawa K, Maughan L, Suga H, Sunagawa K (1988) Cardiac contraction and the pressure-volume relationship. Oxford University Press, New York
3. Kass DA, Yamazaki T, Burkhoff D, Maughan WL, Sagawa K (1986) Determination of left ventricular end-systolic pressure-volume relationships by the conductance (volume) catheter technique. Circulation 73:586–595
4. Grossman W, Braunwald E, Mann T, McLaurin LP, Green LH (1977) Contractile state of the left ventricle in man as evaluated from end-systolic pressure-volume relations. Circulation 56:845–852
5. Magorien DJ, Shaffer P, Bush CA, Magorien RD, Kolibash AJ, Leier CV, Bashore TM (1983) Assessment of left ventricular pressure-volume relations using gated radionuclide angiography, echocardiography, and micromanometer pressure recordings. A new method for serial measurements of systolic and diastolic function in man. Circulation 67:844–853
6. McKay RG, Spears JR, Aroesty JM, Baim DS, Royal HD, Heller GV, Lincoln W, Salo RW, Braunwald E, Grossman W (1984) Instantaneous measurement of left and right ventricular stroke volume and pressure-volume relationships with an impedance catheter. Circulation 69:703–710
7. Gorcsan J, Gasior TA, Mandarino WA, Deneault LG, Hattler BG, Pinsky MR (1993) On-line estimation of changes in left ventricular stroke volume by transesophageal echocardiographic automated border detection in patients undergoing coronary artery bypass grafting. Am J Cardiol 72:721–727
8. Cahalan MK, Ionescu P, Melton HJ, Adler S, Kee LL, Schiller NB (1993) Automated real-time analysis of intraoperative transesophageal echocardiograms. Anesthesiology 78:477–485
9. Gorcsan J, Denault A, Gasior TA, Mandarino WA, Kancel MJ, Deneault LG, Hattler BG, Pinsky MR (1994) Rapid estimation of left ventricular contractility from end-systolic relations by echocardiographic automated border detection and femoral arterial pressure. Anesthesiology 81:553–562
10. Gorcsan J, Gasior TA, Mandarino WA, Deneault LG, Hattler BG, Pinsky MR (1994) Assessment of the immediate effects of cardiopulmonary bypass on left ventricular performance by on-line pressure-area relations. Circulation 89:180–190
11. Perez JE, Waggoner AD, Barzilai B, Melton HJ, Miller JG, Sobel BE (1992) On-line assessment of ventricular function by automatic boundary detection and ultrasonic backscatter imaging. J Am Coll Cardiol 19:313–320
12. Gorcsan J, Morita S, Mandarino WA, Deneault LG, Kawai A, Kormos RL, Griffith BP, Pinsky MR (1993) Two-dimensional echocardiographic automated border detection accurately reflects changes in left ventricular volume. J Am Soc Echocardiogr 6:482–489
13. Gorcsan J, Romand JA, Mandarino WA, Deneault LG, Pinsky MR (1994) Assessment of left ventricular performance by on-line pressure-area relations using echocardiographic automated border detection. J Am Coll Cardiol 23:242–252
14. Denault AY, Gorcsan J, Mandarino WA, Kancel MJ, Pinsky MR (1997) Left ventricular performance assessed by echocardiographic automated border detection and arterial pressure. Am J Physiol 272:H138–H147
15. Kemmotsu O, Ueda M, Otsuka H, Yamamura T, Winter DC, Eckerle JS (1991) Arterial tonometry for noninvasive, continuous blood pressure monitoring during anesthesia. Anesthesiology 75:333–340
16. Kemmotsu O, Ueda M, Otsuka H, Yamamura T, Okamura A, Ishikawa T, Winter DC, Eckerle JS (1991) Blood pressure measurement by arterial tonometry in controlled hypotension. Anesth Analg 73:54–58
17. Sato T, Nishinaga M, Kawamoto A, Ozawa T, Takatsuji H (1993) Accuracy of a continuous blood pressure monitor based on arterial tonometry. Hypertension 21:866–874
18. Little WC, Cheng CP, Mumma M, Igarashi Y, Vinten-Johansen J, Johnston WE (1989) Comparison of measures of left ventricular contractile performance derived from pressure-volume loops in conscious dogs. Circulation 80:1378–1387
19. Dahlgren G, Veintemilla F, Settergren G, Liska J (1991) Left ventricular end-systolic pressure estimated from measurements in a peripheral artery. J Cardiothorac Vasc Anesth 5:551–553
20. Harpole DH, Clements FM, Quill T, Wolfe WG, Jones RH, McCann RL (1989) Right and left ventricular performance during and after abdominal aortic aneurysm repair. Ann Surg 209: 356–362
21. Seeman LH, Biber B, Martner J, Ponten J, Winso O (1996) Cardiovascular responses to experimental infra-renal aortic cross-clamping. Modulating effects of isoflurane, sodium nitroprusside and milrinone. Acta Anaesthesiol Scand 40:408–415
22. Silverstein PR, Caldera DL, Cullen DJ, Davison JK, Darling RC, Emerson CW (1979) Avoiding the hemodynamic consequences of aortic cross-clamping and unclamping. Anesthesiology 50:462–466
23. Gooding JM, Archie JJ, McDowell H (1980) Hemodynamic response to infrarenal aortic cross-clamping in patients with and without coronary artery disease. Crit Care Med 8:382–385
24. Attia RR, Murphy JD, Snider M, Lappas DG, Darling RC, Lowenstein E (1976) Myocardial ischemia due to infrarenal aortic cross-clamping during aortic surgery in patients with severe coronary artery disease. Circulation 53:961–965
25. Seeman LH, Biber B, Ponten J, Sundeman H, Winso O (1997) Low cardiac output abolishes cardiovascular responses to infrarenal aortic cross-clamping in the pig. Acta Anaesth Scand 41: 232–238
26. Reiz S, Peter T, Rais O (1979) Hemodynamic and cardiometabolic effects of infrarenal aortic and common iliac artery declamping in man—an approach to optimal volume loading. Acta Anaesth Scand 23:579–586
27. Askitopolou H, Young CA, Morgan M, Sykes MK (1978) Some cardio-pulmonary effects of infra-renal clamping of the abdominal aorta. Anaesth Intensive Care 6:44–48
28. Gillespie DL, Connelly GP, Arkoff HM, Dempsey AL, Hilkert RJ, Menzoian JO (1994) Left ventricular dysfunction during infrarenal abdominal aortic aneurysm repair. Am J Surg 168:144–147