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Assessment of Left Ventricular Contractile State by Preload-Adjusted Maximal Power Using Echocardiographic Automated Border Detection

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Objectives. We sought to assess the ability of preload-adjusted maximal power measured by echocardiographic automated border detection (ABD) to quantify left ventricular (LV) contractility by determining the effects of alterations in preload, afterload and contractile state.

Background. Preload-adjusted maximal power can reflect LV contractile state relatively independent of changes in loading conditions.

Methods. Eight anesthetized dogs had placement of aortic electromagnetic flow probes, LV and arterial pressure catheters and inferior vena caval (IVC) occluders; four had placement of thoracic aortic balloon occluders. Echocardiographic ABD measures of cross-sectional area were used as a surrogate for LV volume, and flow was estimated as the first derivative of area with respect to time. Power was calculated as the product of flow and pressure.

Results. Preload independence during vena caval occlusions was achieved by preload adjustment (\div [end-diastolic area]^{3/2}). Afterload independence was demonstrated by preload-adjusted

maximal power being unaffected by acute increases in LV systolic pressure induced by aortic occlusion. ABD preload-adjusted maximal power reflected changes in contractile state: increasing with dobutamine infusion from 36 ± 14 to 70 ± 15 mW/cm⁴ ($p < 0.05$ vs. control) and decreasing with propranolol infusion from 35 ± 13 to 17 ± 7 mW/cm⁴ ($p < 0.05$ vs. control). These changes were significantly correlated with calculations of preload-adjusted maximal power using aortic flow ($r = 0.90$, SEE 10.5 mW/cm⁴) and load-independent measures of end-systolic elastance from pressure-area loops ($r = 0.90$, SEE 10.6 mW/cm⁴). Calculations of normalized preload-adjusted maximal power using arterial pressure were also closely correlated with similar calculations using LV pressure ($r = 0.99$, SEE 3%).

Conclusions. Preload-adjusted maximal power using echocardiographic ABD can predict LV contractile state relatively independent of loading conditions and has potential for clinical application.

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Technologic advances continue to enhance the ability of echocardiographic imaging techniques to quantify cardiac function (1-4). Routine indices of left ventricular (LV) function, such as ejection fraction and cardiac output, persist in having the limitation of being sensitive to alterations in loading conditions. Maximal LV power has been shown to reflect contractile state, and preload-adjusted maximal power, accomplished by dividing by end-diastolic volume squared, has been recently shown to be relatively independent of preload, afterload and resistance (5,6). Because calculation of power does not require assessment over a wide range of pressure and volume values like pressure-volume loop studies (7-9), it appears to be an attractive load-independent alternative to assess contractility. Echocardiographic automated border de-

tection (ABD) can calculate cross-sectional area on-line as a surrogate for volume and has been shown to be useful in determining LV performance using pressure-volume relations (10-15). Our hypothesis was that on-line LV area data by ABD, coupled with pressure data, can be a useful quantitative means to assess contractile state. The objective of this study was to test this hypothesis by 1) assessing the effects of alterations in preload and afterload on echocardiographic ABD estimates of maximal power; 2) determining its ability to reflect alterations in contractility induced by pharmacologic inotropic modulation; and 3) assessing the utility of arterial pressure as a less invasive surrogate for LV pressure in the determination of maximal power. These principles were studied in an open-chest canine model with simultaneous measures of maximal power by aortic electromagnetic flow probe data and pressure-volume loop analyses as standards of reference for contractile state.

Methods

Preparation. Eight dogs, weighing 21.2 ± 0.6 kg (range 18.8 to 25.8), were included in the study. The study protocol was approved by the Institutional Animal Care and Use Committee and conformed to the "Position of the American

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Abbreviations and Acronyms

ABD	=	automated border detection
dA/dt	=	rate of change of left ventricular cross-sectional area
dV/dt	=	rate of change of left ventricular volume
IVC	=	inferior vena caval
LV	=	left ventricular

Heart Association on Research Animal Use" adopted by the Association in November 1984. All dogs were anesthetized with sodium pentobarbital (30 mg/kg body weight intravenously), endotracheally intubated and mechanically ventilated using a constant volume ventilator (Harvard). Supplemental intravenous doses of 50 to 75 mg of pentobarbital were given as needed. After a median sternotomy, a circumferential electromagnetic flow probe was placed snugly around the aorta just distal to the coronary arteries and interfaced with a flow meter (Carolina Medical). Flow data were integrated on-line for beat to beat recording of stroke volume. A thermodilution pulmonary artery catheter was placed for initial calibration of the electromagnetic flow meter. A high fidelity micromanometer-tipped catheter (MPC-500, Millar) was advanced into the LV from the right carotid artery. A fluid-filled femoral artery catheter with multiple side holes was advanced to record central arterial pressure. An adjustable inferior vena caval (IVC) occluder, consisting of surgical umbilical tape within a sliding plastic tube, was placed to rapidly alter preload. Four animals also had placement of a hydraulic vascular occluder (Intermedics) around the descending aorta to transiently increase afterload. Aortic and pulmonary artery catheters were connected to pressure transducers (MP-50, Gould).

ABD echocardiography. Two-dimensional images were recorded using an ABD echocardiographic system described in detail elsewhere (model 77035A, Hewlett-Packard) (1,10-15). This system calculates LV cross-sectional area on-line by analysis of the differential ultrasound backscatter characteristics of blood and tissue. A 2.5-MHz ultrasound transducer held stationary by a mechanical support apparatus was used for epicardial imaging. Contact with the epicardium was lightly maintained by a small, thin latex balloon filled with normal saline. Images were recorded from the mid-LV short-axis plane using the mid-papillary muscle level as an anatomic landmark and orienting the transducer to obtain the most circular cavity. This plane was selected because of the previously demonstrated linear relation between cross-sectional area and volume (10-16). Manual adjustments of overall transmit, time gain compensation, lateral gain control and region of interest features of the ultrasound system were made as described previously (10-13). The area of pixels, assigned as blood within the region of interest isolating the LV cavity, was calculated at 30 Hz and displayed as a waveform in real time.

Computer workstation. The ABD ultrasound system was configured to allow for direct recording of the area signal through a customized hardware and software interface, described previously (10-15). This analog area signal and other

physiologic signals, including electrocardiographic lead II, were digitized at 150 Hz for display and storage on a computer workstation (Apollo Computer Inc., model DN3550). The pressure signal was plotted with a variable delay of 40 ± 27 ms with respect to the area signal to correct for the time delay required for the ABD system to calculate area from each frame. The amount of 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.

Protocol. All measurements were made during stable apneic intervals, with respirations suspended at end-expiration to control for the effects of cardiopulmonary interactions. First, the preload and afterload sensitivities of maximal power estimates obtained by echocardiographic ABD were assessed. Left ventricular preload was altered by IVC occlusions maintained for 5 to 10 s. After all hemodynamic variables returned to baseline after release of the occluder, LV afterload was abruptly increased by transient occlusion of the descending thoracic aorta for 5 to 10 s in four dogs. Three consecutive IVC and aortic occlusion and release maneuvers were performed. Second, the effects of altering contractility by positive and negative inotropic modulation with dobutamine and propranolol, respectively, were then investigated. Caval occlusions were performed as described earlier for assessment of pressure-area measures as a load-independent standard of reference (13). After an initial control run, dobutamine was infused at 2 to 5 $\mu\text{g}/\text{kg}$ per min, accompanied by a series of IVC occlusion maneuvers. A 30-min washout period followed. Baseline measurements and IVC occlusions were then repeated for a second control. High dose propranolol (2 to 5 mg bolus) was infused as a negative inotrope to decrease contractility simulating acute heart failure. This dose was accompanied by rapid saline infusion to maintain systolic arterial pressure >90 mm Hg. Caval occlusions were then performed. Digitized physiologic data were transferred into a customized program written in ASYST software (ASYST Software Technologies, Inc.). Signal data were filtered using an inverse Fourier transform algorithm that increases the portion of the data that is in the signal spectrum and suppresses high frequency noise. This filtering process has been shown to decrease high frequency noise without altering the physiologic signal spectrum (13-15). Data were separated into cardiac cycles using a peak detection algorithm for the R wave of the electrocardiogram, allowing the user to eliminate ectopic beats.

Power calculations. Left ventricular power is the product of instantaneous pressure and flow. Flow can be assessed as the rate of volume change (dV/dt) of the ventricle (5,6). For the present study, the rate of change of LV cross-sectional area (dA/dt) obtained by echocardiographic ABD was substituted for dV/dt, so that LV power was estimated as the product of pressure and dA/dt (Fig. 1). Power was also calculated as the product of pressure and flow, using aortic flow measured by the electromagnetic flow probe as an independent standard of reference (5,6). Maximal power was determined as the peak

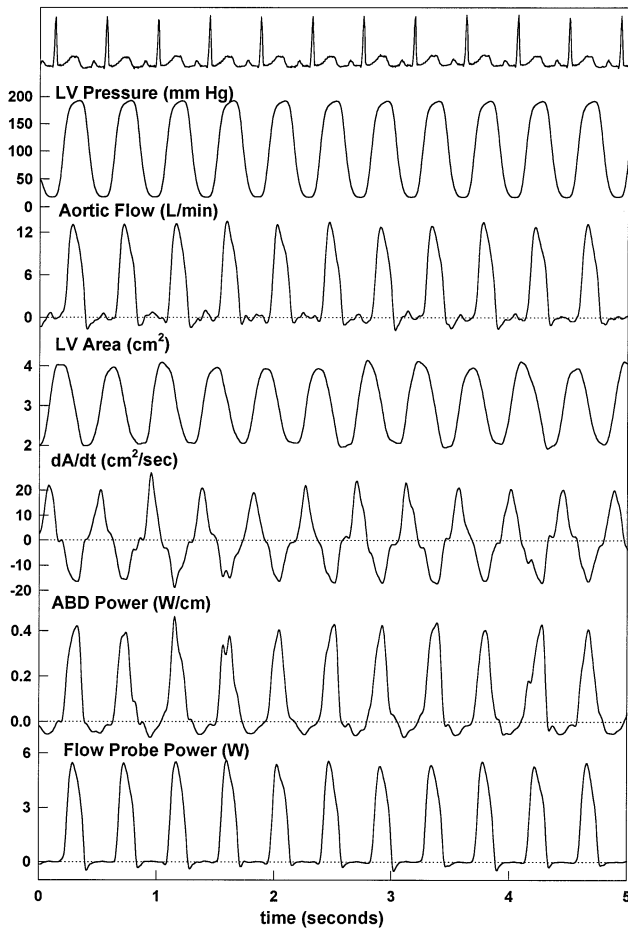


Figure 1. A sample plot of simultaneous LV pressure, aortic flow, echocardiographic ABD cross-sectional area, dA/dt, ABD power and flow probe power.

value during the cardiac cycle. For steady-state measurements, 10 s of data was divided into cardiac cycles, and maximal power values for each cardiac cycle were averaged. Power data during IVC occlusion were normalized to the baseline values for pooled analysis. Because the preload sensitivity of maximal power has previously been shown to be reduced by dividing by (end-diastolic volume)² (5,6), ABD maximal power, using changes in cross-sectional area, was preload adjusted in this manner, and also by dividing by end-diastolic area and (end-diastolic area)^{3/2} as alternate correction factors. To evaluate the less invasive applicability of this measurement, preload-adjusted maximal power calculations were repeated with aortic pressure substituted for LV pressure in the power calculations. LV ejection fractions were also calculated using a modified ellipsoid formula to compare preload-adjusted maximal power results with this common clinical index (17).

Pressure-area relations. End-systolic elastance and preload recruitable stroke work calculations were applied to the pressure-area loops obtained during inotropic modulation as load-independent reference standards for maximal power calculations (13,14). Area data were substituted for volume data for

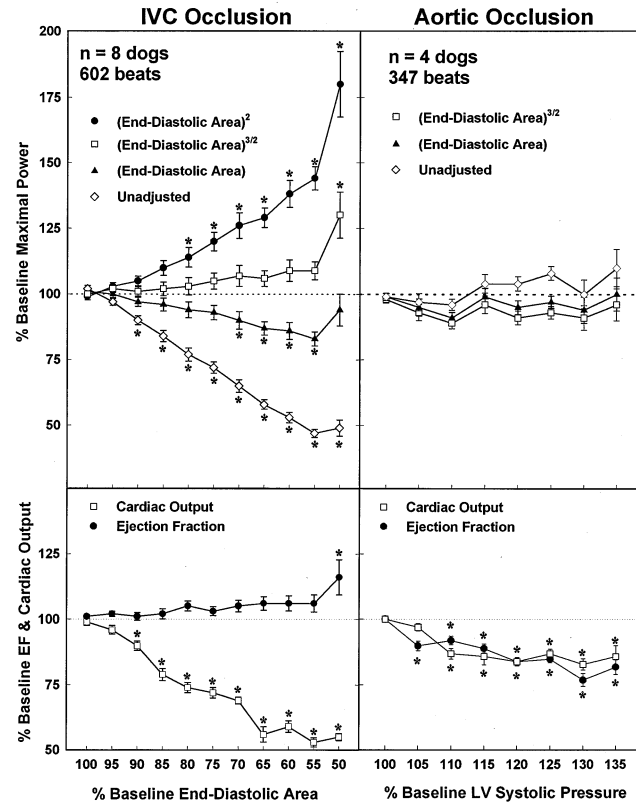


Figure 2. Pooled results of LV functional indexes during acute alterations in loading. Data are presented as mean value \pm SEM normalized to baseline values before IVC or aortic occlusions. Changes in preload and afterload are expressed as percent change in baseline end-diastolic area and percent change in baseline LV systolic pressure, respectively (* $p < 0.05$ vs. baseline). **Top left panel,** Plots of changes in unadjusted and preload-adjusted maximal power during caval occlusion. Preload sensitivity was largely eliminated by dividing by (end-diastolic area)^{3/2}. **Bottom left panel,** Plots demonstrating the relative preload insensitivity of ejection fraction (EF), but marked preload dependence of cardiac output. **Top right panel,** Plots demonstrating little change in preload-adjusted maximal power, despite significant increases in afterload. **Bottom right panel,** Plots showing predicted significant afterload sensitivity of both ejection fraction and cardiac output.

these calculations. Determinations of end-systolic elastance and preload recruitable stroke work were automatically calculated as previously described in detail elsewhere (13,14). Briefly, *end-systolic elastance* was defined as the slope of the maximal pressure-area points from differently loaded beats using an iterative linear regression analysis, in a manner analogous to the end-systolic pressure-volume relation (7-9). Preload recruitable stroke work was calculated as the slope of the linear regression equation obtained from the stroke work end-diastolic area relation with stroke work, estimated by integration of pressure with respect to area, analogous to the calculation of stroke work from pressure-volume loops (18).

Statistical analysis. LV functional indexes for all inotropic conditions were compared using analysis of variance for repeat maneuvers. Estimates of preload-adjusted maximal power using echocardiographic ABD were compared with measures

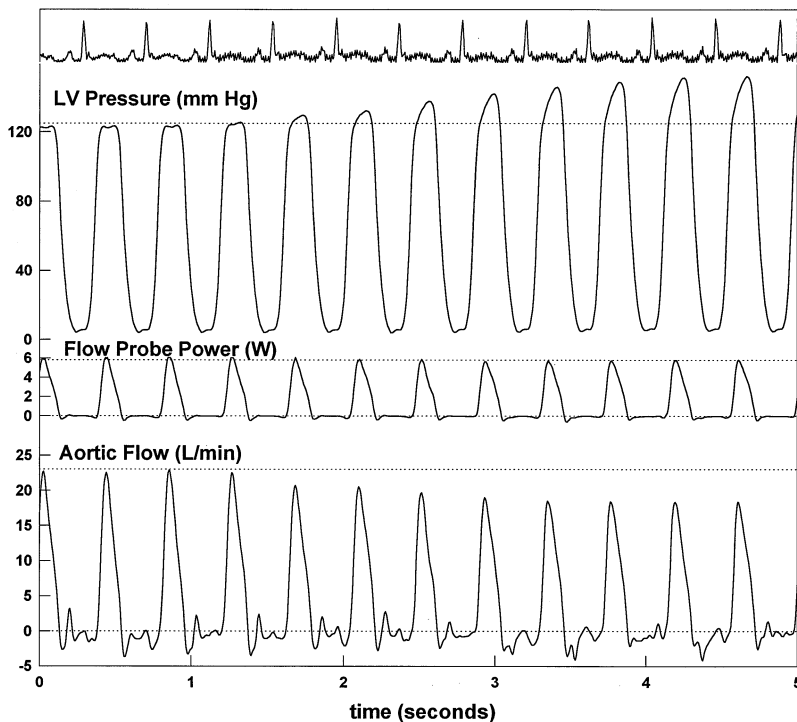


Figure 3. An example of simultaneous LV pressure, power and aortic flow data during acute changes in afterload induced by descending aortic occlusion. Although flow decreased as a result of increases in afterload, power remained unchanged.

of preload-adjusted maximal power using aortic flow, end-systolic elastance and preload recruitable stroke work by least-squares linear regression analysis. Calculations of preload-adjusted maximal power using echocardiographic ABD and LV pressure were normalized to baseline and compared with similar estimates substituting arterial for LV pressure using least-squares linear regression analysis and the method of Bland and Altman (19). Statistical significance is reported at $p < 0.05$. Data in the text are presented as mean value \pm SD and in Figure 2 as mean value \pm SEM for graphic presentation.

Results

Preload and afterload sensitivity. Alterations in preload by IVC occlusion were associated with predicted decreases in ABD maximal power as LV volumes decreased progressively. Adjusting maximal power calculations by dividing by (end-diastolic area)², which has been successful with volume data (5,6), produced a significant overcorrection of values ($p < 0.05$ vs. baseline). Although dividing by end-diastolic area reduced preload sensitivity, the most accurate preload adjustment was achieved by dividing by (end-diastolic area)^{3/2} (Fig. 2). Accordingly, preload adjustment of maximal power by dividing by (end-diastolic area)^{3/2} was chosen for subsequent analysis. Calculations of LV ejection fraction did not demonstrate significant preload sensitivity during IVC occlusions, but marked alterations in cardiac output occurred. Changes in LV afterload demonstrated no significant changes in maximal power using either electromagnetic flow data or echocardiographic data, despite significant increases in LV pressure from

132 \pm 22 to 165 \pm 24 mm Hg ($p < 0.001$ vs. baseline) (Fig. 3). This afterload insensitivity of preload-adjusted maximal power was also preserved (Fig. 2). In contrast, both LV ejection fraction and cardiac output demonstrated predicted significant afterload sensitivity to the identical hemodynamic maneuvers.

Alterations in contractile state by inotropic modulation.

Three dogs had cardiac arrest soon after propranolol infusion, and IVC occlusion runs were not possible. Significant increases in preload-adjusted maximal power occurred with dobutamine infusion, and opposing significant decreases occurred with propranolol infusion (Fig. 4). Pooled results of pharmacologic changes in contractile state appear in Table 1. An increase in contractile state was confirmed by similar significant increases in end-systolic elastance and preload recruitable stroke work by pressure-area loop analysis ($p < 0.01$ vs. control) (Fig. 5, Table 1). ABD estimates of preload-adjusted maximal power induced by inotropic modulation significantly correlated with preload-adjusted maximal power measured by the electromagnetic flow probe ($r = 0.90$, SEE 10.5 mW/cm⁴) (Fig. 6, top), with percent changes from control being even more closely correlated ($r = 0.98$, SEE 19%, $y = 0.83x + 0.5$). Echocardiographic preload-adjusted maximal power also correlated with the load-independent measures of end-systolic elastance ($r = 0.90$, SEE 10.6 mW/cm⁴) (Fig. 6, bottom) and preload recruitable stroke work ($r = 0.83$, SEE 13.1 mW/cm⁴).

Estimates of preload-adjusted maximal power using arterial pressure. Similar significant changes in preload-adjusted maximal power with inotropic modulation occurred when substituting arterial pressure for LV pressure (Table 1). The pooled results of ABD preload-adjusted maximal power using LV pressure and preload-adjusted maximal power using arte-

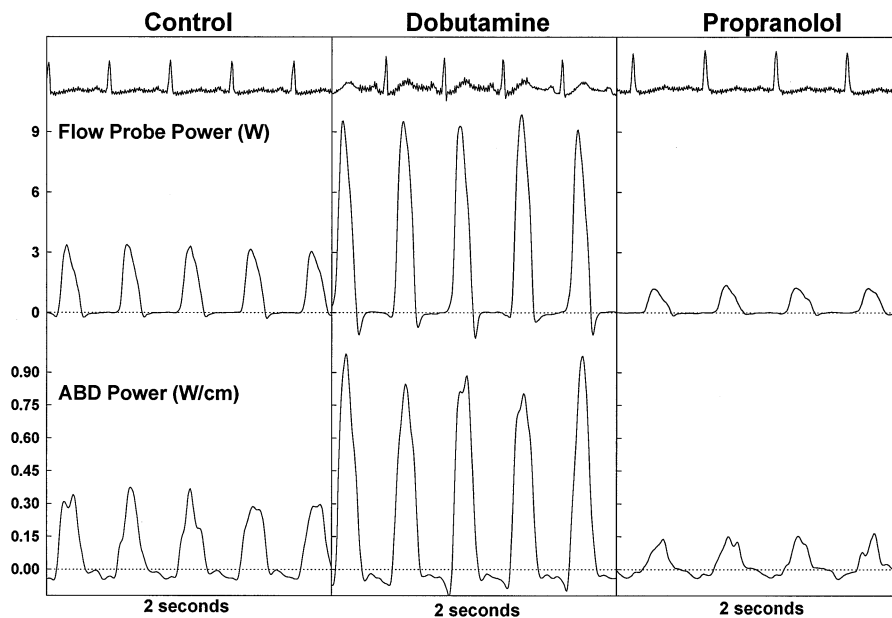


Figure 4. Examples of simultaneous plots of LV power calculated from flow probe data or echocardiographic ABD area data during control conditions and inotropic modulation with dobutamine and propranolol.

rial pressure from control and inotropic modulation portions of this experiment were normalized to baseline values (Fig. 7). A very close linear relation was demonstrated ($r = 0.99$, SEE 3%), with close limits of agreement. These data further support the use of arterial pressure as a less invasive surrogate for LV pressure (20).

Discussion

Power is defined as the time rate at which work is done with units expressed as J/s or as W. When determining the performance of a hydrodynamic pump, power is calculated as the product of pressure and flow. Power was previously reported as a method of determining LV function because it reflects both rates of pressure development and ejection (21-23). Kass and Beyer (5) renewed interest in its use to describe LV performance by proposing a means to reduce its preload sensitivity (dividing by end-diastolic volume squared) and by rigorously

demonstrating its relative load insensitivity in an animal model. These investigators later used preload-adjusted maximal power as a means to assess LV performance in patients with dilated cardiomyopathy and have shown a noninvasive approach using radionuclide angiography and noninvasive arterial pressure (6).

This study confirms the observations of Kass and Beyer of the relative load insensitivity of preload-adjusted maximal power and extends its application to using echocardiographic ABD measures of cross-sectional area as a surrogate for LV volume and preload adjusting by dividing by (end-diastolic area)^{3/2}, rather than (end-diastolic volume)². This load independence was demonstrated in the face of marked changes in the routine indices of ejection fraction and cardiac output induced by the same changes in afterload and preload. Our study also demonstrates the ability of echocardiographic preload-adjusted maximal power to reliably predict alterations in contractile state induced by positive and negative inotropic

Table 1. Left Ventricular Functional Response to Inotropic Modulation

	Control 1	Dobutamine	Control 2	Propranolol
Routine indexes				
Stroke volume (ml)	22 ± 4	29 ± 5*	23 ± 7	16 ± 4†
Cardiac output (liters/min)	2.8 ± 0.4	4.9 ± 1.4*	3.3 ± 1.1	1.9 ± 0.6†
Ejection fraction (%)	59 ± 14	75 ± 7*	60 ± 11	43 ± 4†
Load-independent indexes				
End-systolic elastance (mm Hg/cm ²)	32 ± 16	80 ± 23*	26 ± 10	12 ± 3†
Preload recruitable stroke work (mm Hg)	71 ± 31	156 ± 47*	73 ± 15	36 ± 5†
Preload-adjusted maximal power (mW/cm ⁴)				
LVP	36 ± 14	70 ± 15*	36 ± 13	17 ± 7†
AP	38 ± 12	68 ± 18*	35 ± 11	18 ± 8†

*p < 0.05 versus control 1. †p < 0.01 versus control 2. Data are presented as the mean value ± SD. AP = preload-adjusted maximal power calculated from automated border detection area and arterial pressure; LVP = preload-adjusted maximal power calculated from automated border detection area and left ventricular pressure.

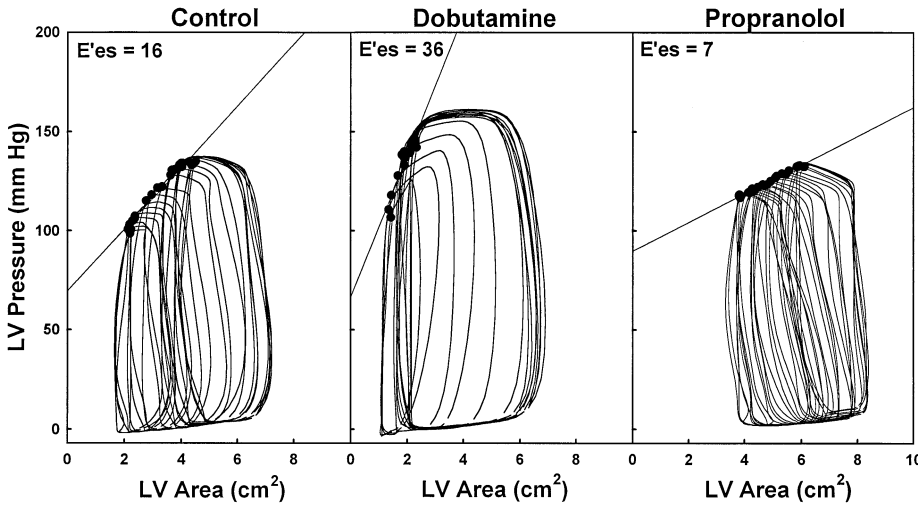
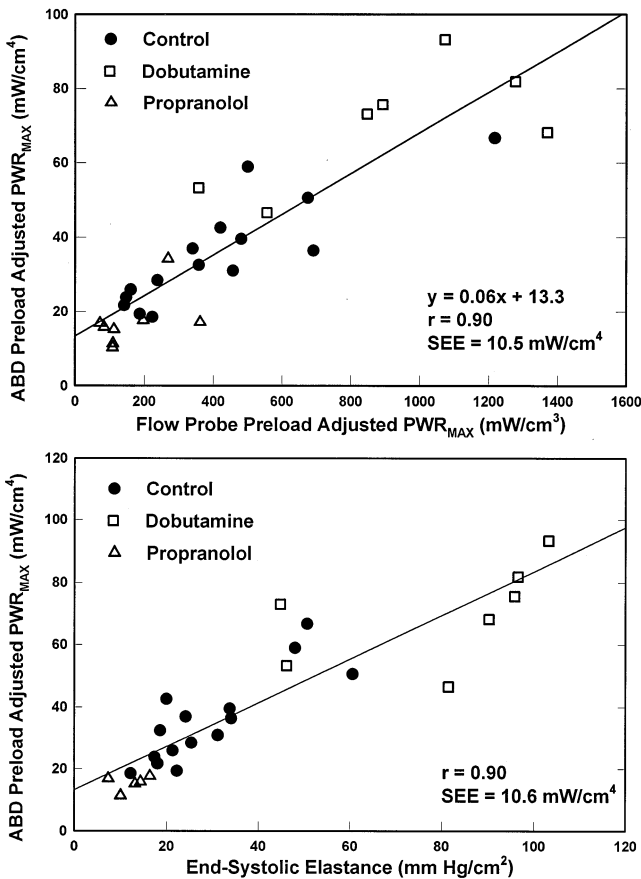


Figure 5. Examples of pressure–area loop plots demonstrating increases in end-systolic elastance (E'es) with dobutamine and decreases with propranolol, consistent with changes in contractility.

modulation and shows a striking similarity of calculations using arterial pressure as a substitute for LV pressure. An advantage of echocardiographic ABD over radionuclide estimates of volume is the ability to make simultaneous beat by beat

Figure 6. Scatterplots showing the relation of changes in echocardiographic ABD preload-adjusted maximal power (PWR_{MAX}) with flow probe preload-adjusted maximal power (top) and end-systolic elastance (bottom).



pressure and flow estimates to assess power on-line. These findings should enable the bedside application of this relatively load-independent method to assess LV performance in patients who are being monitored by arterial pressure catheters in the operating room and intensive care unit (24). Furthermore, more recent advances in on-line noninvasive assessment of arterial pressure (25–27) can be coupled with this echocardiographic technique to assess contractile state in many more clinical settings. Because a range of pressure and volume values do not need to be studied as with pressure–volume loop assessment, this method is attractive for clinical applications.

Study limitations. A known limitation of preload-adjusted maximal power, similar to pressure–volume relations, is that cardiac performance at the LV chamber level may not precisely define contractility at a more basic level because myocardial function cannot be completely uncoupled from loading conditions. However, the ability of this index to characterize global LV contractile state in a less ambiguous manner than with conventional load-dependent ejection phase indices is advantageous. Another limitation of preload-adjusted maximal power is that its load insensitivity may not extend beyond the plateau of physiologic pressure and volume values. Theoretic and experimental data show that loading conditions can affect this measure at the extremes of increases in afterload or decreases in preload (5,6). Accordingly, preload-adjusted maximal power may be less reliable in patients who are severely hypovolemic or who have marked elevated arterial pressures. Another limitation is that autonomic blockage was not performed in these experiments, and the effects of the sympathetic and parasympathetic nervous system on measures of power and pressure–volume relations could not be eliminated. However, autonomic blockage is not performed in clinical scenarios, and the data reported herein have practical significance. A technical limitation of this study is the use of thermodilution cardiac output as a reference standard for flow probe calibration. This may be a source of some random error in our results. Another limitation is that ABD requires a high quality echo-

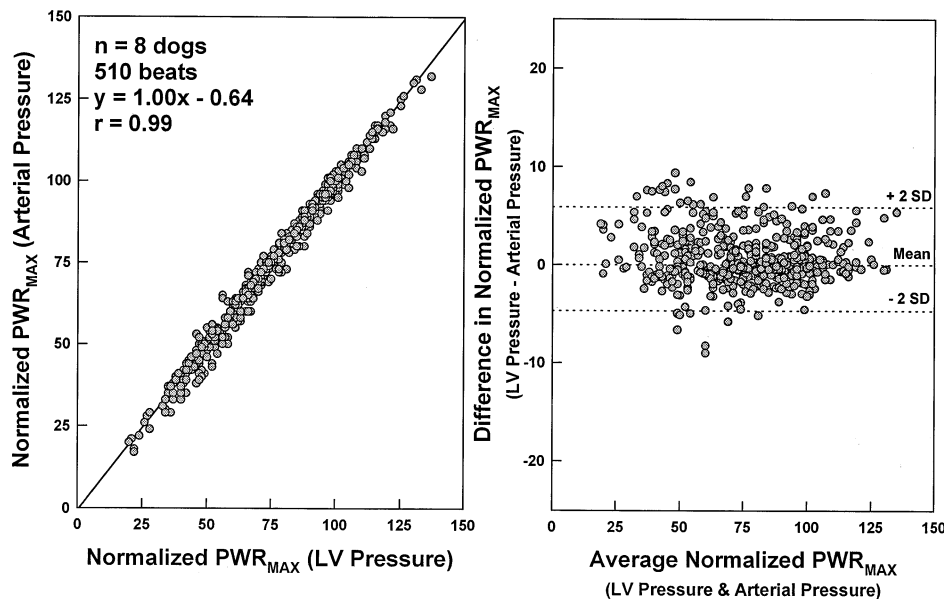


Figure 7. Scatterplots showing the relation of maximal power (PWR_{MAX}) calculated using arterial pressure with maximal power calculated using LV pressure. Data shown are pooled results from all control and inotropic modulation portions of this experiment normalized to baseline values. **Left panel,** Linear regression analysis. **Right panel,** Bland-Altman analysis.

cardiographic image to tract properly. The potential exists to apply these power calculations to Doppler flow estimates. However, a system with real-time calculation of Doppler flow with continuous data output is not presently available. A known experimental limitation is the use of a single two-dimensional tomographic plane to reflect changes in true LV volume. Previous investigators have shown a highly linear relation between the mid-LV short-axis plane and volume (16). We have also shown this relation to be linear over the physiologic range of values using echocardiographic ABD in isolated and intact canine cardiac experimental models and humans (10-13). This area-volume relation becomes curvilinear with very low volumes, and global LV volume may not be accurately represented in patients with geometric distortion, such as ventricular aneurysms. Estimates of preload-adjusted maximal power using arterial pressure may also be inaccurate in patients with aortic valve disease or significant mitral regurgitation. All of these limitations may be easily recognized by routine echocardiographic and Doppler studies, which can be used for proper patient selection. Despite the limitations of the ABD method of assessing preload-adjusted maximal power, it represents a physiologically significant means to assess global LV contractility and has potential for immediate clinical application.

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